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How Do Cognitive Behavioral Therapy and Interpersonal Psychotherapy Improve Youth Depression? Applying Meta-Analytic Structural Equation Modeling to Three Decades of Randomized Trials

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Investigating the mechanisms through which psychotherapy brings about desired change can inform efforts to improve therapies. We applied meta-analytic structural equation modeling (MASEM) to assess putative change mechanisms for cognitive behavioral therapy (CBT) as mediators of youth depression treatment outcome. Then, we tested whether candidate mediators (CMs) showed evidence of treatment-specificity to CBT versus interpersonal psychotherapy (IPT). Literature searches identified 34 randomized trials (27 CBT, 6 IPT, 1 CBT/IPT, 3,868 participants, published 1982–2020) that measured seven CMs: negative cognition, social engagement, family functioning, pleasant activity engagement, problem solving, reframing, or avoidance. We assessed mediational pathways and whether posttreatment CMs mediated treatment effects on posttreatment depression symptoms, covarying pretreatment CMs, and symptoms. Treatment type was tested as a moderator of mediational pathways. Results show that negative cognition (24 trials) and pleasant activities (3 trials) mediated depression symptom outcome in CBT. Social engagement and family functioning showed stronger mediation in IPT (5 and 6 trials) than in CBT (14 and 13 trials). We conclude that negative cognition is a robust mediator of CBT but may not be treatment-specific; pleasant activities may also be a mediator of CBT. However, the lack of treatment or mediation effects involving problem solving and reframing contradicts CBT theory. In contrast, social and family mechanisms appear to be IPT-specific mediators. These conclusions are provisional due to small samples examining IPT and several CMs, limitations in CM measurement (i.e., posttreatment retrospective report), and assumptions of MASEM—and will need to be confirmed when more and better evidence accumulates.

Public Significance Statement

To understand how psychotherapy impacts youth depression, we used a new approach to maximize and evaluate information gained from studies of two prominent therapies published over 3 decades. We found that cognitive behavioral therapy reliably reduced negative thinking—though it is unclear through what process—and in some cases, increased engagement in pleasant activities, with both changes in turn reducing depression symptoms. Interpersonal psychotherapy (IPT) appeared to improve depression via similar pathways to those posited for CBT (reducing negative thinking) as well as different pathways, specifically by improving young people's interpersonal skills and their peer and family relationships. Because few studies were available for some analyses, and because of limitations in the way the data were collected and analyzed, these findings are tentative and need to be confirmed in the future when more and better quality studies are conducted.

Keywords: children and adolescents, depression, cognitive behavioral therapy, interpersonal psychotherapy, meta-analysis

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continued

Treatment researchers have long advocated for investigating the mechanisms through which psychotherapy brings about desired change in clinical problems. Understanding change mechanisms is a critical step toward making therapies more efficacious and efficient (Doss, 2004; Kazdin & Nock, 2003; Kraemer et al., 2002; Ng & Weisz, 2016). More recently, the National Institute of Mental Health (NIMH) has been actively encouraging research on change mechanisms. As part of the experimental therapeutics framework (National Institute of Mental Health [NIMH], 2016), funding is prioritized for the development of treatments explicitly designed to target change mechanisms.

Given the burgeoning interest in therapy change mechanisms, it seems surprisingly difficult to locate research reviews devoted to this topic. A recently published book (Maric et al., 2015) and journal special issues (McKay, 2019; Sumner et al., 2018) address behavior change mechanisms in psychotherapy. However, these excellent sources are far outnumbered by the many research reviews focusing only on psychotherapy outcomes (e.g., Chambless & Ollendick, 2001; Kendall, 1998; Lonigan & Elbert, 1998; Silverman & Hinshaw, 2008; Southam-Gerow & Prinstein, 2014), books (Nathan & Gorman, 1998, 2002, 2007; J. R. Weisz, 2004; Weisz & Kazdin, 2010, 2017), and websites (http://www.blueprintsprograms.com, http://psychologicaltreatments.org/, https://effectivechildtherapy.org/). Indeed, it is quite likely that many researchers, practitioners, and students in clinical psychology and other mental health fields can name several empirically supported treatments (ESTs) and some of the criteria used to evaluate them, but that far fewer could do the same with change mechanisms. Reviews that summarize findings on a broad array of putative change mechanisms, ideally using systematic search procedures, may inform researchers' efforts to identify those mechanisms with the strongest empirical support for in-depth study.

Challenges in Studying and Summarizing Research on Change Mechanisms

One factor that has facilitated progress in identifying ESTs is established approaches to evaluating, organizing, and integrating the available evidence on treatment outcomes (e.g., Chambless & Hollon, 1998; Nathan & Gorman, 2007; Southam-Gerow & Prinstein, 2014). Gold standard approaches to studying treatment outcomes include: (a) randomized controlled trials (RCTs); (b) clear criteria for evaluating the strength of evidence for a treatment's efficacy and the methodological rigor of treatment studies; and (c) meta-analyses, the use of quantitative methods to synthesize the results of the available evidence and estimate mean effects. These approaches have brought the field closer to a consensus on what works for which disorders, how well they work, and what does not seem to work or need further study. However, identifying what psychotherapies work offers little insight into how they work and, relatedly, how they may be modified to maximize or accelerate treatment gains. Addressing both of these questions requires a solid understanding of the change mechanisms that drive improvement in symptoms.

In contrast, there has been much less clarity on the approaches best suited to studying change mechanisms. Although RCTs are often used, investigators have differed in how they analyzed putative change mechanisms. Some have treated them as a type of outcome, demonstrating posttreatment differences between treatment conditions (see Weersing & Weisz, 2002). Others have treated them as predictors of outcome by examining their association with concurrent or subsequent symptom severity or adaptive functioning (see Crits-Christoph et al., 2013). A growing number of researchers have examined them as mediators: intermediate variables evident during the course of treatment that statistically account for the treatment–outcome relationship (Kazdin, 2007; Kraemer et al., 2002). Mediation tests treat the putative change mechanism as both an outcome and a predictor of outcome, thus they are more informative than either test alone (i.e., treatment–mechanism or mechanism–outcome). However, mediation tests are often underpowered because of the relatively small size of RCT samples (Kraemer et al., 2002). Moreover, the most common Baron and Kenny (1986) causal steps test of mediation has particularly low power compared to subsequently developed tests (Fritz & Mackinnon, 2007).

Importantly, some researchers have argued that mediation tests are helpful but insufficient to establish change mechanisms, which require multiple statistical and logical criteria to be satisfied, often across multiple studies, to demonstrate that they cause therapeutic change. Drawing from the scientific and medical literature on establishing causation (e.g., Hill, 1965), Kazdin and Nock (2003; see also Kazdin, 2007) described criteria for change mechanisms that include strength, consistency, and specificity of the mechanism–outcome relationship; a dose–response relationship between mechanism and outcome; plausibility and coherence of the mechanism; frequent assessments of the candidate mechanism and outcome to establish temporal precedence of the mechanism; and direct experimental manipulation of the putative mechanism to establish its causal role (Kazdin, 2007; Kazdin & Nock, 2003).

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All other articles, theses, and dissertations of the included trials are listed in Appendix A (for CBT trials) and Appendix B (for IPT trials).

This meta-analysis was not preregistered. The codebook, data, and syntax used are available at https://osf.io/xpku2/?view_only=5f7543399e1a4de9862e71551665e912.

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A different framework by Doss (2004) distinguishes change mechanisms (i.e., changes in client characteristics and skills outside of sessions) from therapy change processes (i.e., what the therapist does during sessions such as specific techniques used) and client change processes (i.e., what the client does during sessions or as a result of the sessions such as in-session engagement or between-session homework completion). Doss recommended experimental manipulation for studying change processes and mediation analysis for change mechanisms. The multiple criteria–multiple study approaches are valuable as aspirational targets, but they are logistically challenging, time- and resource-intensive—it is unclear whether any psychotherapy construct has satisfied all of these criteria. Thus, they seem most helpful as guidelines for planning future research rather than for analyzing existing data.

The variety of constructs closely related to change mechanisms and the different approaches used to study them has no doubt complicated attempts to summarize the evidence base. There is no established framework for systematically classifying putative change mechanisms based on the strength of the evidence or rigor of methods used. Meta-analyses often synthesize only the treatment–mechanism or mechanism–outcome relationship (Lipsey & Wilson, 2001). Furthermore, many of these examine only one type of putative change mechanism (e.g., McLeod, 2011; see Chu & Harrison, 2007, for an exception), thereby precluding comparisons among different change mechanisms. Systematic reviews have been conducted on putative change mechanisms analyzed as treatment mediators, but relatively few RCT investigators report findings of mediation tests (and even fewer report nonsignificant findings, perhaps due in part to publication bias), and reported findings tend to be conflicting and derived from different methods (Ng et al., 2020; Weersing & Weisz, 2002)—these problems make it difficult to draw conclusions about which variables are mediators, which are mediators under certain conditions, and which are not mediators.

Advancing Change Mechanism Research Through Meta-Analysis of Mediation Models

To advance the study of psychotherapy change mechanisms, we propose using meta-analysis to identify robust treatment mediators across RCTs that could serve as promising candidates for future resource-intensive research needed to meet the multiple criteria required to establish change mechanisms. Consistent with Kazdin and Nock (2003), we use the term, *mediator*, as a statistical construct, denoting a variable that significantly accounts for the treatment–outcome effect in an RCT, and the term, *change mechanism*, as a substantive construct, denoting an event or process that causes desired change due to treatment, throughout this article. We also refer to variables that we will test for mediation as *candidate mediators* (CMs).

Our approach draws on an emerging methodology known as model-based meta-analysis (Becker, 2009) or meta-analytic structural equation modeling (MASEM; Cheung & Hafdahl, 2016). MASEM combines meta-analysis and structural equation modeling (SEM) techniques—effect sizes are first synthesized into a pooled correlation or covariance matrix, and then the matrix is used to fit the models of interest, which may include mediation models.

Applying MASEM to putative change mechanisms offers a number of advantages. First, it moves beyond the treatment–mechanism and mechanism–outcome bivariate relationships analyzed separately in prior systematic and meta-analytic reviews to address the question of whether a putative change mechanism mediated treatment effects in a mediation model with three or more variables. Second, it can help resolve the mixed findings resulting from mediation analysis in individual RCTs by estimating a mean mediation effect per putative change mechanism while giving greater weight to larger samples, which usually yield more precise estimates than smaller samples. Third, it facilitates the comparison of different putative change mechanisms because the conjoint mediational pathway (*ab*) is measured on a standardized metric (i.e., product of regression coefficients computed from a matrix of pooled correlations that are scale-independent), thus distinguishing stronger from weaker mediators. Fourth, it permits the examination of categorical moderators of mediation effects, which can shed light on conditions under which mediation does or does not occur, and clarifies the relationship between the moderator and the indirect effect that is examined (Cheung, 2022). Fifth, it can address the lack of power to detect mediation effects in individual RCTs by pooling data from multiple RCTs to increase the total sample of youths. Sixth, MASEM has demonstrated superior performance on multiple metrics (e.g., bias, precision, coverage) compared to other methods of synthesizing indirect effects (van Zundert & Miočević, 2020). Finally, it offers a way to fully exploit evidence from existing RCT data because MASEM can incorporate data from RCTs that reported partial data (i.e., some of the correlations in the matrix)—this last advantage may be especially important given that only a fraction of RCTs that measured putative change mechanisms reported testing them as mediators (Ng et al., 2020; Weersing & Weisz, 2002).

Despite the utility of MASEM, published meta-analyses of treatment mediation models are rare. We are aware of one pertaining to marital and family therapies (Shadish & Sweeney, 1991), and another to mindfulness-based therapies for adults (Gu et al., 2015). To our knowledge, none has focused on youth psychotherapies.

Obstacles and Opportunities in Examining How Psychotherapy Treats Youth Depression

The aims of the present meta-analysis are to demonstrate the use of MASEM to identify psychotherapy mediators, to quantify mediation effects, and to answer substantive questions about how psychotherapy improves youth depression. We focused on youth depression because persistently modest psychotherapy effects (Eckshtain et al., 2020; Weisz et al., 2006; Weisz, Kuppens, et al., 2017) and the absence of an increase in effect size across the decades (Weisz et al., 2019) suggest a need to understand pathways to improvement. We focused on cognitive behavioral therapy (CBT) and interpersonal psychotherapy (IPT)—the two treatment types that met criteria as ESTs in the latest systematic review of psychotherapies for youth depression commissioned by the Society of Clinical Child and Adolescent Psychology, Division 53 of the American Psychological Association (Weersing, Jeffreys, et al., 2016). Both treatments have also repeatedly outperformed control groups as shown in meta-analyses, although mean effect sizes varied considerably within and between meta-analyses depending on control type, additional treatment components, analytic approach, and other factors (CBT: Eckshtain et al., 2020, $g = 0.31$, 95% CI [0.18, 0.44], number of studies, $k = 34$; Weisz, Kuppens, et al., 2017, $g = 0.35$, $k = 31$; Zhou et al., 2015, $g = 0.46$, 95% CrI [0.18, 0.74],

Benefits of MASEM

$k = 33$; IPT: Eckshtain et al., 2020, $g = 0.78$, 95% CI [0.43, 1.13], $k = 5$; Zhou et al., 2015, $g = 0.59$, 95% CrI [0.18, 1.00], $k = 8$).

Both ESTs are based on clear and distinct theoretical rationales. CBT theory posits that depression is precipitated or perpetuated by maladaptive thoughts and behaviors. Therefore, treatment focuses on cognitive restructuring to change these thoughts to more positive, rational ones, as well as pleasant activity scheduling to increase engagement in reinforcing, adaptive behaviors; for youths, modules to improve problem solving, relaxation, communication, and social skills may also be delivered (e.g., Rohde, 2017). In contrast, IPT theory posits that interpersonal problems and a lack of positive relationships lead to distress, isolation, and depression. Thus, treatment involves building interpersonal skills and fostering understanding and expression of affect; for youths, treatment focuses on relationship issues particular to adolescence, including negotiating increased autonomy from parents, coping with parental separation/divorce, beginning romantic relationships, and handling peer pressure (e.g., Jacobson et al., 2018).

In spite of their documented efficacy and well-articulated theories, there is little evidence indicating that CBT and IPT ameliorate youth depression via their putative change mechanisms. In a meta-analysis of 14 CBT trials for depressed youths, Chu and Harrison (2007) found only small–medium treatment effects on cognitive mechanisms ($d = 0.35$, range [−0.64, 1.36], $p < .01$, $k = 16$) and nonsignificant effects on behavioral ($d = 0.01$, range [−0.38, 0.39], $k = 8$) and coping ($d = 0.05$ range [−0.03, 0.14], $k = 3$) mechanisms—all much smaller than the effects on depression symptoms ($d = 0.60$, range [−0.51, 1.69], $k = 20$). This meta-analysis did not examine the mechanism–outcome association or the treatment–mechanism–outcome conjoint mediational pathway.

Moreover, as suggested earlier, systematic reviews of mediators of ESTs targeting youth depression have yielded sparse and mixed findings. Weersing, Jeffreys, et al. (2016) identified only five RCTs of CBT that tested for treatment mediators. In our recent systematic review of putative change mechanisms and change processes in articles published through 2016 (Ng et al., 2020), we added another three RCTs (2 CBT, 1 IPT) that directly tested for mediational relationships for youth depression outcomes. Our systematic review identified the following as statistically significant mediators: negative cognition in four of six CBT trials, problem solving in two of two CBT trials, pleasant activity scheduling in one of two CBT trials, and social engagement in one of one IPT trial (and 0 of 2 CBT trials). Since 2016, we have found only one more mediation study—social engagement and family functioning were significant mediators of IPT in a subsample of depressed adolescents (Reyes-Portillo et al., 2017). All these putative change mechanisms failed to mediate outcome significantly in other trials or for similar measures within the same trial, and the small number of mediation studies made it difficult to discern true differences from chance variation due to varying study characteristics or analytic methods. Our systematic review found that all the remaining CMs were each tested in only one RCT and showed nonsignificant indirect effects, which renders their nonmediator status tentative.

More important was what we did *not* find—three quarters of the 34 RCTs did *not* conduct formal mediation tests, although they measured putative change mechanisms and change processes (Ng et al., 2020). We were struck by how underutilized the evidence base was, and the untapped opportunity these studies embodied for understanding how ESTs treat youth depression. Although our systematic review employed a simple vote count and a modified version of the criteria for ESTs (see Chambless & Hollon, 1998; Southam-Gerow & Prinstein, 2014) to tentatively classify the putative mediators by their level of evidence, a meta-analysis of mediation models would be a more comprehensive and precise approach to synthesize the evidence on treatment mediators in ESTs for youth depression.

