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A Meta-Analysis of Cognitive-Behavioral Therapy for Alcohol or Other Drug Use Disorders: Treatment Efficacy by Contrast Condition

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Abstract

Objective.—This meta-analysis examined 30 randomized controlled trials (32 study sites; 35 study arms) that tested the efficacy of cognitive-behavioral therapy (CBT) for alcohol or other drug use disorders (AUD/SUD). The study aim was to provide estimates of efficacy against three levels of experimental contrast (i.e., minimal [$k = 5$]; non-specific therapy [$k = 11$]; specific therapy [$k = 19$]) for consumption frequency and quantity outcomes at early (1 – 6 months [$k_{es} = 41$]) and late (8+ months [$k_{es} = 26$]) follow-up time points. When pooled effect sizes were statistically heterogeneous, study-level moderators were examined.

Method.—The inverse-variance weighted effect size was calculated for each study and pooled under random effects assumptions. Sensitivity analyses included tests of heterogeneity, study influence, and publication bias.

Results.—CBT in contrast to minimal treatment showed a moderate and significant effect size that was consistent across outcome type and follow-up. When CBT was contrasted with a non-specific therapy or treatment as usual, treatment effect was statistically significant for consumption frequency and quantity at early, but not late, follow-up. CBT effects in contrast to a specific therapy were consistently non-significant across outcomes and follow-up time points. Of ten pooled effect sizes examined, two showed moderate heterogeneity, but multivariate analyses revealed few systematic predictors of between-study variance.

Conclusions.—The current meta-analysis shows that CBT is more effective than a no treatment, minimal treatment, or non-specific control. Consistent with findings on other evidence-based therapies, CBT did not show superior efficacy in contrast to another specific modality.

Keywords

Alcohol Treatment; Cognitive Behavioral; Drug Treatment; Meta-Analysis; Relapse Prevention

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Introduction

Cognitive Behavioral Therapy (CBT) is a leading behavioral approach for intervention with alcohol or other drug use disorders (Substance Abuse and Mental Health Services Administration, 2014). Despite its widespread application, the last meta-analysis of CBT efficacy for substance use was conducted 10 years ago (i.e., Magill & Ray, 2009). This is a significant gap, given the role meta-analysis plays in guiding clinical practice decisions at both micro (e.g., individual providers) and macro (e.g., community agency administrators, public service funders) levels.

We define CBT as a time-limited, multi-session intervention that targets cognitive, affective, and environmental risks for substance use and provides training in coping skills to help an individual achieve and maintain abstinence or harm reduction. Applications in the field are often based on Marlatt and Gordon's (1985) model of relapse prevention, and there are several manuals available for use with alcohol (e.g., Epstein & McCrady, 2009; Kadden et al., 1992; Monti, Abrams, Kadden & Cooney, 1989) or other drug use disorders (e.g., Carroll, 1998). CBT for addictions has a well-established evidence base, but this literature continues to evolve (Carroll & Kiluk, 2017). Further, qualitative reviews have concluded that CBT is more effective than no treatment, but have reported mixed results on key questions such as efficacy over another evidence-based therapy (Longabaugh & Morgenstern, 1999), effect variability by primary drug type (Mastroleo & Monti, 2013; McHugh, Hearon, & Otto, 2010), and the optimal timing of intervention effects (Carroll & Onken, 2005).

In quantitative reviews, Irvin and colleagues (1999) examined 26 experimental and quasi-experimental studies of relapse prevention. They reported a small overall effect size ($r = .14$, $p < .05$), and suggested relapse prevention was more effective for alcohol use disorder than for other substances and when delivered in combination with a pharmacological intervention. In 2009, Magill and Ray followed up this work with a meta-analysis of 53 experimental CBT studies, reporting a similar overall effect size ($g = .15$, $p < .005$), although findings related to superior effects with alcohol use disorder were not replicated. The latter meta-analysis additionally noted the conservative nature of the overall effect size given the types of contrast conditions in the clinical trials reviewed. Specifically, the effect of CBT over no treatment was large ($g = .79$, $p < .005$), but this type of contrast was rare ($k = 6/53$). In comparison, effects for non-specific contrasts (i.e., a passive, but time-matched or usual care control condition; e.g., treatment as usual, supportive therapy, drug counseling) and specific contrasts (i.e., another manualized therapy condition; e.g., Motivational Interviewing, Contingency Management) were small and more common in the literature ($g = .15$, $p < .005$, $k = 32/53$; $g = .11$, $p < .05$, $k = 17/53$, respectively). To our knowledge, this was the last CBT meta-analysis across substances, or for individual substances, conducted to date.