The Present Meta-Analysis

In the present meta-analysis, we apply that more comprehensive and precise approach to a database updated through September 2020 and included all RCTs of ESTs for youth depression that measured a CM. Whereas the systematic review (Ng et al., 2020) identified which CMs were measured, counted the number of RCTs in which CMs were measured and tested as treatment mediators by trial investigators, and summarized those investigators' mediation findings; this meta-analysis quantified mediation effects across CBT trials for the most commonly measured CMs and examined whether these effects differed significantly from those in IPT trials. The overarching goal of the present meta-analysis goes beyond documenting what trial investigators have measured, tested, and found, to evaluate the strength of empirical support for the CBT and IPT models, and for the theory that psychotherapy for youth depression may work via mechanisms specific to treatment type.

We used univariate meta-analysis to quantify treatment effects on CMs and then employed MASEM to fit mediation models for the CMs that evidenced treatment effects. Our approach is intended to maximize the information gained, statistical power to detect mediation effects, and coverage of the overall evidence base. By applying the same mediation tests to estimate mean mediation effects across all RCTs and change mechanisms, we also hoped to minimize differences due to the analytic method, average out variation due to chance, and facilitate comparisons between putative change mechanisms and between treatments. Our research questions, analyses, and results are organized under two studies.

Study 1 focuses on testing mediators in CBT trials. Putative change mechanisms that significantly mediate outcomes across all CBT trials could be considered robust mediators that may play an important role in reducing depression symptoms. If cognitive, behavioral, problem solving, and other change mechanisms targeted by CBT emerge as robust mediators, these findings would provide confirmatory evidence that CBT works the way it was intended. Additionally, robust mediators could be accorded priority in future research to establish temporal precedence and causality in relation to symptom outcomes. Moreover, clinical researchers and practitioners may consider activating robust mediators earlier, and more intensively, to enhance the effects of CBT (Doss, 2004; Kraemer et al., 2002). Alternatively, a CBT protocol may be simplified to include only those modules targeting the robust mediators to make them more applicable to everyday clinical, school, or primary care settings (see Weisz, Kuppens, et al., 2017).

Study 2 includes data from CBT and IPT trials to test whether mediation effects depend on treatment type. Because of the small number of RCTs that tested IPT, we combined the CBT and IPT samples and tested treatment type as a moderator of treatment and mediation effects. Putative change mechanisms targeted by CBT that are robust mediators in CBT trials, and on which CBT exerts greater effects compared to IPT, may be considered to be specific to

Although they were not conclusive, authors had another systematic review for some of the meditators

CBT and would strengthen support for CBT theory. Furthermore, the identification of robust, treatment-specific mediators would contribute evidence that could help to resolve a longstanding debate about whether psychotherapies work primarily via specific factors—mechanisms specific to the theory on which the psychotherapy is based—or via common factors—mechanisms common to all *bona fide* psychotherapies (i.e., those that are intended to be therapeutic rather than designed to serve as a control; see DeRubeis et al., 2005; Wampold, 2005).

Study 1: Mediators of Cognitive Behavioral Therapy

Study 1 addresses the questions: Which putative change mechanisms are robust mediators of outcome in RCTs of CBT, and what is the relative strength of their mediation effects? Which putative change mechanisms consistently fail to mediate treatment outcomes, and which need to be assessed in future research to obtain sufficient data for analyses? To answer these questions, we selected seven CMs with minimally sufficient data for analysis: negative cognition, social engagement, family functioning, problem solving, pleasant activities, reframing, and avoidance. We then extracted data from each contributing CBT study on the statistical relationships needed to test for mediation and fit meta-analytic structural equation models to those data. Treatment mediation would be most strongly supported by significant treatment effects on CMs (Path *a*) and depression symptom outcomes (Path *c*), significant associations between CMs (Path *b*) and depression outcomes, and significant mediational *ab* pathways (see Figure 1).

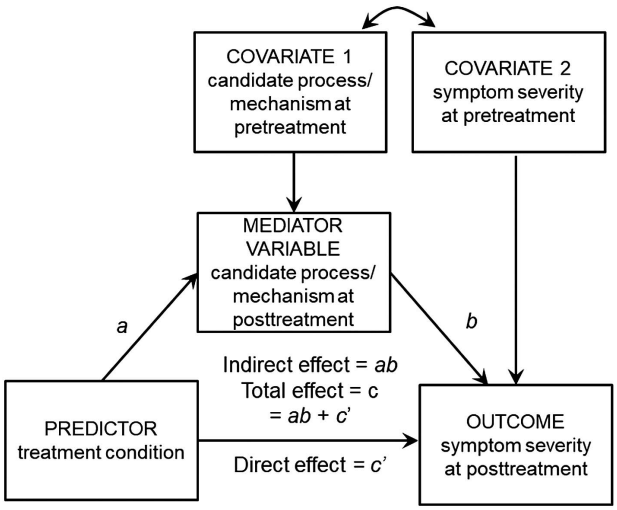
Study 1: Method

Inclusion Criteria and Literature Search

Studies for this meta-analysis were drawn from a larger database of youth psychotherapy RCTs for internalizing or externalizing problems. That database included RCTs that randomly assigned individual participants to any psychotherapy versus waitlist/no treatment, attention or pill placebo, case management, or usual care control; recruited youths with a mean age between 4 and 18 years with elevated symptoms of depression, anxiety, conduct disorder, or attention-deficit hyperactivity disorder; and assessed posttreatment or follow-up outcomes using one or more measures of the aforementioned disorders (Weisz, Kuppens, et al., 2017; Weisz et al., 2019). Study 1 includes the subset of trials that sampled youths for elevated depression symptoms or a diagnosis of a depressive disorder, tested CBT, and included at least one continuous measure each of depression symptoms and a CM at posttreatment.

To assemble the larger database, we conducted literature searches for RCTs published from 1963 through September 2020 in English-language peer-reviewed journals. We searched APA PsycInfo using a set of 21 psychotherapy-related terms (e.g., psychother-, counseling) and Pubmed using the Medical Subject Headings indexing system in the latter, setting limits for clinical or outcome assessment, child and adolescent age group, human subjects, and published in English. All study abstracts were reviewed by one research assistant; potentially eligible studies had their full texts reviewed by two research assistants independently to identify outcome articles meeting our inclusion criteria. They resolved differences via discussion or consultation with a third research assistant. For the present meta-analysis, we reviewed recent meta-analyses (e.g., Cuijpers et al., 2020; Eckshtain et al., 2020) for any potentially eligible RCTs and also conducted supplementary searches for secondary analysis articles related to the identified outcome articles. Specifically, we used APA PsycInfo and Pubmed to search for articles that cited the outcome articles and articles authored by the outcome articles' first or second author and published the same year as, or after, the earliest outcome article. In addition, we requested information and data about depression symptom outcomes and CMs from the authors of outcome articles and from the NIMH data repositories. Finally, we retrieved dissertations or theses from the authors, university libraries, or ProQuest Dissertations and Theses electronic database. Figure 2 displays a flowchart documenting these search procedures.

Figure 1
Two-Wave Model of Treatment Mediation

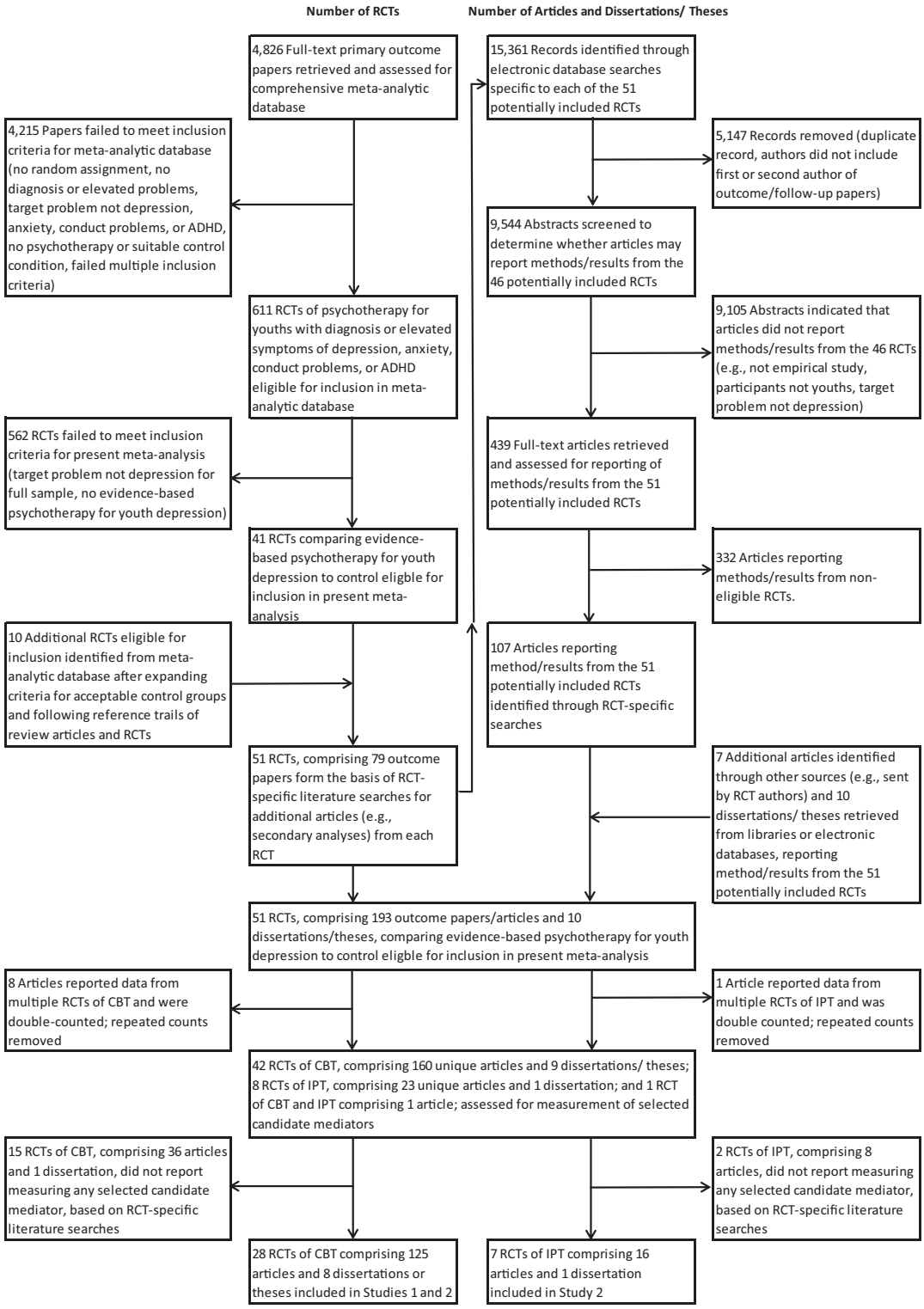


Study Characteristics, Mediation Model, and Candidate Mediators

Study characteristics were coded for the existing meta-analytic database by eight postdoctoral or doctoral psychology trainees who had established good intercoder reliability with a sample of 20–30 RCTs. We used the variables, target problem (kappa, $\kappa = .89$), and treatment/control condition ($\kappa = .83$) to identify RCTs targeting depression and comparing CBT to control for inclusion. Other study characteristics relevant to the present meta-analysis include mean participant age (ICC = .99); percent male/female (ICC = .95); percent Caucasian, African American, Latino, Asian, Native American, other, and unknown race/ethnicity (mean ICC = .80); and diagnosis of depressive disorder required ($\kappa = .79$). CMs and outcome measures were coded for the present meta-analysis by three research assistants who had established good intercoder reliability with a sample of 12 RCTs. They coded each measure as a depressive or internalizing symptom outcome, or one of 26 CM categories ($\kappa = .91$); administration at pre-, mid-, and posttreatment and follow-up time point (mean $\kappa = .82$); and source as the youth, caregiver, sibling, peer, teacher, therapist, researcher, or life-event data ($\kappa = .89$).

Figure 2

Flowchart of the Numbers of RCTs, Articles, and Dissertations/Theses Retrieved, Included, and Excluded



Note. RCT = randomized controlled trial; ADHD = attention-deficit hyperactivity disorder; CBT = cognitive behavioral therapy; IPT = interpersonal therapy.

Our mediation model and selection of CMs were based in part on the availability of data. CMs were measured commonly at pre- (100.0%) and posttreatment (97.0%) and less frequently during treatment (24.2%) and follow-up (63.6%). Thus, we employed a mediation model that tested whether posttreatment levels of the CM mediated the effects of CBT on posttreatment symptom outcomes, controlling for pretreatment levels of the CM and symptoms (Figure 1). This approach is similar to assessing whether differential change in CM due to treatment mediates change in symptoms, but avoids several problems of computing pre–post change scores for analyses (e.g., change scores are often unreliable; see MacKinnon, 2008). To obtain minimally sufficient data for this mediation model, we followed Gu et al.'s (2015) precedent and selected CMs measured at pre- and posttreatment in at least three RCTs.

Of the 26 CMs coded, seven met these criteria: (a) negative cognition (e.g., cognitive distortion, dysfunctional attitudes, pessimistic attributions, external locus of control, hopelessness, low self-esteem), (b) social engagement (e.g., social support, adjustment, skills, impairment, or communication, loneliness, peer/dating relationship satisfaction), (c) family functioning (e.g., family or parent–youth communication, conflict, cohesion, involvement, or relationship satisfaction, parenting behavior), (d) problem solving (e.g., engagement in active or primary control coping, i.e., changing external conditions to deal with stress, defining problems, generating solutions, family problem solving, action stage of change), (e) reframing (e.g., engagement in secondary control coping, i.e., changing thinking or feelings to deal with stress, reappraisal, positive attitudes or orientation toward problems), (f) avoidance (e.g., avoiding, denying, or distancing from the problem/stress, wishful thinking, precontemplation stage of change), and (g) pleasant activities (e.g., scheduling or engagement in enjoyable, relaxing, or productive activities). Further details about the creation of these CM categories are provided in the supplemental material.

Effect Size Extraction and Computation

We coded statistics quantifying the bivariate relationship between each pair of variables in Figure 1. For the treatment–CM and treatment–outcome relationship, we coded the mean score, standard deviation, and sample size of the treatment and control groups at pre- and posttreatment. For mediator–outcome, mediator–mediator, and outcome–outcome relationships, we coded zero-order Pearson's product–moment correlations (r) and the number of participants (n) associated with each correlation. Each correlation was extracted by one coder and then checked by another coder from articles, dissertations, theses; or materials provided directly by RCT authors; or computed from de-identified data sets sent by authors. When correlations or data sets were not provided by authors, we also searched for but did not find other statistics that could be converted to correlations using formulae detailed in Lipsey and Wilson (2001). When the only information we had about the relationship between two variables was that it is nonsignificant in a bivariate analysis, we assigned $r = 0$ (Smith, 1980); this occurred for four correlations (all from Reed, 1994, treatment effects on 3 depression symptom outcomes and 1 CM categorized as social engagement).

To quantify treatment effects on CMs and outcomes, we computed Hedges's g, an estimate of the standardized mean difference effect size corrected for any small sample bias, defined as follows (Borenstein et al., 2009a; Lipsey & Wilson, 2001):

$$g = \left[1 - \frac{3}{4N - 9}\right] \times d, \tag{1}$$

where N is the total sample size. Cohen's d is given by

$$d = \frac{M_t - M_c}{s_{\text{pooled}}}, \tag{2}$$

where M_t is the mean of treatment group, M_c is the mean of control group, and s_{pooled} is the pooled estimate of the population standard deviation, computed as

$$s_{\text{pooled}} = \sqrt{\frac{(n_t - 1)s_t^2 + (n_c - 1)s_c^2}{n_t + n_c - 2}}, \tag{3}$$

where n_t is the sample size of the treatment group, n_c is the sample size of the control group, s_t is the standard deviation of the treatment group, and s_c is the standard deviation of the control group.

We used zero-order correlations, r, to quantify all bivariate relationships in Figure 1. For the a and c paths in Figure 2, we converted g to a point-biserial correlation using the following formulae (Borenstein et al., 2009b):

$$r = \frac{g}{\sqrt{g^2 + a}}, \tag{4}$$

where a is a correction for unequal sample sizes of the treatment and control groups, given by

$$a = \frac{(n_t + n_c)^2}{n_t n_c}. \tag{5}$$

For all other paths in Figure 1, including the b path and the paths involving covariates (i.e., pre- to posttreatment CM, pre- to posttreatment outcome, nondirected path between pretreatment CM and pretreatment outcome), Pearson's product–moment correlations (r) were directly extracted from the data, thus no further conversion was needed before analysis.