Given the long-standing use of CBT in addictions care as well as its continued evolution, an up-to-date meta-analysis is needed. Additionally, reviews of cognitive therapy more broadly highlight a need for meta-analytic knowledge on effectiveness with substance-using populations (Butler, Chapman, Forman, & Beck, 2006; Hofmann, Asnaani, Vonk, Sawyer, &

Fang, 2012). The present meta-analysis provides an updated assessment of CBT efficacy with alcohol or other drug use disorders, and may also offer greater conceptual clarity than past quantitative reviews. Specifically, CBT efficacy when delivered in a stand-alone format, and not combined with another psychosocial (Dutra et al., 2008; Magill & Ray, 2009; McHugh et al., 2010) or pharmacological (Irvin et al., 1999; Magill & Ray, 2009) intervention, was the focus of the present report. These latter topics are worthy of meta-analysis in their own right, and including such studies could obscure effect size estimation for the effect modifying factors of primary interest to this study: 1) CBT efficacy by contrast type (i.e., minimal; non-specific therapy; specific therapy), 2) CBT efficacy by consumption outcome type (i.e., frequency; quantity) and 3) CBT efficacy at early (i.e., 1–6 months post-treatment) and late (i.e., 8+ months post-treatment) follow-ups. Effect estimates were additionally examined for validity and stability in sensitivity analyses (i.e., tests of heterogeneity, study influence, and publication bias).

Method

Primary Study Inclusion

Studies meeting inclusion criteria were English language, peer-reviewed articles published between 1980 and 2018. These were primary outcome reports of randomized controlled trials. All types of experimental control were of interest given the importance of this factor in predicting effect size magnitude in the addictions, mental health, and in psychotherapy more broadly (Imel, Wampold, Miller, & Fleming, 2008; Wampold & Imel, 2015; Wampold, Mondin, Moody, Stich, Benson, & Ahn, 1997; Wampold, 2001). Studies were included if they targeted adult populations (age ≥ 18) meeting criteria for an alcohol or other drug use disorder (DSM III-R through V; American Psychiatric Association, 1987; 1994; 2000; 2013) or problematic use (e.g., Saunders et al., 1993). The treatment must have been identified as either Cognitive Behavioral or Relapse Prevention, although some studies were included based on a description of key CBT elements such as functional analysis, avoidance of high risk situations, and/or coping skills training (see Supplemental Table 1 for details). Studies of CBT delivered in either individual or group format were included, but we excluded studies of CBT delivered as an integrative therapy combined with another psychosocial (e.g., COMBINE Cognitive Behavioral Intervention, Mindfulness-Based Relapse Prevention) or pharmacological intervention.

Literature Search

A literature search was conducted through June of 2018 to identify eligible studies for a large-scale, meta-analytic project on CBT in addictions care (R21AA026006). The first step involved a title, abstract, and keyword search by treatment (‘cognitive behavioral therapy’ OR ‘relapse prevention’ OR ‘coping skills training’), AND outcome (‘alcohol’ OR ‘cocaine’ OR ‘methamphetamine’ OR ‘stimulant’ OR ‘opiate’ OR ‘heroin’ OR ‘marijuana’ OR ‘cannabis’ OR ‘illicit drug’ OR ‘substances’ OR ‘dual disorder’ OR ‘polysubstance’ OR ‘dual diagnosis’), AND study terms (‘efficacy’ OR ‘randomized controlled trial’ OR ‘randomized clinical trial’) in the PubMed database. Then, a search of the Cochrane Register and EBSCO database (i.e., Medline, PsycARTICLES) was performed, removing duplicates from the results of the PubMed search. Abstract screening occurred by two raters in