While the treatment condition is always coded 1 for the treatment group and 0 for the control group, CMs and outcomes might be scaled by study authors so that higher values are more desirable (e.g., better problem solving, improved social skills) or so that lower values are more desirable (e.g., less frequent negative cognition, lower symptom severity). The direction in which these variables are scaled will have corresponding influence on the algebraic sign of the correlational effect sizes when calculated as described above, which can be misleading with regard to the actual nature of the relationship of the underlying constructs. To avoid confusion, the signs on the correlation coefficients were altered when necessary so that positive correlations always represent more desirable treatment effects on CMs and outcomes. Similarly, the signs on the correlations between CMs and outcomes are adjusted as needed so that they are positive when both variables are scaled in the same direction and negative when one is scaled in the opposite direction from the other.

Effect sizes were computed for each eligible treatment–control pair within each RCT, separately for each CM. RCTs with multiple treatment or control conditions had multiple treatment–control pairs.

Although means and standard deviations of measures were often available for each treatment/control pair, correlations among CMs and outcomes were sometimes available for only the full sample of participants across the multiple treatment/control pairs. In three RCTs (Gillham et al., 2012; Kahn et al., 1990; Stark et al., 1987), we computed a total of 10 full-sample correlations among CMs and outcomes to maximize the use of available data. When data from multiple measures of the same outcome or CM category at the same time point within an RCT were available, we took the mean of those effect sizes and related statistics (e.g., sample sizes, variances) so that each treatment–control pair contributed only one effect size to each bivariate relationship in the mediation model, following other MASEM studies (Carraro & Gaudreau, 2013; Murayama & Elliot, 2012).

Data Analysis

First, we conducted univariate meta-analysis to estimate the mean treatment effect on each of the seven CMs (Path *a*), the mean treatment effect on depression symptom outcomes (Path *c*), and the association between CMs and outcomes at posttreatment (Path *b*) for the subset of CBT trials measuring that CM. We fit random-effects models due to potential heterogeneity in effect sizes stemming from, for example, different forms of CBT and control conditions, samples with different characteristics, or different measures of the same construct. In addition, random-effects models reflect an intent to generate findings generalizable beyond the specific studies analyzed. We weighted the effect sizes with the inverse variance and chose the restricted maximum-likelihood estimator of residual heterogeneity as it is approximately unbiased and efficient (Viechtbauer, 2005). We used the trim-and-fill method (Duval & Tweedie, 2000) to assess the potential impact of publication bias on treatment–CM and treatment–outcome effects, as treatment effects are usually the focus of articles published from RCTs. This method estimates the number of studies that might be missing due to selective reporting of only large or statistically significant findings and further estimates the adjusted mean effect size if those studies had been included. The adjusted mean effect size is not considered a more accurate estimate of the mean effect size but rather a test of how robust the findings are to possible publication bias (Viechtbauer, 2010). These analyses were carried out using the *metafor* package (Viechtbauer, 2010), implemented in *R* Version 4.0.2.

We proceeded to fit the mediation model (Figure 1) only when the mean treatment effect sizes on the CM and outcome were at least 0.10. There is no clear empirical basis for selecting a cutoff, but we consider 0.10 to be the smallest potentially meaningful treatment effect, given the relatively small statistical effects of psychotherapy on depression outcomes (mean effect size of 0.36 reported by Eckshtain et al., 2020). We avoided using a cutoff based on statistical significance due to its dependence on sample size. We employed a cutoff because a very small treatment effect on the CM (Path *a* in Figure 1) makes it unlikely that the CM would have enough influence to mediate the treatment outcome. Additionally, a very small treatment effect on the outcome (Path *c* in Figure 1) indicates that there is very little treatment-induced change for the CM to mediate.

For each CM that met the cutoff, we conducted two-stage structural equation modeling (TSSEM; Cheung, 2014; Cheung & Chan, 2005). In Stage 1, a pooled correlation matrix was synthesized from the individual RCT correlation matrices and participant sample sizes (i.e., the maximum *N* across all cells of each matrix) using a random-effects model. In Stage 2, the pooled correlation matrix elements were weighted by their precision using a weighted least squares approach and a structural model (Figure 1) was fit to the weighted matrix. There are several advantages to using TSSEM to conduct MASEM. TSSEM handles dependency in correlations across cells in the matrix (i.e., when multiple correlations of different variable pairs come from the same RCT) and synthesizes correlation matrices with missing data (i.e., when RCTs collect or report data on only some of the relationships in the model; Cheung & Chan, 2005). Incomplete correlation matrices were included in the present analyses to fully exploit existing data, with maximum-likelihood estimates provided by TSSEM for the missing data. In addition, the participant sample size is likely to vary across correlations in the pooled matrix because some studies did not have data for all relationships in the model. TSSEM uses the total sample size (i.e., the sum of participants across all studies), which eliminates ambiguity and arbitrary decisions about choice of sample size (e.g., arithmetic mean, harmonic mean, median) for analyses (Cheung & Chan, 2005). TSSEM was conducted using the *metaSEM* package (Cheung, 2015), which is based on *OpenMx* (Boker et al., 2011) and implemented in *R* Version 4.0.2.

Random-effects models (Cheung, 2014) were fit in both stages whenever possible. However, very small heterogeneity variances ($\tau^2 < 1 \times 10^{-8}$) were generated for some correlations, leading to estimation problems for some of the mediation models, especially those with fewer studies. In those instances, we fixed τ^2 to zero, thus essentially conducting fixed effects analyses for these highly similar correlations while allowing larger heterogeneity variances to vary in a user-defined, modified random-effects model (Cheung, 2017). If estimation problems did not occur, then heterogeneity variances were not fixed to zero, even if they were very small. The hypothesized mediation models were fit to each pooled correlation matrix, with tests of direct and indirect effects specified. The parameter estimates obtained are the standardized regression coefficients, with their 95% confidence intervals, for the relationships between each pair of variables, adjusted for the influence of other variables in the model.

We conducted sensitivity analyses to evaluate the impact of statistical dependency due to multiple treatment–control pairs from the same RCT and due to missing data. We repeated both univariate meta-analysis and MASEM while including only one treatment–control pair per RCT and compared the results to the original analyses. We selected the pair with the treatment or control condition that was more frequent among the included RCTs. For example, when group CBT and group CBT plus a parent component were tested in the same RCT, we chose the group CBT–control pair because group CBT was a more common treatment condition among the included RCTs. Similarly, we chose waitlist controls over attention placebo controls because waitlist was more common. As a further sensitivity test, we repeated the MASEM analyses with those same treatment–control pairs but with only complete matrices (i.e., those with no missing data), recognizing that the TSSEM maximum-likelihood estimation is influenced by the amount and nature of available and missing data.

Transparency and Openness

This meta-analysis was not preregistered. The codebook, data, and syntax used are available at https://osf.io/xpku2/.

Study 1: Results

The pool of RCTs in Study 1 included 28 trials that tested CBT against a control condition for youth depression. The publication years of all included articles and dissertation/theses ranged from 1982 to 2020; however, the primary outcome articles were published between 1986 and 2016. To our surprise, no primary outcome articles published after 2016 met the inclusion criteria. Six of the 28 trials contributed two treatment–control comparisons to analyses. The pooled sample of 3,521 youths had a mean age of 13.9 years (SD = 2.2) and included more adolescents than children—60.7% of samples had mean age ≥13.0 years. On average, nearly half the youths in the respective samples were male (M = 45.0%, SD = 11.8) and Caucasian (M = 51.3%, SD = 34.5); the remainder were Asian/Pacific Islander (M = 10.2%, SD = 23.5), Latino (M = 9.9%, SD = 21.8), African American (M = 7.5%, SD = 13.2), Native American (M = 0.4%, SD = 1.9), other (e.g., multiracial, South African, M = 4.2%, SD = 6.7), or unknown race/ethnicity (M = 16.4%, SD = 35.1). A diagnosis of depressive disorder was required in 28.6% of CBT trials. A table of CBT trials and their characteristics is presented in Table 1, and the full references of articles, dissertations, and theses reporting study methods and data are listed in Appendix A.

Negative Cognition in CBT

Analyses for the negative cognition CM synthesized data from 24 RCTs, comprising 3,367 participants assigned to 28 CBT–control comparisons, although data on treatment effects on negative cognition were only available for 27 comparisons due to missing data from one trial (Garber et al., 2009; see supplemental Figure S1, for individual comparison effect sizes). CBT exerted a statistically significant mean effect on negative cognition (mean g = 0.25, 95% CI [0.17, 0.33], $p < .001$, $\tau^2 = 0.002$, $k = 27$, $N = 2{,}678$). This effect appears highly robust to potential publication bias; the trim-and-fill method estimated four missing studies on the left side of the plot and made minimal adjustment to the mean treatment effect on negative cognition (0.22, 95% CI [0.14, 0.31], $p < .001$, $\tau^2 = 0.005$, $k = 31$). Sensitivity analyses using only the 23 independent comparisons (Garber et al., 2009, was excluded due to missing data) also generated a similar mean effect size (g = 0.27, 95% CI [0.17, 0.37], $p < .001$, $\tau^2 = 0.008$, $k = 23$, $N = 2{,}050$). The mean effect of CBT on outcome (i.e., depression symptoms) was also statistically significant when all 28 comparisons were pooled (g = 0.30, 95% CI [0.20, 0.40], $p < .001$, $\tau^2 = 0.024$, $k = 28$, $N = 3{,}127$). This effect appears to be somewhat vulnerable to publication bias; the trim-and-fill method estimated nine missing studies on the left side of the plot and produced a smaller adjusted mean treatment effect size (g = 0.19, 95% CI [0.05, 0.32], p = .008, τ^2 = 0.101, k = 37). Sensitivity analyses using only the 24 independent comparisons generated a mean effect size similar to the treatment effect before adjustment (g = 0.30, 95% CI [0.20, 0.40], $p < .001$, $\tau^2 = 0.013$, $k = 24$, N = 2,428). There was a positive correlation between negative cognition and outcome (r = 0.43, 95% CI [0.35, 0.51], $p < .001$, $\tau^2 = 0.022$, $k = 18$ available comparisons, N = 1,634; r = 0.44, 95% CI [0.35, 0.53], $p < .001$, $\tau^2 = 0.021$, $k = 15$ independent comparisons, N = 1,251), indicating that more desirable (i.e., less frequent or extensive) levels of negative cognition were associated with better outcome. Table 2 displays the key effect size estimates.

Because treatment effects on both negative cognition and depression outcomes were greater than 0.10, we proceeded to MASEM for the 28 comparisons (N = 3,367). Stage 1 analyses indicated significant between-study heterogeneity, $Q(210)$ = 289.64, $p < .001$, with the amount of heterogeneity varying across paths in the mediation model (τ^2 range = 1.171×10^{-10}—.012, Mdn = .001). The Stage 2 analyses found that all the paths in the mediational model were significantly greater than zero, including the conjoint mediation pathway critical to mediation: CBT improved negative cognition compared to control conditions, which predicted improvement in depression symptoms at posttreatment beyond the effect of treatment (see Figure 3a). The indirect effect of treatment condition on depression outcome via change in negative cognition was statistically significant (Path ab β = .030, 95% CI [.019, .043]), indicating a mediational relationship for negative cognition. The indirect effect remained significant (Path ab β = .136, 95% CI [.088, .186]) in sensitivity analyses conducted with 15 independent, complete matrices (N = 1,432; nine of the 24 independent matrices had missing data; see Figure 3a).

Social Engagement in CBT

Analyses for the social engagement CM synthesized data from 14 RCTs, comprising 2,020 participants assigned to 18 CBT–control comparisons, although treatment effects on social engagement were only available for 15 comparisons due to missing data from three trials (Asarnow et al., 2002, 2005; Reed, 1994; see supplemental Figure S2, for individual comparison effect sizes). CBT did not produce greater improvement than control on social engagement (mean g = 0.07, 95% CI [−0.03, 0.18], p = .177, $\tau^2 = 0.001$, k = 15, N = 1,407). This finding was replicated using the trim-and-fill method (g = 0.001, 95% CI [−0.12, 0.12], p = .984, $\tau^2 = 0.017$, k = 19), which estimated four missing studies on the left side of the plot. Sensitivity analyses using only the 11 independent comparisons (the three trials with missing data were excluded) generated a slightly larger effect size for the social engagement effect that still fell short of statistical significance (g = 0.11, 95% CI [−0.03, 0.25], p = .118, τ^2 = 0.005, k = 11, N = 961). The effect of CBT on depression outcome remained statistically significant for the 18 comparisons (g = 0.30, 95% CI [0.17, 0.44], $p < .001$, $\tau^2 = 0.029$, k = 18, N = 1,863) and appears to be somewhat vulnerable to publication bias. The trim-and-fill method estimated six missing studies on the left side of the plot and reduced the mean treatment effect (g = 0.18, 95% CI [0.02, 0.35], p = .032, τ^2 = 0.095, k = 24). Sensitivity analyses using only the 14 independent matrices generated an effect similar to that before adjustment (g = 0.29, 95% CI [0.16, 0.42], $p < .001$, $\tau^2 = 0.012$, k = 14, N = 1,388). There was a positive correlation between social engagement and outcome (r = 0.22, 95% CI [0.11, 0.33], $p < .001$, $\tau^2 = 0.022$, k = 11 available comparisons, N = 1,220; r = 0.20, 95% CI [0.06, 0.34], p = .006, $\tau^2 = 0.029$, k = 8 independent comparisons, N = 829), indicating that more desirable (i.e., more frequent and competent) levels of social engagement were associated with better outcome. Table 2 displays key effect size estimates.

Table 1
Characteristics of 28 Randomized Controlled Trials of Cognitive Behavioral Therapy for Youth Depression and Measures of Candidate Mediators Examined in the Present Meta-Analysis

Primary outcome article	*N*	M_{age} (y)	Diagnosis required?	EST	Control	Candidate mediators
Ackerson et al. (1998)	30	15.9	No	CBT bibliotherapy	Waitlist	COG: ATQ; DAS
Asarnow, Jaycox, Duan, LaBorde, Rea, Tang, et al. (2005)	418	17.2	No	CBT individual (enhanced collaborative primary care)	Usual care (in primary care + brief provider training)	SOC: Number of friends; satisfaction with relationship with friends FAM: Satisfaction with relationship with parents
Asarnow et al. (2002)	23	10.0	No	CBT group	Waitlist	COG: ATQ—negative thoughts, positive thoughts; SPPC—scholastic competence, behavioral conduct, global self-worth, social acceptance SOC: Children's Self-Efficacy for Peer Interaction PRO: Self-Report Coping Scale—problem solving AVO: Self-Report Coping Scale—distancing
Brent et al. (1997)	72	15.6	Yes, MDD	CBT individual	Nondirective supportive therapy	COG: BHS; CNCEQ FAM: FAD—affective involvement parent, affective involvement self, affective responsiveness parent, affective responsiveness self, behavioral control parent, behavioral control parent self, communication parent, communication self, general functioning parent, general functioning self, roles parent, roles self; FICS—adolescent involvement, dyadic conflict PRO: FAD—problem solving parent, problem solving self; FICS—adolescent problem solving
De Cuyper et al. (2004)	22	10.0	No	CBT group	Waitlist	COG: SPPC (Dutch)—athletic competence, behavioral conduct, global self-worth, physical appearance, scholastic competence, social acceptance
Fleming et al. (2012)	32	14.9	No	CBT computerized	Waitlist	COG: Children's Noicki-Strickland Internal–External Control Scale; HSC
Garber et al. (2009)	316	14.8	No	CBT group + usual care	Usual care (allowed to seek nonstudy services)	COG: BHS FAM: CRPBI; CBQ
Gillham et al. (2012)	408	12.5	No	1. CBT group 2. CBT group + parent group	Usual care ("school-as-usual")	COG: CASQ—negative events; HSC FAM: CRPBI—psychological control versus autonomy PRO: Children's Coping Strategies Checklist–Revision 1 active coping scale
Gillham et al. (2006)	271	11.5	No	CBT group	Usual care (unspecified)	COG: CASQ—negative events, positive events
Kahn et al. (1990)	34	12.1	No	CBT group + parent group	Waitlist	COG: Piers-Harris Children's Self-Concept Scale