ABSTRKR (Wallace, Small, Brodley, Lau, & Trikalinos, 2012). A bibliographic search of systematic reviews and meta-analyses of CBT was also performed to identify any candidate studies not identified by the original search methods (Carroll, 1996; Carroll & Kiluk, 2017; Irvin et al., 1999; Longabaugh & Morgenstern, 1999; Magill & Ray, 2009; Mastroleo & Monti, 2013; Miller, Wilbourne, & Hettema, 2003; Prendergast, Podus, Chang, & Urada, 2002). Finally, three studies published between 1980 and 1989 were added during peer review to achieve an overlapping date range with Magill & Ray (2009; i.e., Donovan & Ito, 1988; Jones, Kanfer, & Lanyon, 1982; Kadden, Cooney, Getter, & Litt, 1989). Figure 1 provides a visual representation of study inclusion for the present report on stand-alone CBT efficacy with adult AUD/SUD; the figure follows QUORUM guidelines (Moher et al., 1999). The final meta-analytic sample was comprised of $K = 30$ studies, with 32 study sites (i.e., two studies provided two sites each, Project MATCH Research Group, 1997; McAuliffe, 1990), and a total of 5,398 participants.

Primary Study Characteristic Variables

There were several study characteristic variables of interest to this meta-analysis, as a priori effect size modifiers (i.e., subgroup variables) as well as potential pooled effect variance predictors (i.e., meta-regression covariates) in instances of systematic (i.e., versus random) between-study heterogeneity. Effect size modifiers were: 1) contrast condition type (i.e., minimal [e.g., waitlist, brief psychoeducation]; non-specific therapy [e.g., treatment as usual, supportive therapy, drug counseling]; specific therapy [e.g., Motivational Interviewing, Contingency Management]), 2) substance use outcome type (i.e., frequency; quantity), and 3) follow-up time point (early = 1–6 months post-treatment; late = 8+ months post-treatment). Study-level descriptors and potential meta-regression covariates were mean age, percent female participants, percent white participants, percent black participants, percent Latino/a participants, primary drug outcome (i.e., alcohol, other drug), substance use severity (i.e., dependence, abuse or heavy use), treatment length (i.e., number of planned sessions), treatment delivery (i.e., individual format, group format), study context (i.e., community sample, specialty substance use or mental health clinic, medical setting, college setting, criminal justice setting, other setting), publication country (i.e., United States, other country), use of biological assay outcome measure (i.e., yes, no), and study-level risk-of-bias (Higgins et al., 2011). Data extraction guidelines were detailed in a study codebook available, upon request, from the first author. Data were extracted in two independent passes conducted by trained raters (i.e., fourth and fifth authors), and showed a between-rater agreement rate of 95.3%. Final data entry, where disagreement was observed, required a consensus review with the first author.

Primary Study Outcome Variables

The standardized mean difference was used to measure efficacy outcomes in this meta-analysis.[1] Hedges' *g* includes a correction, *f*, for a slight upward bias in the estimated population effect (Hedges, 1994).

[1]Effect size magnitude was interpreted using the following benchmarks: 0.2 "small", 0.5 "medium", and 0.8 "large" (Cohen, 1988). However, these are generic guidelines and should be considered conservative in the absence of empirically-based effect size distributions for adult SUD/AUD samples. For example, Tanner-Smith, Durlak, & Marx (2018) suggest that effect size magnitudes of 0.05, 0.10, and 0.15 approximate the 25th, 50th, and 75th percentiles (respectively) for behavioral outcomes in youth drug prevention.

$$g_i = \frac{M_{ti} - M_{ci}}{s_{pi}} * [f] \text{ where } f = 1 - (3/(4 * df - 1), \quad \text{and } s_{pi} = \frac{\sqrt{(n_{ti} - 1)s_{ti}^2 + (n_{ci} - 1)s_{ci}^2}}{n_{ti} + n_{ci} - 2}$$