(*table continues*)

Table 1 (*continued*)

Primary outcome article	*N*	M_{age} (y)	Diagnosis required?	EST	Control	Candidate mediators
Lewinsohn et al. (1990)	74	16.2	Yes, MDD, mDD, or IDD	1. CBT group 2. CBT group + parent group	Waitlist	COG: DAS (abbreviated); Personal Beliefs Inventory (abbreviated); Perceived Competence Scale SOC: Interpersonal Events Schedule FAM: IC—parent, self REF: Frequency of Self-Reinforcement Attitudes Scale; Subjective Probability Questionnaire (abbreviated) ACT: PES
Liddle and Spence (1990)	31	9.2	No	CBT group	1. Waitlist 2. Attention placebo (group drama program)	SOC: Matson Evaluation of Social Skills for Youngsters–self-report total score, teacher's version PRO: List of social situation problems
McCarty et al. (2013)	120	12.7	No	CBT group	Attention placebo (individual support)	COG: BASC-2—locus of control, self-esteem, sense of inadequacy SOC: BASC-2—interpersonal relations, social skills parent, social skills teacher FAM: BASC-2—relations with parents; parent–child Communication Scale Parent—child emotional expression, child empathy/ listening, parent communication; parent–child Communication Scale Self—child communication, conflict, parent communication
Merry et al. (2012)	187	15.6	No	CBT computerized	Usual care (services at youth clinics, schools, or primary care)	COG: HSC
Reed (1994)	18	16.5	Yes, MDD or DYS	CBT group	Attention placebo (art and imagery exercises, creative and verbal expression)	COG: CSEI SOC: Personality Inventory for Children—social skills
Reynolds and Coats (1986)	19	15.7	No	CBT group	Waitlist	COG: Academic Self-Concept Scale–high school version; RSES
Rohde et al. (2004)	93	15.1	Yes, MDD + conduct disorder	CBT group	Case management	COG: ATQ; BHS; DAS SOC: PES—social skills; SAS-SR FAM: Family Environment Scale—family cohesion; IC REF: Coping skills ACT: PES—pleasant activities, relaxation
Rohde et al. (2014)	378	15.5	No	1. CBT group 2. CBT bibliotherapy	No treatment (educational brochure)	COG: ACSQ (short form) SOC: NRI—peer; SAS-SR FAM: NRI—parent

(*table continues*)

Table 1 (*continued*)

Primary outcome article	*N*	M_{age} (y)	Diagnosis required?	EST	Control	Candidate mediators
Rosselló and Bernal (1999)	48	14.7	Yes, MDD or DYS	CBT individual (also tested IPT individual)	Waitlist	COG: Piers-Harris Children's Self-Concept Scale SOC: CBCL—social abilities adolescent, social abilities parent; Social Adjustment Scale for Children and Adolescents FAM: FEICS—intensity of emotional involvement, perceived criticism
Santomauro et al. (2016)	23	15.8	No, but autism spectrum disorder was required	CBT group	Waitlist	SOC: Asperger Syndrome Diagnostic Interview—social interaction impairments; Australian Scale for Autism Spectrum Conditions—difficulty with social communication; Autism Spectrum Quotient—social skills REF: Emotional Regulation Questionnaire—reappraisal
Stark et al. (1987)	19	11.2	No	CBT group	Waitlist	COG: CSEI SOC: CBCL—social withdrawal parent
Stasiak et al. (2014)	34	15.2	No	CBT computerized	Attention placebo (computerized psychoeducation)	PRO: ACS—problem solving, reference to others AVO: ACS—nonproductive coping
Stice et al. (2008)	253	15.6	No	1. CBT group 2. CBT bibliotherapy	No treatment (education brochure)	COG: ACSQ; ATQ; BHS; RSES SOC: Loneliness Scale; Network of Relationships Inventory—friend social support; SAS-SR FAM: Network of Relationships Inventory—parental social support ACT: PES (abbreviated)
Szigethy et al. (2007)	41	15.0	No	CBT individual	Waitlist	COG: CASQ; Perceived Control Scale for Children FAM: Child FACES Family Score—cohesion + adaptability; Parent FACES Family Score—cohesion + adaptability
Treatment for Adolescents with Depression Study Team (2004)	223	14.6	Yes, MDD	CBT individual	Pill placebo	COG: BHS; CASQ; CNCEQ; CPRS-R—perfectionism; Cognitive Triad Inventory for Children—views about the self, views about the world; DAS; SPSI-R—negative problem orientation SOC: CPRS-R—Social Problems subscale FAM: CBQ—perception of dyad conflict parent, perception of dyad conflict self; Conners-Wells' Adolescent Self-Report Scale (long)—family problems; FAM-III—general child, general parent; IC—child interactions with parents, IC—parent interactions with teen PRO: SPSI-R—rational problem solving; SOCQ—action REF: SPSI-R—positive problem orientation AVO: SPSI-R—avoidant coping; SOCQ—precontemplation

(*table continues*)

Table 1 (*continued*)

Primary outcome article	*N*	M_{age} (y)	Diagnosis required?	EST	Control	Candidate mediators
Vostanis et al. (1996b)	57	12.7	Yes, MDD, mDD, or DYS	CBT individual	Attention placebo	COG: Self-Esteem Inventory—parent, self SOC: SAICA—peers, schooling FAM: SAICA—family life ACT: SAICA—spare time
Weisz et al. (2009)	57	11.8	Yes, MDD, mDD, or DYS	CBT individual	Usual care	COG: Perceived Control Scale for Children PRO: RSQ (modified)—primary control engagement coping youth, primary control engagement coping parent REF: RSQ (modified)—secondary control engagement coping youth, secondary control engagement coping parent; PASCET skills—cognitive skills youth, cognitive skills parent AVO: RSQ (modified)—disengagement coping youth, disengagement coping parent; PASCET skills—avoidance youth, avoidance parent ACT: PASCET skills—behavioral activation youth, behavioral activation parent
Yu and Seligman (2002)	220	11.8	No	CBT group	Waitlist	COG: CASQ

Note. y = years; ACT = pleasant activities; ACS = General Short Form of the Adolescent Coping Scale; ACSQ = Adolescent Cognitive Style Questionnaire; ATQ = Automatic Thoughts Questionnaire; AVO = avoidance; BASC-2 = Behavior Assessment Scale for Children–Second Edition; BHS = Beck Hopelessness Scale; CASQ = Children's Attributional Style Questionnaire; FACES = Family Adaptability and Cohesion Evaluation Scale; CNCEQ = Children's Negative Cognitive Error Questionnaire; CBCL = Child Behavior Checklist; CBQ = Conflict Behavior Questionnaire; CBT = cognitive behavioral therapy; COG = negative cognition; CRPBI = Children's Report of Parenting Behavior Inventory; CPRS-R = Connors Parent Rating Scale–Revised; CSEI = Coopersmith Self-Esteem Inventory; DAS = Dysfunctional Attitude Scale; DYS = dysthymia; EST = empirically supported treatment; FAD = Family Assessment Device; FAM = family functioning; FAM-III = Family Assessment Measure–III; FAD = Family Assessment Device; FEICS = The Family Emotional Involvement and Criticism Scale; FICS = Family Interaction Coding System; HSC = Hopelessness Scale for Children; IC = Issues Checklist; IDD = intermittent depressive disorder; IPT = interpersonal psychotherapy; MDD = major depressive disorder; mDD = minor depressive disorder; RSQ = Responses to Stress Questionnaire; NRI = Network of Relationships Inventory; PASCET = Primary and secondary control enhancement training; PES = Pleasant Events Schedule; RSES = Rosenberg Self-Esteem Scale; PRO = problem solving; REF = reframing; SAICA = Social Adjustment Inventory for Children and Adolescents; SAS-SR = Social Adjustment Scale–Self-Report; SOC = social engagement; SOCQ = Stage-of-Change Questionnaire; SPSI-R = Social Problem-Solving Inventory–Revised; SPPC = Self-Perception Profile for Children.

Table 2

Mean Effect Sizes of Mediational Pathways and Significance of Mediational Test for Candidate Mediators of Cognitive Behavioral Therapy for Youth Depression

Candidate mediator	No. of comparisons (RCTs)	Path *a, r* (*g*)	Path *c, r* (*g*)	Path *b, r*	Test mediation?	Mediation significant?
Negative cognition	28 (24)	0.14 (0.25)***	0.18 (0.30)***	0.43***	Yes	Yes
Social engagement	18 (14)	0.04 (0.07)	0.17 (0.30)***	0.22***	No, Path $a < 0.10$	
Independent comparisons[a]	14 (14)	0.06 (0.11)	0.15 (0.29)***	0.20**	Yes	No
Family functioning	17 (13)	<|0.01| (0.01)	0.15 (0.27)***	0.24***	No, Path $a < 0.10$	
Problem solving	9 (7)	0.03 (0.06)	0.08 (0.16)*	0.16**	No, Path $a < 0.10$	
Pleasant activities	7 (5)	0.06 (0.12)	0.24 (0.46)***	0.23***	Yes	No
Independent comparisons[b]	5 (5)	0.09 (0.19)	0.21 (0.43)***	0.23***	Yes	Yes
Reframing	6 (5)	−0.01 (−0.02)	0.20 (0.40)[c]	0.16**	No, Path $a < 0.10$	
Avoidance	4 (3)	<|0.01| (<|0.01|)	0.04 (0.07)	0.29***	No, Path $a < 0.10$	

Note. To facilitate comparison across paths, all effect sizes are reported as correlations, *r*. Because treatment effects are conventionally synthesized as standardized mean differences, Hedges's *g* values are also reported in parentheses for treatment effects on the candidate mediator (Path *a*) and on the outcome (Path *c*). When the mean effect size for both Paths *a* and *c* were at least $g = 0.10$ (regardless of statistical significance, which varies with sample size) for a particular candidate mediator, we proceeded to test for mediation. These cutoffs are what we consider to be the smallest potentially meaningful treatment effect. Results are reported for the full sample of treatment–control comparisons available for each candidate mediator unless otherwise specified. RCT = randomized controlled trial.

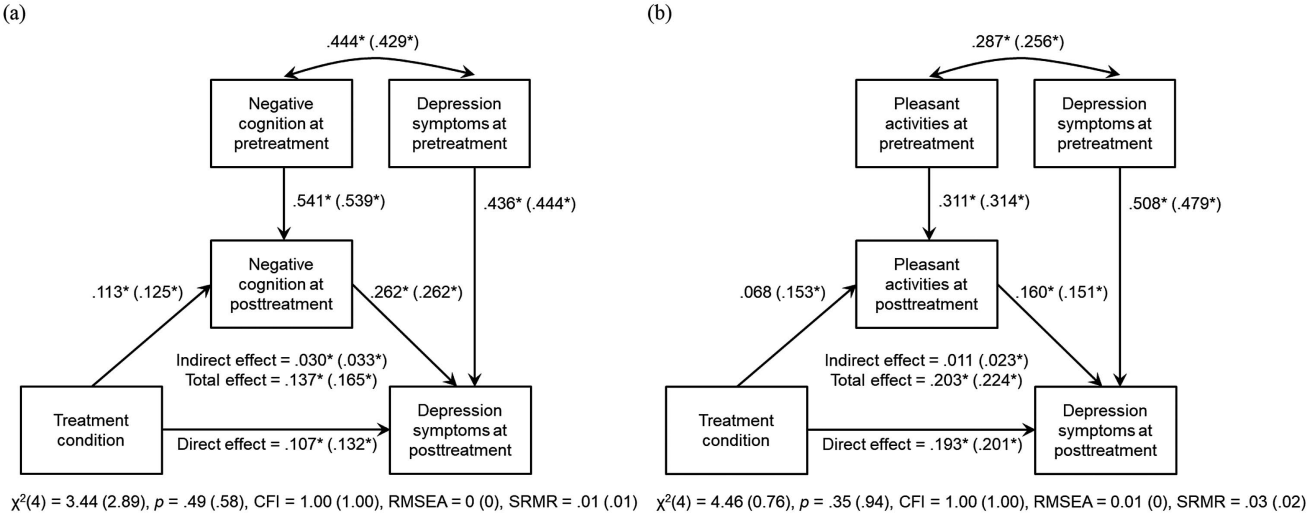
[a] For social engagement, the cutoffs were met only for the subsample of independent comparisons (selecting only one out of multiple treatment–control comparisons within a randomized trial), thus results are reported for both this subsample and the full sample of comparisons. [b] For pleasant activities, the cutoffs were met for the full sample of comparisons and the subsample of independent comparisons, but mediation was significant only in the subsample, thus results are reported for both the subset and the full sample of comparisons. [c] For reframing, Path *c* was nonsignificant ($p > .05$) when estimated from *g*; when converted into *r*, Path *c* became significant ($p < .05$). For all other paths and all other candidate mediators, the level of significance remained unchanged upon conversion from *g* to *r*.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Because treatment effects on both social engagement and outcome were greater than 0.10 for the subset of 14 independent matrices ($N = 1{,}543$), we proceeded to MASEM for this subsample. Stage 1 analyses indicated nonsignificant between-study heterogeneity, $Q(88) = 108.79$, $p = .066$, and little variation in the amount of heterogeneity across paths in the mediation model (τ^2 range = 9.330×10^{-10}—.009, *Mdn* = .001). Due to estimation problems in Stage 1 analyses, the four τ^2 that were near 0 ($< 1 \times 10^{-8}$) were fixed to 0 in a user-defined modified random-effects model, and Stage 1 analyses were repeated

Figure 3

Significant Mediation Tests of the Effects of Cognitive Behavioral Therapy on Posttreatment Depressive Symptoms via Negative Cognition and Pleasant Activities in Meta-Analytic Structural Equation Models



Note. Path estimates and direct, indirect, and total effects for each candidate mediator are displayed for two overlapping study samples: a larger sample of complete matrices and incomplete matrices (i.e., with missing data), some of which may be dependent (i.e., come from the same randomized trial); and a smaller subsample of only complete and independent matrices used in sensitivity analyses shown in parentheses. The (a) negative cognition model was fitted to 28 matrices (15 independent, complete matrices); (b) the pleasant activities model fitted to seven matrices (3 independent, complete matrices). CFI = comparative fit index; RMSEA = root-mean-square error of approximation; SRMR = standardized root-mean-square residual.

* Estimates are significantly different from zero (i.e., 95% likelihood-based confidence intervals do not contain zero).

before proceeding to Stage 2. Stage 2 analyses showed that CBT significantly improved depression symptoms but not social engagement at posttreatment relative to control conditions, contributing to a nonsignificant indirect effect (Path *ab*; β = .007, 95% CI [−.0002, .018]). The indirect effect remained nonsignificant (Path *ab* β = .007, 95% CI [−.001, .019]) in sensitivity analyses conducted with eight independent, complete matrices ($N = 988$; 6 of the 14 independent matrices had missing data). These findings thus provide no evidence that social engagement is an important mediator of CBT effects on depression outcomes.

Family Functioning in CBT

Analyses for the family functioning CM synthesized data from 13 RCTs, comprising 2,865 participants assigned to 17 CBT–control comparisons, although data on treatment effects on family functioning were only available for 13 comparisons due to missing data for four comparisons from three trials (Asarnow, Jaycox, Duan, LaBorde, Rea, Tang, et al., 2005; Garber et al., 2009; Gillham et al., 2012; see supplemental Figure S3, for individual comparison effect sizes). CBT had minimal effect on family functioning (mean $g = 0.01$, 95% CI [−0.10, 0.11], $p = .894$, $\tau^2 < 0.001$, $k = 13$, $N = 1{,}376$), a finding replicated using the trim-and-fill method ($g = -0.02$, 95% CI [−0.12, 0.08], $p = .716$, $\tau^2 < 0.001$, $k = 16$), which estimated three missing studies on the left side of the plot. The sensitivity analyses using only the 10 independent comparisons (the 3 trials with missing data were excluded) generated a slightly larger effect size ($g = 0.03$, 95% CI [−0.10, 0.15], $p = .671$, $\tau^2 < 0.001$, $k = 10$, $N = 972$). The mean effect of CBT on depression outcome remained statistically significant for the 17 comparisons ($g = 0.27$, 95% CI [0.17, 0.37], $p < .001$, $\tau^2 = 0.015$, $k = 17$, $N = 2{,}638$). This effect appears to be somewhat vulnerable to publication bias; the trim-and-fill method estimated six missing studies on the left side of the plot and reduced the mean treatment effect ($g = 0.18$, 95% CI [0.06, 0.31], $p < .001$, $\tau^2 = 0.053$, $k = 23$). Sensitivity analyses using only the 13 independent comparisons generated a similar effect to that before adjustment ($g = 0.28$, 95% CI [0.19, 0.38], $p < .001$, $\tau^2 = 0.003$, $k = 13$, $N = 1{,}939$). There was a positive correlation between family functioning and outcome ($r = 0.24$, 95% CI [0.13, 0.34], $p < .001$, $\tau^2 = 0.021$, $k = 12$ available comparisons, $N = 1{,}284$; $r = 0.22$, 95% CI [0.08, 0.36], $p = .002$, $\tau^2 = 0.031$, $k = 9$ independent comparisons, $N = 888$), indicating that more desirable (i.e., less conflictual, more cohesive) levels of family functioning were associated with better outcome. Table 2 displays key effect size estimates. Because the treatment effect on family functioning was smaller than 0.10, we did not proceed to MASEM.

Problem Solving in CBT

Analyses for the problem solving CM synthesized data from seven RCTs, comprising 998 participants assigned to nine CBT–control comparisons, although data on treatment effects on problem solving were only available for eight comparisons due to missing data from one trial (Asarnow et al., 2002; see supplemental Figure S4 for individual comparison effect sizes). CBT had little effect on problem solving (mean $g = 0.06$, 95% CI [−0.07, 0.20], $p = .353$, $\tau^2 < 0.001$, $k = 8$, $N = 844$), a finding replicated using the trim-and-fill method ($g = 0.05$, 95% CI [−0.08, 0.18], $p = .466$, $\tau^2 < 0.001$, $k = 9$), which estimated one missing study on the left side of the plot. The sensitivity analyses using only the six independent matrices ($g = 0.06$, 95% CI [−0.10, 0.23], $p = .460$, $\tau^2 < 0.001$, $k = 6$, $N = 579$) also produced nonsignificant effect sizes. The effect of CBT on depression outcome remained statistically significant for the nine comparisons ($g = 0.16$, 95% CI [0.03, 0.29], $p = .018$, $\tau^2 < 0.001$, $k = 9$, $N = 879$). This effect appears to be vulnerable to publication bias and influenced by dependent matrices. The trim-and-fill method estimated four missing studies on the left side of the plot and reduced the mean treatment effect ($g = 0.12$, 95% CI [−0.01, 0.24], $\tau^2 < 0.001$, $k = 13$), rendering it no longer significant ($p = .068$). Sensitivity analyses using only the seven independent comparisons generated an effect size smaller than the one before adjustment ($g = 0.17$, 95% CI [0.02, 0.33], $\tau^2 < 0.001$, $k = 7$, $N = 613$) but that remains significant ($p = .032$). There was a positive correlation between problem solving and outcome ($r = 0.16$, 95% CI [0.05, 0.27], $p = .004$, $\tau^2 < 0.001$, $k = 4$ available, independent comparisons, $N = 313$), indicating that more desirable (i.e., more skilled and frequent) levels of problem solving were associated with better outcome. Table 2 displays key effect size estimates. Because the treatment effect on problem solving was smaller than 0.10, we did not proceed to MASEM.

Pleasant Activities in CBT

Analyses for the pleasant activities CM synthesized data from five RCTs, comprising 645 participants assigned to seven CBT–control comparisons (see supplemental Figure S5, for individual comparison effect sizes). CBT had a nonsignificant effect on pleasant activity engagement ($g = 0.12$, 95% CI [−0.09, 0.34], $p = .255$, $\tau^2 = 0.030$, $k = 7$, $N = 605$). This effect appears to be vulnerable to publication bias; the trim-and-fill method estimated one missing study on the left side of the plot, resulting in a slightly smaller adjusted mean treatment effect ($g = 0.09$, 95% CI [−0.12, 0.29], $p = .417$, $\tau^2 = 0.032$, $k = 8$). Sensitivity analyses using only the five independent matrices generated a nonsignificant mean effect size on pleasant activities larger than that before adjustment ($g = 0.19$, 95% CI [−0.10, 0.47], $p = .193$, $\tau^2 = 0.046$, $k = 5$, $N = 403$). The mean treatment effect size on depression outcome in the seven comparisons was 0.46 (95% CI [0.22, 0.71], $p < .001$, $\tau^2 = 0.054$, $k = 7$, $N = 605$). This effect appears to be unaffected by publication bias or dependent matrices; the trim-and-fill method estimated no missing studies on the left side of the plot, and sensitivity analyses using only the five independent matrices also generated a similar effect size ($g = 0.43$, 95% CI [0.21, 0.66], $p < .001$, $\tau^2 = 0.011$, $k = 5$, $N = 403$). There was a positive correlation between pleasant activities and outcome ($r = 0.23$, 95% CI [0.13, 0.32], $p < .001$, $\tau^2 < 0.001$, $k = 5$ available comparisons, $N = 396$; and $r = 0.23$, 95% CI [0.11, 0.36], $p < .001$, $\tau^2 < 0.001$, $k = 3$ independent comparisons, $N = 222$), indicating that more desirable (i.e., more frequent) levels of pleasant activity engagement were associated with better outcome. Table 2 displays key effect size estimates.

Because treatment effects on both pleasant activities and outcome were greater than 0.10, we proceeded to MASEM for the seven comparisons ($N = 645$). Stage 1 analyses indicated nonsignificant between-study heterogeneity, $Q(49) = 64.37$, $p = .069$; the

extremely low heterogeneity across paths in the mediation model (τ^2 range = 1.012×10^{-10} – .027, *Mdn* = 1.061×10^{-10}) is likely due to the small sample size. Due to estimation problems in Stage 1 analyses, the six τ^2 that were near 0 ($<1 \times 10^{-8}$) were fixed to 0 in a user-defined modified random-effects model and Stage 1 analyses were repeated before proceeding to Stage 2. Consistent with the univariate analyses, CBT significantly improved depression symptoms, but not pleasant activities at posttreatment relative to control conditions, contributing to a nonsignificant indirect effect (β = .011, 95% CI [−.007, .030]; see Figure 3b). Sensitivity analyses conducted with three independent, complete matrices (N = 270; 2 of the 5 independent matrices had missing data) produced different findings: All model paths were significantly greater than zero, including the effect of CBT on pleasant activities, which contributed to a significant indirect effect (β = .023, 95% CI [.006, .049]; see Figure 3b). The findings for pleasant activities as a mediator were thus inconsistent, with the mediational pathway reaching statistical significance only for the subset of three independent, complete matrices out of the seven available for analysis.

Reframing in CBT

Analyses for the reframing CM synthesized data from five RCTs, comprising 493 participants assigned to six CBT–control comparisons, although data on treatment effects on reframing were only available for five comparisons due to missing data from one trial (Rohde et al., 2004; see supplemental Figure S6, for individual comparison effect sizes). CBT had minimal effect on reframing (mean g = −0.02, 95% CI [−0.24, 0.20], p = .868, $\tau^2 < 0.001$, k = 5, N = 319), unaffected by potential publication bias and dependent matrices: the trim-and-fill method estimated no missing studies on the left side of the plot and sensitivity analyses using only the four independent comparisons also generated a similar effect size (g = −0.01, 95% CI [−0.25, 0.22], p = .910, $\tau^2 < 0.001$, k = 4, N = 281). The effect of CBT on depression outcome was comparable to that found in analyses of other CMs for the six comparisons but did not attain statistical significance, likely due to the smaller sample size in this analysis (mean g = 0.40, 95% CI [−0.002, 0.79], p = .051, τ^2 = 0.158, k = 6, N = 416). This effect appears to be unaffected by potential publication bias; the trim-and-fill method estimated no missing studies on the left side of the plot. Sensitivity analyses using only the five independent comparisons generated a smaller mean treatment effect on outcome (g = 0.17, 95% CI [−0.05, 0.40], τ^2 = 0.008, k = 5, N = 378) that was also nonsignificant (p = .138). There was a positive correlation between reframing and outcome (r = 0.16, 95% CI [0.05, 0.28], p = .004, τ^2 = 0.001, k = 5 available comparisons, N = 313; r = 0.19, 95% CI [0.07, 0.30], p = .002, $\tau^2 <$ 0.001, k = 4 independent comparisons, N = 274), indicating that more desirable (i.e., more frequent and competent) levels of reframing were associated with better outcome. Table 2 displays key effect size estimates. Because the treatment effect on reframing was smaller than 0.10, we did not proceed to MASEM.

Avoidance in CBT

Analyses for the avoidance CM synthesized data from four RCTs, comprising 337 participants assigned to 4 CBT–control comparisons (all independent), although data on treatment effects on avoidance were only available for three comparisons due to missing data from one study (Asarnow et al., 2002; see supplemental Figure S7, for individual comparison effect sizes). CBT had minimal effect on avoidance (g = −0.002, 95% CI [−0.29, 0.28], p = .991, τ^2 = 0.010, k = 3, N = 250). This effect appears to be somewhat vulnerable to publication bias in the opposite direction; the trim-and-fill method estimated two missing studies on the right side of the plot, resulting in a larger, nonsignificant adjusted mean treatment effect (g = 0.12, 95% CI [−0.10, 0.34], p = .268, $\tau^2 < 0.001$, k = 5). CBT also had little effect on depression outcome for these four comparisons (g = 0.07, 95% CI [−0.17, 0.31], p = .559, $\tau^2 < 0.001$, k = 4, N = 279), which appears unaffected by publication bias: the trim-and-fill method estimated two missing studies on the left side of the plot and led to a similar adjusted mean treatment effect (g = 0.02, 95% CI [−0.19, 0.24], p = .836, $\tau^2 < 0.001$, k = 6). There was a positive correlation between avoidance and outcome (r = 0.29, 95% CI [0.18, 0.40], $p < .001$, $\tau^2 < 0.001$, k = 3 available, independent comparisons, N = 250), indicating that more desirable (i.e., less frequent) levels of avoidance were associated with better outcome. Table 2 displays key effect size estimates. Because treatment effect on both avoidance and outcome was smaller than 0.10, we did not proceed to MASEM.

Study 1: Discussion

Perhaps the most important significant findings we expected involved negative cognition: CBT improved negative cognition, which was associated with outcome, and this improvement in negative cognition mediated improvement in outcome. Although researchers have long theorized that CBT reduces depression symptoms via changing negative cognitions, empirical tests of this theory have been plagued by the small number of RCTs with which these tests have been conducted and their small sample sizes, as well as mixed findings within and between RCTs. Our meta-analysis showed that changes in negative cognition do indeed mediate depression symptom reduction, on average, in a pooled sample of over 3,000 youths from 24 RCTs and are thus likely to be reliable. Although there is no established standard for the minimum number of studies required for MASEM, a pooled sample size of at least 1,000 participants across 10 studies is considered desirable (Cheung & Chan, 2005). Moreover, tests of potential publication bias and sensitivity analyses including only independent and complete data comparisons did not substantially change the findings. The significance and the magnitude of the mediation effect provide empirical support for negative cognition as a robust mediator of CBT for youth depression.

Findings were partially consistent with expectations that pleasant activities would play a mediational role. Increasing pleasant activities is a key component of CBT for youth depression; however, youths' participation in pleasant activities was measured with only 600+ youths from five RCTs; significant mediation by improvement in pleasant activities occurred only with a subsample of 270 youths from three independent comparisons with complete data. CBT improved pleasant activities nonsignificantly, and this improvement mediated symptom reduction significantly in the subsample, though not in the full sample. This discrepancy appears to be a result of the Path *a* effect size being nearly twice as large in the subsample as in the full sample. The small sample size calls the reliability of the findings into question and makes it difficult to discern whether differences in treatment delivery format or measure

type may explain discrepant findings. Nevertheless, significant mediation in this small subsample provides preliminary empirical support for pleasant activities as mediator of CBT for youth depression—at least under certain conditions that are to be determined.

For the remaining CMs, the effects of CBT were not only nonsignificant but also modest in magnitude, even though all CMs were significantly associated with depression symptom outcomes. Surprisingly, problem solving and reframing were among these CMs, as both are key intervention targets of CBT for youth depression. Analyses of problem solving were conducted with over 800 youths from seven RCTs, and treatment effects on this outcome were significant for this sample. Moreover, the investigators of two of these trials had found problem solving to be a significant mediator of CBT. However, closer examination of these two RCT findings suggests that their influence on mean effects may have been limited. The first RCT, TADS, found significant mediation in omnibus tests across the four treatment conditions (CBT + antidepressant, antidepressant only, CBT only, and pill placebo) but did not conduct pairwise comparisons for CBT only vs. pill placebo (Lewis et al., 2009), which was the only comparisons included in the present meta-analysis. The second RCT found that CBT improved one of three measures of problem solving (an observational coding measure of adolescent negotiation and compromise to resolve a conflict with their mother), and that this improvement mediated outcome for a subset of youths whose mothers had mild–moderate depression symptoms (Dietz et al., 2014). However, the statistics needed to compute effect sizes were available from the investigators only for the two measures of problem solving (based on parent- and youth-report questionnaires) that did not show improvement by CBT (Kolko et al., 2000; G. Porta, personal communication, December 15, 2016). Taken together, these findings do not support problem solving as a mediator of CBT for youth depression—any possible mediational role may be limited to a specific subgroup of depressed adolescents and a specific measure. The sample for examining treatment effects on reframing was much smaller, with fewer than 400 participants from four RCTs. The effect size of CBT on depression outcome for this sample was comparable to that in the other CMs samples but failed to reach significance, likely due to the smaller sample size. However, the effect size of CBT on reframing was close to zero. These findings do not support reframing as a mediator in CBT for youth depression—this is unexpected given that reframing is thought of as the strategy to reduced negative cognition in CBT. These discrepant findings between reframing and negative cognition are interpreted and discussed further in the general discussion.

More consistent with expectations was the lack of significant treatment effects on social engagement, family functioning, and avoidance. CBT did not improve any of these CMs significantly, and none of these are key intervention targets of CBT protocols for youth depression (see Ng et al., 2016). Although the effect size for social engagement just met our criteria for testing mediation among the subsample of roughly 1,500 youths from 14 RCTs (independent comparisons), this improvement did not mediate symptom reduction significantly, suggesting that social engagement plays a consistently modest role—or no role at all—in symptom reduction across CBT protocols. Similarly, analyses of treatment effects on family functioning were conducted with over 1,000 youths from 13 RCTs, and treatment effects on outcome were significant for this sample, suggesting that CBT protocols do not reduce symptoms by improving family functioning. By contrast, analyses of avoidance were conducted with only 200 plus participants from three RCTs, and the near-zero treatment effect on outcome would have constrained any indirect effects. Thus, the lack of treatment effects of CBT on avoidance should be considered very preliminary and may change with additional studies.

Although there were no treatment or mediation effects of CBT involving several CMs, these CMs may nevertheless mediate outcomes of other therapies developed to target them more directly. All CMs were significantly associated with depression outcomes in the expected direction and, if activated, could potentially lead to symptom improvement. Consistent with the focus of IPT on building social skills and addressing relationship issues, for example, peer functioning was found to be a significant mediator in one IPT trial (Dietz et al., 2015), and both peer and family functioning were significant mediators among Latino adolescents in a second IPT trial (Reyes-Portillo et al., 2017). Further research is needed to ascertain how specific the mediational pathways may be to different intervention modalities. In the present context, for example, we could ask whether the CMs that failed to mediate CBT outcomes might nonetheless still mediate outcomes of other treatments that target them more directly. We have taken an initial step toward answering this question in Study 2.

Study 2: Treatment Specificity of Mediation Effects in CBT and IPT

In Study 1, we examined seven CMs for CBT effects on youth depression with an overall sample of 28 RCTs involving more than 3,000 youths. Relatively robust evidence emerged for the mediational role of negative cognition, and somewhat inconsistent evidence for pleasant activities left room for it as a possible mediator. The lack of treatment effects on the other five CMs, however, gave little indication that any of them mediated CBT effects, though the small number of relevant studies limited statistical power for some of these analyses.

Study 2 is a preliminary attempt to explore the treatment specificity of mediators for interventions targeting youth depression. It would be pertinent to know, for example, whether the mediational role of negative cognition found for CBT also appears for other efficacious treatments. Conversely, do the CMs that do not appear to mediate CBT effects on depression nonetheless mediate the effects of other efficacious treatments? This exploration is limited by a paucity of demonstrably efficacious treatments for youth depression. As the only other recognized EST for youth depression, IPT is an appropriate comparison treatment. However, IPT has a limited overall sample of seven RCTs involving fewer than 400 youths. At least three of those seven RCTs provide data on each of the following CMs tested in Study 1: negative cognition, social engagement, and family functioning. For these three CMs, we examined whether tests of mediation in this smaller sample showed results similar to those found for CBT, suggesting common change mechanisms, or results that differ, suggesting treatment-specific change mechanisms.

To make this comparison, the CBT and IPT data were combined, and treatment type (CBT vs. IPT) was tested as a moderator of the mediational paths. First, we estimated mean effects for the three mediational pathways among IPT trials only. Then, we tested treatment type as a moderator of treatment effects on CMs (Path *a*)

and of the associations between posttreatment CMs and depression outcomes (Path *b*) using univariate meta-analysis for the combined sample. Treatment specificity of mediators would be most clearly supported by moderation of Path *a* but *not* Path *b* because this pattern of results indicates that CBT and IPT differentially activate the CM, which is measured in similar ways across CBT and IPT trials and thus associated with outcome to the same extent. Moderation of Path *b* provides less clear support—this could indicate that the CM measures for CBT trials were different from those for IPT trials (e.g., possibly focusing on different aspects of mediator category that are differentially associated with outcome), or the CM–outcome association is stronger in the context of one type of treatment compared to the other. Next, we tested whether the mediational *ab* pathway differed between IPT and CBT subgroups using MASEM—which more directly addresses our research question about treatment specificity of mediation.

Study 2: Method

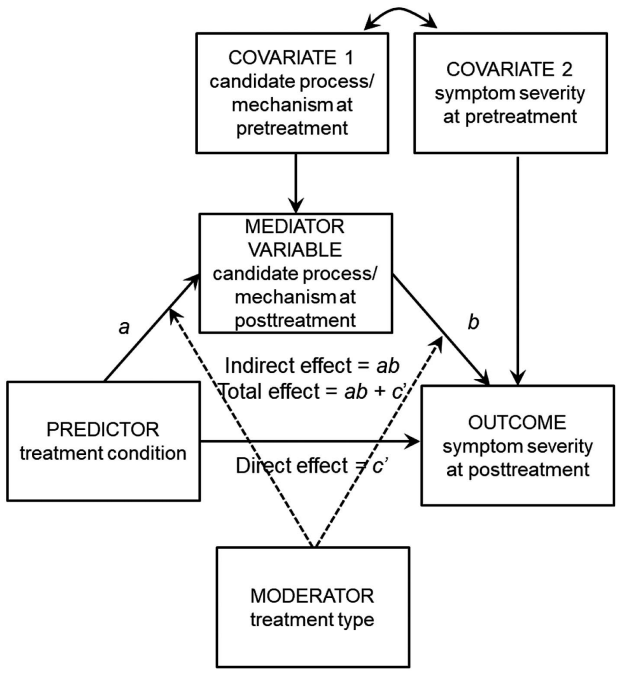
We have described study inclusion criteria, literature search, coding of study characteristics, selection of CMs, and the mediation model in Study 1. Of the CMs that could be tested in Study 1, there was only sufficient data from the IPT trials to test negative cognition, social engagement, and family functioning as potential mediators. Study 2 includes the respective subsets of CBT and IPT trials that measured at least one of these three CMs in common. Figure 1 displays a flowchart documenting the search procedures.

Effect Size Extraction and Data Analysis

We used the same methods as reported in Study 1 to extract and compute effect sizes for the IPT RCTs and to synthesize these effect sizes with univariate meta-analysis and MASEM. However, we omitted the sensitivity analyses. There was only one treatment–control pair per IPT trial, thus sensitivity analyses with independent matrices were not necessary, and we opted not to analyze only complete IPT matrices due to the small number of trials for which all except one had complete data. Analysis of treatment type as a moderator of the mediational effects was conducted on the combined CBT–IPT study sample. We conducted univariate analyses testing treatment type as a moderator of treatment effects on the selected CMs (Path *a*), and of associations between those CMs and posttreatment outcomes (Path *b*), using mixed effects models with the restricted maximum-likelihood estimator of residual heterogeneity. We also tested treatment type as a moderator of the full *ab* pathway within the mediation model (Figure 4), following Jak and Cheung's (2018) MASEM subgroup approach. We first used TSSEM with random-effects models or user-defined random-effects models to fit the mediation model to the CBT and the IPT samples separately, repeating the MASEM analyses in Study 1 with CBT and adding them for IPT. Then, we tested the equivalence of the two paths across the CBT and IPT samples by constraining the path estimates to be equal across samples and comparing the χ^2 statistic for the unconstrained and constrained models. A significant increase in χ^2 after adding the constraints indicates that at least one of the path estimates is not equal across samples. Univariate analyses were conducted using the *metafor* package (Viechtbauer, 2010), and MASEM was conducted using the *metaSEM* package, both implemented in *R* Version 4.0.2.

Figure 4

Two-Wave Model of Treatment Mediation With Treatment Type Tested as a Moderator of the Mediational ab Path



Study 2: Results

The pool of RCTs in Study 2 included the 28 CBT trials in Study 1 and another seven IPT trials. The publication years of all IPT articles and dissertations/theses ranged from 1999 to 2019, with primary outcome articles published between 1999 and 2015. We found no eligible IPT primary outcome articles that met inclusion criteria published after 2015 through September 2020. The sample characteristics of the 28 CBT trials were reported in Study 1. The seven IPT trials had a pooled sample of 370 youths with a mean age of 14.2 years (SD = 1.8) and comprised mostly adolescents—85.7% of samples had mean age of 13.0 years or older. On average, roughly one third of youths were male (M = 30.9%, SD = 12.1) and more than half were Latino (M = 57.7%, SD = 41.2); the remainder were Caucasian (M = 11.3%, SD = 30.0) African American (M = 7.0%, SD = 13.2), Asian American (M = 0.2%, SD = 0.6), other (M = 9.5%, SD = 11.4), or unknown race/ethnicity (M = 14.3%, SD = 37.8). A diagnosis of depressive disorder was required in more than half (57.1%) of IPT trials. A table of IPT trials and their characteristics is presented in Table 3, and the full references of articles reporting study methods and data are listed in Appendix B.

Negative Cognition Moderation Analyses

Analyses of negative cognition synthesized data from 167 participants assigned to three IPT–control comparisons (all independent). IPT had a statistically significant effect on negative cognition (g = 0.62, 95% CI [0.13, 1.12], p = .014, τ^2 = 0.076, k = 3, N = 126). The trim-and-fill method estimated two missing studies on the right side of the plot and adjusted the mean treatment effect

Table 3

Characteristics of Seven Randomized Controlled Trials of Interpersonal Psychotherapy for Youth Depression and Measures of Candidate Mediators Examined in the Present Meta-Analysis

Primary outcome article	*N*	M_{age} (y)	Diagnosis required?	EST	Control	Candidate mediators
Dietz et al. (2015)	42	10.6	Yes, MDD, DYS, or DD–NOS	IPT individual family-based	Nondirective supportive therapy	SOC: SAS-SR (modified)—peer impairment, social impairment FAM: CBQ—parent, self
Mufson et al. (2004)[a]	63	15.1	Yes, MDD, DYS, DD–NOS, or ADJ	IPT individual	Usual care (school counseling)	SOC: SAS-SR—friends, dating FAM: SAS-SR—family; CBQ—father, mother
Mufson et al. (1999)	48	15.8	Yes, MDD	IPT individual	Attention placebo (clinical monitoring)	COG: SPSI-R—negative problem orientation SOC: SAS-SR—friends, dating FAM: SAS-SR—family PRO: SPSI-R—rational problem solving REF: SPSI-R—positive problem-solving orientation AVO: SPSI-R—avoidance coping
Rosselló and Bernal (1999)	46	14.7	Yes, MDD or DYS	IPT individual (also tested CBT individual)	Waitlist	COG: Piers-Harris Children's Self-Concept Scale SOC: Child Behavior Checklist—social abilities adolescent, social abilities parent; Social Adjustment Scale for Children and Adolescents FAM: Family Emotional Involvement and Criticism–Self-Report—intensity of emotional involvement, perceived criticism
Tang et al. (2009)	73	15.3	No	IPT individual	Usual care (school counseling)	COG: Beck Hopelessness Scale
Young et al. (2006a)	41	13.4	No	IPT group	Usual care (school counseling)	FAM: CBQ—father, mother
Young et al. (2010)	57	14.5	No	IPT group	Usual care (school counseling)	SOC: SAS-SR—dating, friends FAM: SAS-SR—family

Note. y = years; ADJ = adjustment disorder with depressed mood; AVO = avoidance; CBQ = Conflict Behavior Questionnaire; CBT = cognitive behavioral therapy; COG = negative cognition; DD–NOS = depressive disorder–not otherwise specified; DYS = dysthymia; EST = empirically supported treatment; FAM = family functioning; IPT = interpersonal therapy; MDD = major depressive disorder; PRO = problem solving; REF = reframing; SAS-SR = Social Adjustment Scale–Self-Report; SOC = social engagement; SPSI-R = Social Problem-Solving Inventory–Revised.

[a] In this trial, both clinicians and youths were randomized for most of the sample, but for a small subset (n = 7) of youths in one school, only clinicians were randomized because each clinician was restricted to treating students on a particular floor.

upwards ($g = 0.98$, 95% CI [0.42, 1.54], $p < .001$, $\tau^2 = 0.269$, $k = 5$). However, these findings should be interpreted with caution, as only three studies were available for this analysis. IPT also had a statistically significant effect on depression symptom outcomes in these studies ($g = 0.79$, 95% CI [0.44, 1.14], $p < .001$, $\tau^2 < 0.001$, $k = 3$, $N = 138$), which appears to be unaffected by publication bias as no missing studies were estimated by the trim-and-fill method. The mean effects of IPT appear to be more than twice as large as that of CBT on negative cognition ($g = 0.25$) and on outcome ($g = 0.30$; see Study 1 and Table 2). The association between negative cognition and outcome in IPT comparisons ($r = 0.19$, 95% CI [−0.85, 1.24], $p = .715$, $\tau^2 = 0.543$, $k = 2$, $N = 57$) appears to be half as strong as that among CBT comparisons ($r = 0.43$; see Study 1).

In the combined sample of 27 CBT and three IPT comparisons ($N = 2{,}804$), treatment type significantly moderated the effect of treatment on negative cognitions, QM(1) = 4.93, $p = .026$, accounting for 78.6% of heterogeneity (depicted in Figure 5). Treatment type did not moderate the association between negative cognitions and outcome at posttreatment, QM(1) = 0.41, $p = .521$, $N = 1{,}691$. MASEM revealed significant mediation for CBT (β = .030, 95% CI [.019, .043], $k = 28$, $N = 3{,}367$) but not for IPT (β = .046, 95% CI [−.051, .181], $k = 3$, $N = 167$) trials. When the direct paths linking treatment to negative cognition, and negative cognition to outcomes were constrained to be equal across CBT and IPT comparisons, model fit worsened, but fell short of statistical significance, $\Delta\chi^2(2) = 5.87$, $p = .053$, $N = 3{,}534$. The null hypothesis of no difference between subgroups thus cannot be rejected, indicating either that the path estimates are comparable in CBT and IPT comparisons or that statistical power was insufficient to detect differences. These findings are consistent with the idea that negative cognition may mediate treatment effects in IPT to a similar extent that they do in CBT.

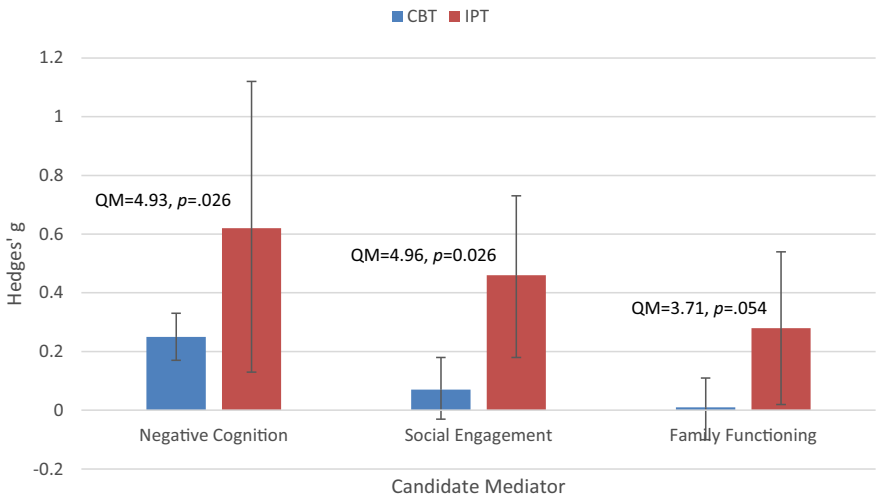
Social Engagement Moderation Analyses

Analyses of social engagement synthesized data from 256 participants assigned to five IPT–control comparisons (all independent). IPT exerted a statistically significant effect on social engagement ($g = 0.46$; 95% CI [0.18, 0.73], $p = .001$, $\tau^2 < 0.001$, $k = 5$, $N = 222$). The trim-and-fill method estimated no missing studies, suggesting minimal impact of publication bias. IPT also had a statistically significant effect on outcome in this subset of studies ($g = 0.61$, 95% CI [0.33, 0.88], $p < .001$, $\tau^2 < 0.001$, $k = 5$, $N = 226$). The trim-and-fill method decreased the mean effect slightly ($g = 0.50$, 95% CI [0.25, 0.74], $p < .001$, $\tau^2 < 0.001$, $k = 7$), with two missing studies estimated on the left side of the plot, suggesting mild potential for publication bias. The mean effects of IPT appear to be four times as large as that of CBT on social engagement ($g = 0.11$ for 11 available, independent comparisons) and twice as large as that of CBT on outcome ($g = 0.29$; see Study 1 and Table 2). The association between social engagement and outcome in IPT comparisons ($r = 0.33$, 95% CI [0.18, 0.48], $p < .001$, $\tau^2 = 0.010$, $k = 5$, $N = 219$) appears to be somewhat stronger than that among CBT comparisons ($r = 0.20$; see Study 1).

In the combined sample of 11 independent CBT and five IPT control comparisons ($N = 1{,}183$), treatment type significantly moderated the effect of treatment on social engagement, QM(1) = 4.96, $p = .026$, accounting for 81.7% of heterogeneity (depicted in Figure 5). Treatment type did not moderate the association between social engagement and outcome at posttreatment, QM(1) = 1.00, $p = .317$, $N = 1{,}084$. MASEM revealed significant mediation for IPT (β = .052, 95% CI [.021, .096], $k = 5$, $N = 256$) but not for CBT (β = .007, 95% CI [−.0002, .018], $k = 14$, $N = 1{,}543$) trials. When the direct paths linking treatment to social engagement, and social engagement to outcomes were constrained to be equal across CBT

Figure 5

Treatment Type as a Moderator of Treatment Effects on Negative Cognition, Social Engagement, and Pleasant Activities at Posttreatment, Synthesized Across Randomized Trials of Cognitive Behavioral Therapy (k = 28) and Interpersonal Therapy (k = 7)



Note. Hedges's g values are adjusted using a small sample correction. Error bars denote the 95% confidence intervals for each mean effect size. QM is the test statistic for moderation. CBT = cognitive behavioral therapy; IPT = individual interpersonal therapy. See the online article for the color version of this figure.

and IPT matrices, model fit worsened significantly, $\Delta\chi^2(2) = 9.53$, $p = .009$, $N = 1{,}799$. The null hypothesis of no difference between subgroups is thus rejected, indicating that the path estimates are not the same in CBT and IPT trials. The larger and significant indirect effect point estimate, along with the significantly larger Path *a* effect sizes among IPT trials, points to social engagement as a mediator of IPT but not of CBT.

Family Functioning Moderation Analyses

Analyses of family functioning synthesized data from 297 participants assigned to six IPT–control comparisons (all independent). IPT exerted a statistically significant effect on family functioning ($g = 0.28$; 95% CI [0.02, 0.54], $p = .033$, $\tau^2 < 0.001$, $k = 6$, $N = 253$). The trim-and-fill method estimated no missing studies, suggesting minimal impact of publication bias. IPT also had a statistically significant effect on outcome in this subset of studies ($g = 0.75$, 95% CI [0.43, 1.06], $p < .001$, $\tau^2 = 0.050$, $k = 6$, $N = 266$), which appears to be unaffected by publication bias as no missing studies were estimated by the trim-and-fill method. The mean effects of IPT appear to be nearly 30 times as large as that of CBT on family functioning ($g = 0.01$) and 3 times as large as that of CBT on outcome ($g = 0.27$; see Study 1 and Table 2). The association between family functioning and outcome in IPT comparisons ($r = 0.36$, 95% CI [0.17, 0.55], $p < .001$, $\tau^2 = 0.040$, $k = 6$, $N = 252$) appears to be somewhat stronger than that among CBT comparisons ($r = 0.24$; see Study 1).

In the combined sample of 13 CBT and six IPT control comparisons ($N = 1{,}629$), treatment type did not moderate the effect of treatment on family functioning, QM(1) = 3.71, $p = .054$, just failing to reach significance (depicted in Figure 5). Treatment type did not moderate the association between family functioning and outcome at posttreatment, QM(1) = 1.74, $p = .188$, $N = 1{,}536$. MASEM revealed significant mediation for IPT ($\beta = .029$, 95% CI [.004, .064], $k = 6$, $N = 297$) but not for CBT ($\beta = .001$, 95% CI [−.006, .009], $k = 17$, $N = 2{,}865$) trials. When the direct paths linking treatment to family functioning, and family functioning to outcomes were constrained to be equal across CBT and IPT matrices, model fit worsened significantly, $\Delta\chi^2(2) = 6.04$, $p = .049$, $N = 3{,}162$. The null hypothesis of no difference between subgroups is thus rejected, indicating that the path estimates are not the same in CBT and IPT trials. The larger and significant indirect effect point estimate, along with the nonsignificantly larger Path *a* effect sizes among IPT trials, are consistent with the idea that family functioning may mediate treatment effects in IPT but not in CBT.

Study 2: Discussion

The moderation analyses produced evidence that some mediational pathways differ between CBT and IPT, in some ways expected, and in other ways unexpected.

Both CBT and IPT had effects on negative cognition, though somewhat surprisingly, the effect size was larger for IPT; and negative cognition played a mediational role in depression outcomes for both CBT and IPT. One interpretation of these findings is that negative cognition may be a mediator and potential change mechanism that is not treatment-specific. Another interpretation is that negative cognition may be conceptually similar and strongly associated with depression symptoms; therefore, negative cognition could be seen as something of a proxy for depression symptoms. This second interpretation is supported by our findings that the association between negative cognition and depression outcome among CBT trials ($r = .43$) appears stronger than the associations between all other CMs and depression outcome among CBT or IPT trials (range: $r = .16$–36). Also consistent with this interpretation is Kolko et al.'s (2000) finding that hopelessness, a CM with a high degree of overlap with depression symptoms, was improved by both CBT and family therapy, whereas cognitive distortion, a CM with less overlap with depression symptoms, was improved only by CBT.

In line with IPT's emphasis on improving social interaction and communication and addressing relationship issues with parents and peers, IPT showed larger treatment effect estimates than CBT on both social engagement and family functioning, although the difference fell just short of statistical significance for family functioning. Treatment type did not moderate the association between either CM and depression symptoms, nor, for that matter, did it moderate the association between negative cognition and depression symptoms. This is reassuring in that it shows that the inherent relationship between each of these CMs and depression symptoms is estimated to be the same irrespective of the treatment applied, as we would expect absent large differences in the nature of the samples or the methods used.

Treatment specificity in the mediators was shown by the mediational role of both social engagement and family functioning in the effects of IPT on depression symptoms, whereas these CMs showed no mediational role in CBT effects. This pattern of findings points to social engagement as a mediator and potential change mechanism specific to IPT with a similar, albeit weaker, pattern of findings suggesting that family functioning may also be an IPT-specific mediator.

General Discussion

To extract maximum information from the available evidence base on how efficacious therapies bring about their effects on youth depression, we conducted a meta-analysis to assess putative change mechanisms as mediators of treatment outcome. Our meta-analysis enabled us to address some weaknesses of prior reviews by greatly expanding the number of contributing studies, fitting the same mediation model to all studies with data on a particular CM, and quantifying mediation effects. Here, we discuss the theoretical and clinical implications of our findings and raise questions for future investigation.

Does Changing Cognitions Matter to CBT Outcomes for Youth Depression?

Despite the identification of negative cognition as a robust mediator of CBT in 24 trials with over 3,000 depressed youths, the overall pattern of findings challenge the assertion that changing maladaptive thoughts, a core part of CBT, is a key active ingredient in ameliorating depression. First, the absence of significance or even a meaningful effect size is especially unexpected for reframing—thought to be the process through which individuals reduce their negative cognitions. Compared to negative cognition, reframing is a more direct measure of the skills that CBT purports to foster and overlaps less conceptually with depression symptoms. Moreover,

reframing is typically measured in the opposite direction from depression symptoms, thus the two are less likely to be confounded (Crits-Christoph et al., 2013; Weersing & Weisz, 2002). Support for the cognitive change part of CBT theory would certainly have been stronger if reframing had also emerged as a mediator, or at least had shown improvement among youths receiving CBT. Second, CBT had smaller effects on negative cognition than IPT, and negative cognitions functioned as treatment mediator in both CBT and IPT trials, even though IPT does not emphasize changing cognitions. Third, the correlation between negative cognition and depression outcomes appeared to be roughly twice as large as the correlations between other CMs and depression outcomes in CBT trials (though in IPT trials, negative cognition appeared to have a smaller correlation with depression outcomes compared with other CMs). Our findings resemble some results from a recent trial comparing CBT and IPT for depressed adults (Bruijniks et al., 2022)—IPT led to larger reductions in dysfunctional thinking than CBT. However, these reductions were associated with subsequent increase in depressive symptoms, in contrast to what we found. Nevertheless, work from our research team and others converges on the possibility that change in negative cognition may be a nonspecific change mechanism or even a correlate of recovery from depression without exerting any influence on symptoms.

Interestingly, the same study that found cognitive change to be nonspecific to treatment of adults also found that CBT increased use of CBT skills relative to IPT, which in turn mediated subsequent decrease in depression symptoms (Bruijniks et al., 2022). In addition, three small CBT trials in the present meta-analysis had, in addition to a control condition, a behavioral treatment condition (e.g., relaxation, problem solving, and activity scheduling) that was not included in our analyses; two of them found that the CBT condition was superior to the behavioral treatment in improving negative cognition (Kahn et al., 1990; Stark et al., 1987), suggesting that cognitive therapy strategies may have a unique impact on cognitive change. Although one did not (Reynolds & Coats, 1986), none found that the behavioral treatment was superior to CBT in changing cognition. Moreover, evidence from youth studies that did not meet our inclusion criteria suggests that reframing may be central to reducing depression symptoms for some patients. Reframing was found to be a mediator of family-based CBT on depression symptom outcomes among child or adolescent offspring of parents with a history of major depression (Compas et al., 2010), and among children with bipolar disorder (MacPherson et al., 2016).

In sum, our findings indicate that cognitive change occurs as a result of efficacious therapy but fail to support the hypothesis that therapy processes designed to change cognitions matter in treating depressed youths. It is possible that cognitive change can be an important step toward reducing youth depression, but the methods used to date to initiate such change have not been very effective with young people. To provide a more conclusive picture, more work is needed to validate measures of reframing and to elucidate which specific reframing skills (e.g., positive problem orientation, rational reappraisal, imagining how the situation would look to someone else, estimating how likely it is that a particular thought is true, thinking of what one would say to a friend who has the same belief, considering whether it would really be so bad if the thought were true) might improve negative cognition among depressed youths.

Does Behavioral Activation Matter to CBT Outcomes for Youth Depression?

We found mixed support for increasing adaptive behaviors as a treatment focus in CBT, given positive (albeit nonsignificant) treatment effects on pleasant activities in five trials, and significant mediation of depression outcomes in a subset of three trials with complete data. Our findings on pleasant activities align somewhat with recent research indicating that depressed youths' understanding and use of behavioral but not cognitive skills, and the quality of therapists' behavioral but not cognitive intervention strategies were associated with improvement in outcomes (Arora et al., 2019; Webb et al., 2019).

Perhaps more robust findings were not obtained because existing CBT protocols may not be emphasizing behavioral change sufficiently or in the most effective ways. Behavioral activation (BA) therapy is a designated EST for adult depression (Society of Clinical Psychology, n.d.) and has demonstrated large effects (g = 0.83, 95% CI [0.58, 1.08], $p < .01$, $I^2 = 63$, $k = 19$, $N = 844$) on adult depressive symptoms against control conditions in a recent meta-analysis (A. T. Stein et al., 2021). More pertinent to our own findings, the same meta-analysis found medium–large effects (g = 0.64, 95% CI [0.39, 0.88], $p < .01$, $I^2 = 18$, $k = 8$, $N = 358$) of BA on measures of activation. BA overlaps substantially with activity scheduling in CBT; however, it differs in its increased emphasis on idiographic functional analysis, addressing avoidance, and in some versions, a discussion of values to guide activity selection (Chu et al., 2016; McCauley et al., 2016; A. T. Stein et al., 2021). Thus, the lack of focus on avoidance by CBT protocols for youth depression is consistent with our findings that only three CBT trials examined avoidance.

For youth depression, BA has not yet achieved EST status (Weersing, Jeffreys, et al., 2016), but there are ongoing efforts to develop and evaluate BA for youth (Chu et al., 2016; McCauley et al., 2016). Thus far, results have been mixed. One research team found a large, near-significant change in a youth-reported (albeit not parent-reported) measure of scheduled activity engagement, goal-directed activation, and avoidance among depressed or anxious youths receiving transdiagnostic BA, compared to waitlist (Chu et al., 2016). However, another team found no significant change in scheduled activity engagement and goal-directed activation in both BA and the comparison condition comprising CBT or IPT, despite depression symptom reduction in both groups (McCauley et al., 2016). Our own findings from a previous study (Ng et al., 2016) of coping strategies used by depressed youth indicated that the majority of strategies they perceived to be effective at boosting their mood involved engaging in pleasant activities. Perhaps pleasant activity engagement needs to be precisely personalized to fit the individual youth's needs and values, and avoidance needs to be concurrently targeted—as emphasized by BA—in order to mediate treatment outcomes.

In sum, our findings provide preliminary support for the hypothesis that increasing pleasant activities matters in treating depressed youths. The mixed findings from our meta-analysis and prior literature highlight the need for more research to clarify whether activity engagement and other behavioral mechanisms mediate reductions in CBT or closely related treatments such as BA.

If Changing Cognitions and Behaviors Do Not Matter in CBT for Youth Depression, Then What Does?

We found no support for a mediational role by any of the other CMs available in the CBT trials included in our analyses. The stunning lack of meaningful treatment effects on CMs critical to CBT theory, and the pattern of evidence that contradicts changing cognitions as a change mechanism in CBT for youth depression has forced us to confront three (nonmutually exclusive) possibilities.

First, the psychometric characteristics of measures used to assess CMs might not have been uniformly strong within and across CM categories, thereby attenuating mean treatment effects on CMs and muddying any true differences that may exist between CMs. We noticed that nearly all trials assessed outcomes using one of several extensively validated depression symptom measures (e.g., Beck Depression Inventory, Children's Depression Inventory, Center for Epidemiological Studies–Depression Scale, Moods and Feelings Questionnaire). On the other hand, assessment of each CM category involved a large variety of measures across trials (see Table 1 note). Over half of the negative cognition measures were used in multiple trials, but all of the reframing measures, and most of the other CM measures appear to be used in just one trial in the present meta-analysis. We surmise that the validation efforts might have been uneven across this large group of measures. Moreover, assessment experts stress that psychometric characteristics of a measure are conditional on the participant sample and the purpose of the assessment (Hunsley & Mash, 2007). It is possible, for example, that some of these measures were well-validated with adults but not with youths, or that reliability and convergent validity were established—thus explaining the significant small–moderate correlations with depressive symptoms—but not sensitivity to treatment response.

Second, some of the CMs—at least the way that they have been measured—might in fact be nonmalleable, trait-like characteristics that correlate with depression symptoms. Kraemer et al. (2002) describe pre- or posttreatment measures that are correlated with outcome as "nonspecific predictors of treatment outcome." In this case, CBT may still activate some process other than the ones we have examined that reduces negative cognitions, in turn, ameliorating depression symptoms. It is unclear what this other process might be, but it is plausible that noncognitive processes may have the effect of reducing negative cognitions. This interpretation is consistent with our findings that IPT had larger effects on negative cognition than CBT.

Third, CBT may act directly on depressive symptoms without going through intermediate change mechanisms or processes. This provocative idea is consistent with a recent alternative perspective of psychopathology—the symptom network approach (Borsboom, 2017). This approach conceptualizes depression as a network of interconnected, individual depression symptoms. Once activated by genetic and environmental risk factors, individual symptoms may then activate other symptoms, leading to an elevated cluster of symptoms that mutually reinforce one another. Research on symptom networks has focused on identifying central symptoms—those that are most closely interconnected and potentially influential in the experience, onset, and maintenance of depression (Boschloo et al., 2016; Fisher et al., 2017; Mullarkey et al., 2019). Researchers are beginning to investigate these central symptoms as mediator variables on which CBT directly intervenes, and through which CBT exerts indirect effects on other symptoms (Boschloo et al., 2019). Symptom networks do not deny the existence of change mechanisms distinct from a disorder and its component symptoms, but they offer a model of how treatment may impact symptoms without going through change mechanisms.

Does IPT Work via Different Mechanisms From CBT for Youth Depression?

Social engagement and family functioning failed to demonstrate a mediational role across multiple CBT trials, but preliminary evidence from a handful of IPT trials suggests that they may serve as mediators of IPT. Not only did IPT have a larger treatment effect on these CMs than CBT—significantly for social engagement and near-significantly for family functioning—the treatment–CM–outcome mediational pathway was significantly different between IPT and CBT trials for both CMs. These findings are consistent with the assumption that depression can be alleviated by improving interpersonal skills and relationships, and that such improvements are treatment-specific. Although some CBT protocols address social skills (e.g., Bearman & Weisz, 2009) or social problem solving (e.g., Rohde, 2017), existing CBT protocols, on average, may not deliver a strong enough dose to effect reliable change on relevant measures. On the other hand, IPT has a dedicated focus on improving peer, dating, and family relationship issues and has demonstrated substantial change in measures of these constructs. In the only trial to compare CBT and IPT against a control condition among depressed youths, Rosselló and Bernal (1999) found that only IPT significantly improved social adjustment and self-esteem, although they did not test either CM as mediator. By comparing multiple CBT trials against multiple IPT trials in the same analysis, our meta-analysis may have provided one of the strongest pieces of evidence supporting the IPT theoretical model—not only do interpersonal putative change mechanisms mediate outcomes of youth depression, they are specific to a treatment with an explicit focus on important relationships in a young person's life. On the other hand, negative cognition's mediational role did not appear to be specific to CBT.

However, we note that IPT studies with adults have produced findings that conflict with the idea of IPT-specific change processes and mechanisms. Coding of session transcripts from the NIMH Treatment of Depression Collaborative Research Program revealed that processes adhering to the CBT prototype appeared to be smuggled into IPT sessions; furthermore, adherence to CBT processes and adherence to IPT processes were each associated with improvement in depression outcomes across treatments (Ablon & Jones, 2002). Similarly, a recent trial found that both IPT and CBT increased use of IPT skills, which led to depression symptom reduction (Bruijniks et al., 2022). Both studies suggest that active ingredients associated with one treatment type may nevertheless be used by therapists or patients during the course of another type of treatment and may yield improvement in symptoms.

Due to the small pool of IPT trials with youths, more IPT trials are needed to clarify whether all the moderator findings would generalize to a larger sample. Because the number of studies and the sample sizes involving IPT were so small, it is possible that the significance of mediation effects may change with additional RCTs added to the sample. A related issue is whether these findings would generalize to a broader sample of IPT studies led by different

research teams. We noted that five out of the seven IPT trials were led by the developers of IPT for adolescents. The IPT protocols and measures of CMs and outcomes used may have been unusually consistent across these trials, and treatment may have been delivered with especially high fidelity, thereby producing a very clear signal with respect to effects on specific CMs and outcomes. On the other hand, CBT has developed into a relatively broad array of treatment protocols developed and tested by different research teams and thus were likely to have greater variability in terms of which CBT elements were emphasized in their protocols, how closely study therapists adhered to their protocols, and the specific measures of CMs and outcomes used. For example, four out of five IPT trials (Dietz et al., 2015; Mufson et al., 1999, 2004; Young et al., 2021) measuring social engagement used the same measures, whereas only two out of 14 CBT trials shared one measure of social engagement, and another two shared a different measure—none of the other CBT trials shared any social engagement measure. This heterogeneity could have made it much less likely that any two treatment teams would have carried out CBT the same way, thereby producing a weaker signal than in IPT with respect to effects on specific CMs and outcomes. To explore this possibility, we have reported heterogeneity statistics derived from our univariate meta-analyses (see supplemental Table S1), with the caveat that these statistics are not well estimated with small numbers of effect sizes, notably for the IPT trials. Thus, the evidence is inconclusive as to whether treatment effects on outcomes or CMs were more heterogeneous for CBT than for IPT.

In addition, our findings add to those of the few trials with depressed youths that tested treatment effects on different CMs for multiple therapy conditions to assess the specificity of mediators to treatment type. Stice, Rohde, Seeley, et al. (2010) compared group CBT, bibliotherapy CBT, and a group supportive-expressive intervention against a no-treatment control. They found that negative cognition and loneliness were improved by group CBT only, whereas pleasant activities and emotional expression were both improved by group CBT and supportive-expressive intervention; bibliotherapy CBT did not lead to improvement in any CM. They also reported that negative cognitions and pleasant activities mediated the effects of group CBT and emotional expression mediated the effects of supportive-expressive intervention—without reporting whether the CMs not hypothesized for a specific treatment mediated that treatment's outcome. Kolko et al. (2000) compared individual CBT, systemic behavioral family therapy, and nondirective supportive therapy. They reported that effects on cognitive distortion were specific to CBT, but effects on hopelessness were common to both CBT and family therapy. Similarly, they found that improvement in some measures of family functioning (i.e., family conflict, parent–child relationship problems) was specific to family therapy, improvement in others (i.e., marital discord, behavioral control aspect of family functioning) was specific to CBT, and improvement in general family functioning was common to both therapies. Findings from these RCTs directly comparing therapies suggest that treatment specificity may depend not only on the treatment but also on the specific measure of the change mechanism. Unfortunately, we are unable to test this hypothesis due to the small number of studies that used any one measure. Identifying treatment-specific mediators would require more trials assessing various measures of each CM.

Limitations

Chief among the present review's limitations is the issue of temporal precedence of the CMs—the Achilles' heel of research on mediators and mechanisms. Indeed, Stice, Rohde, Seeley, et al.'s (2010) mediation findings suggest that for most participants, changes in negative thoughts and pleasant activities did not occur before symptom reduction at posttreatment. Our mediation models were constrained by the data available at each time point; restricting the CM data to the midtreatment time point would have severely restricted our sample size. A less severe sample size restriction would result from including posttreatment CM data and follow-up outcome data in the model, but the results from this model would focus on sustained effects after treatment and have a substantively different interpretation from one where the outcome is measured at posttreatment. In either case, neither of the above models would preclude the outcome from changing concurrently with or before the CM. More intensive measurements at multiple time points during treatment would be required to build the kind of model that can establish temporal precedence of the CM, but this measurement schedule is rarely utilized in youth psychotherapy RCTs (see Marker et al., 2013, for an exception). The temporal precedence of the mediators identified in the present study needs to be determined in order to meet the criteria for change mechanism status, and this will require expansion of measurement models in future studies.

Other limitations are sample-related. The relatively few RCTs in some of the mediation models render some of our conclusion tentative as they may not generalize to the full range of RCTs of ESTs for youth depression, much less to ESTs as they are carried out in usual clinical care settings. With a few exceptions (e.g., Weisz et al., 2009), the majority of included RCTs appeared to be efficacy trials conducted in research settings, with study-employed therapists and recruited youths, rather than the more clinically representative effectiveness trials conducted in practice settings, with practicing clinicians and referred youths—which unfortunately is not a problem unique to youth depression treatment research (see Weisz et al., 2014). We were also surprised by our inability to find eligible RCTs with primary outcome articles published since 2016. Recent trials were excluded for various reasons, such as not having measured one of the selected CMs at posttreatment (e.g., Fristad et al., 2019; Wright et al., 2017) or recruiting a mixed sample of participants who had either depression or anxiety (e.g., Ehrenreich-May et al., 2017; Weersing et al., 2017), or conduct problems (e.g., Weisz et al., 2020). Several had ineligible treatment conditions that integrated techniques from CBT with those from other theoretical models (e.g., interpersonal, family, attachment; see Poole et al., 2018; Tompson et al., 2017). Others lacked eligible control groups because the design involved comparing two treatments (e.g., CBT vs. mindfulness-based intervention; see Shomaker et al., 2017), different versions of the same treatment (e.g., IPT with parent involvement vs. without, Gunlicks-Stoessel & Mufson, 2011), or different ways of combining and sequencing treatments (e.g., Gunlicks-Stoessel et al., 2016). We believe that on the whole, these trials reflect current directions in the youth psychotherapy literature and represent progress in the field beyond straightforward comparisons of CBT or IPT to a control group for a single diagnostic category. Unfortunately, they were not suited to answering research questions about change mechanisms of CBT and IPT for youth depression. In addition, we may have missed RCTs that were not

published in English-language journals that might have met all other inclusion criteria.

Several limitations stem from the recent development of MASEM methods; common extensions of univariate meta-analysis have yet to be tested with MASEM. For example, our transformation of treatment effects on the mediator and outcome variables from standardized mean differences to point biserial correlations is common in univariate meta-analysis and has precedent in MASEM (Collins et al., 2007; Gu et al., 2015; Wilson et al., 2016). However, methodologists are only beginning to examine how such transformations may affect the accuracy of MASEM (see de Jonge et al., 2020). Furthermore, we did not use sophisticated techniques such as multilevel meta-analysis (Van Den Noorgate & Onghena, 2003) and robust variance estimation (Hedges et al., 2010) because these have not been developed to address dependency in MASEM. Our handling of dependence among multiple treatment or control groups from the same RCTs and among multiple measures of the outcome or CM constructs from the same RCT (i.e., taking the mean), although commonly undertaken, was not ideal. We note, however, that our sensitivity analyses for dependency and missing data did not render any significant findings nonsignificant. Thus, we believe that our findings are likely to hold up if later analyzed using improved MASEM methods. Approaches that incorporate multilevel meta-analysis into TSSEM to account for dependent correlations (Wilson et al., 2016) are in development and could be helpful for addressing dependency in future studies using MASEM.

Some of the above limitations reflect the reality of the field, such as the small number of studies, the low frequency at which CMs and outcomes were measured during treatment, and the difficulty of obtaining data for all the bivariate relationships in the model for all studies. Nearly all authors responded when additional data were requested, but several simply did not have the time or resources to retrieve the data needed, despite our attempts to contribute resources to assist in data retrieval. Sometimes the data were irretrievable, having been stored on old equipment predating the ubiquitous use of personal computers. We consider this meta-analytic attempt as a working model for one way to move forward from the scant and mixed findings that individual mediation studies in youth psychotherapy so often produce. We encourage other researchers to improve on our methods and add to the findings, and we will start by suggesting future research directions.

Future Directions

We believe that improvements in how and when we measure candidate change mechanisms are integral to advancing our understanding of how any psychotherapy works. Measurement issues have surfaced multiple times throughout the process of conducting this meta-analysis, including questions about the psychometric properties of measures of CMs for depressed youths and the generally low frequency with which CMs and outcomes were measured. Measure development work focused on the putative change mechanisms of CBT and IPT, addressing multiple psychometric characteristics (not only reliability, but also various forms of validity, responsiveness to treatment-related change, and clinical utility; see Hunsley, 2015), and conducted with depressed youths, would be a welcome contribution to the field. For example, CMs involving compensatory skills (i.e., effort made to reduce negative cognitions or their impact, such as reframing, problem solving; see supplemental material) may be measured in terms of how much a young person agrees with them, how well they have been learned, how frequently they are utilized, and how effectively they are applied (at the right time and at a high enough level of competence). Research that distinguishes between these aspects of skill measurement, validates youth or parent report against behavioral observations, and examines their sensitivity to clinical change would fill a critical gap in the literature (Lindhiem et al., 2014). Well-validated performance-based and biological measures of change mechanisms would also be informative, in line with recommendations from NIMH (2016; see also Insel et al., 2010). Nearly all measures in the present meta-analysis were based on subjective report by youths, parents, school staff, or researchers. Given that subjective report measures of negative cognition, pleasant activities, social engagement, and family functioning have already shown evidence of mediating outcome across trials, these may be the most promising to investigate using multiple methods, including some that do not rely on subjective reports. Furthermore, given the importance of frequent measurement for capturing key moments in nonlinear change, and for clarifying temporal relations between CMs and outcomes, brief, low-burden measures—perhaps modified from, or validated against longer, more comprehensive measures—would be especially valuable. The near-universality of smartphone access among adolescents across racial, ethnic, and socioeconomic groups (Anderson & Jiang, 2018) makes it feasible now to collect not only frequent brief self-reports of CMs as young people go about their daily lives, but also behavioral data from mobile sensors without effort from the user, that may serve as proxies for some CMs (e.g., using global positioning system to estimate time spent away from home and school as a proxy for pleasant activity engagement; see Lind et al., 2018). Mobile and wearable technologies present exciting opportunities for measure and quantitative methods development to examine change mechanisms, but these also present unique risks and challenges when used with youths (Bagot et al., 2018). Progress in understanding youth psychotherapy change mechanisms will require expanding the current evidence base beyond retrospective report measures. Thus, we strongly encourage researchers to utilize measures designed to be administered multiple times during treatment, and those that more directly assess behavioral or biological processes.

Another future direction centers on investigating individual differences and other sources of variability in therapeutic change. There was significant heterogeneity between studies in some of the paths that make up the mediation models for most of the CMs. With more complete data, it would be possible to examine moderators that could provide information about whether treatment and mediation effects differ by control type, specific CM measures, specific subsets of depression symptoms, and participant characteristics such as age or symptom severity. Research with depressed adults indicates that CBT, IPT, and antidepressant medication produce equal benefit when averaged across individuals, but that some particular individuals may do much better with one of these treatments than with the others (DeRubeis et al., 2014; Huibers et al., 2015). In addition, the diagnostic category of depression comprises a heterogeneous cluster of symptoms—there is considerable individual variation in the specific constellation of symptoms that may signal different underlying psychopathology, leading to differential

treatment response across individuals (Forgeard et al., 2011). The change mechanisms may differ not only by treatment but also by participant characteristics and specific symptoms assessed as the outcome. Indeed, a recent RCT (Young et al., 2021) demonstrated that youths with high cognitive and low interpersonal risks responded better to a cognitive behavioral depression prevention program, and those with high interpersonal and low cognitive risks responded better to interpersonal program. This striking finding serves as evidence for intervention moderation effects and also hints at the specificity of mediation effects. Presumably, the subgroups that benefited the most received the program that more optimally addressed their specific risk profile (e.g., interpersonal program addressing those with high interpersonal risks). Currently, moderation analysis in MASEM is limited to testing categorical moderators one at a time; advances in MASEM methods that include multiple and continuous moderators could accelerate research on change mechanisms and the circumstances under which they differ.

Next, more complex statistical models may be needed to shed light on change mechanisms. In our meta-analysis, the total effect was glaringly larger than the indirect effect in all tested mediator models. The relatively small indirect effect, even when statistically significant, indicated that the influence of any one mediator was quite modest. Examining whether multiple mediators have a larger joint indirect effect relative to the total treatment effect would help elucidate the more complex change mechanisms that likely operate in any successful treatment. However, the feasibility of testing a multiple mediator model would depend on the number of RCTs with data available for multiple mediator constructs, including correlations across the respective measures, and the capability of the MASEM method for synthesizing correlation matrices that have substantial missing data, as RCTs would differ in the categories for which data are available.

Finally, a framework for classifying putative change mechanisms by the strength of supporting and conflicting evidence, as well as the rigor of methods used to generate that evidence, may propel the field's collective understanding of how therapies work. As a starting point, we could use the criteria for ESTs (Chambless & Hollon, 1998), several of which are readily applicable to change mechanisms, such as supporting evidence yielded by RCTs that used an active control (e.g., usual care, another treatment), and that were led by independent teams of researchers. We had previously suggested classifications as (a) "probable mediators" if the aforementioned methods criteria were met and over half the tests conducted by trial investigators found significant mediation for a CM; or (b) "possible mediators" if the evidence came from RCTs led by the nonindependent teams or that used passive controls (e.g., waitlist, placebo); (c) "experimental" if mediators were not tested in any RCT; and (d) "questionable" if a CM were tested and did not significantly mediate outcome in one or more RCTs (Ng et al., 2020). Other researchers have suggested additional methods and criteria such as assessing multiple CMs and measuring CMs and outcomes over three or more time points to examine temporal precedence of CMs (Lemmens et al., 2016). When mean mediation effects estimated by MASEM are available, the evidence criteria could incorporate the number of RCTs included in analyses and the direction, magnitude, and statistical significance or precision of the mediation effect. For example, based on the optimal sample size suggested by the TSSEM developers (Cheung & Chan, 2005), we could specify that a "probable mediator" designation would require a statistically significant mediation effect in the expected direction synthesized from a minimum of 10 RCTs with a total of 1,000 participants and a "questionable" designation would require a nonsignificant indirect effect synthesized from a sample of similar size. A "possible mediator" designation might suggest mediation in the expected direction but not meet sample size requirements or statistical significance cutoffs, but be of clinically meaningful magnitude (which will need to be determined). Given our findings indicating that some CMs are treatment specific, and the extant literature suggesting individual differences in optimal treatments for depression (e.g., Cohen & DeRubeis, 2018), this classification framework will need to accommodate differences in evidentiary strength of CMs by treatment type, and ideally, by key potential moderators as well.

Our suggestions for further primary research and secondary meta-analytic research on change mechanisms demand considerable time, resources, and expertise, on the part of the investigators conducting the research, and those asked to share their data for meta-analyses. Hence, making progress in understanding change mechanisms of existing therapies may require significant adjustments in journal and funder policies, such as allocating funding to support the training and work of meta-analysts and to compensate trial investigators for retrieving data from existing data sets, and requiring or incentivizing investigators to share raw data or summary statistics from their treatment trials (Ng et al., 2020). Our team and our colleagues, anecdotally, have had mixed experiences with identifying and obtaining funding for meta-analysis. Some have enjoyed success obtaining funding for meta-analytic work from multiple federal agencies and private foundations, others have acquired limited funding adequate to complete only a portion of the proposed work, and yet others have conducted meta-analysis with no funding at all other than institutional support provided for teaching or clinical positions or traineeships, sometimes over many years. Our impressions are that the last group forms the majority of meta-analytic researchers, and that there appears to be a real or perceived lack of funding for meta-analyses of studies within our areas of interest. Of note, the current focus of the NIMH (2022; see strategic plan objective 3.1) on identifying change mechanisms prioritizes those in new interventions rather than those in existing ones—even though those mechanisms are still not well understood in ESTs such as CBT for adolescent depression. The result is that meta-analytic work, especially more complex endeavors such as MASEM that have the potential to shed light on the change mechanisms of existing interventions, may be limited to the relatively small pool of researchers who have time and other resources for their own support, and who are thus able to set aside the time required for such labor-intensive work. Increased funding and higher visibility of funding for the work of meta-analysis could change all that.

Conclusion

To maximize the information gained from 3 decades of randomized trial research on CBT and IPT for youth depression, we conducted a meta-analysis to assess putative change mechanisms as mediators of treatment outcome. Improvement in negative cognition reliably mediated the effects of CBT for youth depression across studies, and there is preliminary evidence for pleasant activities as a mediator of CBT, and social engagement and family functioning as treatment-specific mediators of IPT. Surprisingly,

problem solving and reframing were not improved by CBT. Importantly, this meta-analysis highlighted gaps in the literature, especially the small numbers of IPT trials, and of CBT trials measuring key putative change mechanisms, as well as the low frequency and subjective method of measurement. Nevertheless, our findings have generated provisional conclusions about how two ESTs ameliorate youth depression—which await confirmation with an expanded evidence base—and have suggested a research agenda for the years ahead.

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Appendix A

Articles, Dissertations, and Theses for the CBT Trials in Studies 1 and 2

Articles, dissertations, and theses reporting methods or results from the CBT trials included in Studies 1 and 2, except for the primary outcome articles, which are listed in the References section of the article and indicated by an asterisk there.

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Appendix B

Articles, Dissertations, and Theses for the IPT Trials in Study 2

Articles, dissertations, and theses reporting methods or results from the IPT trials included in Study 2 except for the primary outcome articles, which are listed in the References section of the article and indicated by an asterisk there.

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