Prior to pooling, effect sizes were weighted by the inverse of the estimate variance to allow larger studies more influence on the overall effect size (Hedges & Olkin, 1985). Primary studies typically provided data on more than one outcome; therefore, data for effect size estimation were selected based on a decisional hierarchy in the following order: 1) biological assay measures, 2) measures of frequency or quantity in the form of means and standard deviations, 3) sample proportions, and 4) other outcomes (e.g., diagnostic measures [Addiction Severity Index; McLellan, Luborsky, Woody, & O'Brien, 1980]). When multiple months of follow-up date were provided, the latest time point in two time intervals was selected (i.e., 1–6 months, 8 + months). Effect sizes were reverse scored as needed (e.g., number of days drank) such that a positive effect size indicated a positive treatment outcome. Finally, when univariate outcome data were not reported, test statistics were transformed using available formulae (e.g., Lipsey & Wilson, 2001). When data from publications were insufficient for effect size calculation, raw data were obtained from authors where possible (i.e., Kadden, Litt, Cooney, Kabela, & Getter, 2001; Project MATCH, 1997). One eligible study was removed due to author non-response to data request (Källmén, Sjöberg, & Wennberg, 2003).

Data Analysis

Alcohol and other drug use effect sizes were pooled using a random effects model. In this approach, there is an assumed distribution for the population effect size with both systematic and random sources of variability (Hedges & Vevea, 1998). The significance of the *Q*-test determined whether statistically significant between-study heterogeneity existed within a given pooled estimate and the I^2 provided a percent heterogeneity estimate, regardless of statistical significance.[2] When I^2 estimates exceeded 40%, indicating that 40% of the variance in effect sizes was due to systematic variance (Borenstein, Hedges, Higgins, & Rothstein, 2015), a multivariate regression model was used to examine potential effect size moderators. Candidate variables were entered in participant (i.e., age, sex, race, primary drug, substance use severity), implementation, (i.e., treatment length, treatment delivery), and methodological (i.e., study risk-of-bias) blocks. Analyses were conducted with Wilson's (2005) METAREG for Maximum Likelihood regression (ML; SPSS Version 24), and variables with significant regression coefficients were placed into a final predictive model along with residual variance estimates. Missing variable codes for regression covariates were mean imputed, and a predictor was removed from the analysis if imputed values reached 20% of total cases (Pigott, 1994). We conducted sensitivity analyses throughout data analysis and considered heterogeneity and moderator analyses as two primary methods for examining effect size validity. Trimmed estimates with influential studies removed (Baujat, Mahé, Pignon, & Hill, 2002) were also provided.[3] Finally, to test for potential publication

[2] I^2 magnitude can be interpreted using the following benchmarks: 0 – 40% "might not be important", 30 – 60% "may represent moderate heterogeneity", 50 – 90% "may represent substantial heterogeneity", 75 – 100% "considerable heterogeneity" (Higgins & Green, 2011).

bias, the relationship between error and effect size was assessed using rank correlation (Begg & Mazumdar, 1994) and graphical methods (Egger, Smith, Schneider, & Minder, 1997). Here, small sample/small effect studies are assumed to characterize unpublished research, resulting in a significant and negative relationship, thus an asymmetrical funnel plot, when publication bias is present.

Results

Primary Study Descriptive Characteristics

The sample included 30 randomized trials, with 32 study sites, targeting CBT for adult substance use disorders published between 1982 and 2018. The median sample size was 102 participants with a minimum of 39 (Donovan & Ito, 1988) and a maximum of 952 (Project MATCH, 1997). The primary substance targets within these clinical trials were alcohol (k = 15), marijuana (k = 3), opiates (k = 2), stimulants (k = 6), and polydrug (k = 6) use. The samples' mean age was 37 (SD = 6), samples were 30% female (SD = 20%) on average, and although report of race and ethnicity were inconsistent, the percentiles were as follows: 68% white (SD = 38%; k = 31/32), 36% black (SD = 37%; k = 18/32), and 12% Latino/a (SD = 27%; k = 13/32). Diagnostically, study inclusion primarily targeted individuals with alcohol or drug dependence (78%). The CBT interventions were 53% individual and 44% group delivered, and one study utilized a mixture of individual and group sessions (McKay et al., 2004). The median number of planned sessions was 12 (*range* = 6 to 40), and recruitment contexts included specialty substance use or mental health clinics (k = 18), community advertising (k = 11), and other specialty settings (e.g., college campus, medical setting, criminal justice system; k = 3). Study-level risk-of-bias assessment showed 60% of studies were low risk (Higgins et al., 2011). When studies were designated as unclear or high risk, this was typically due to the presence or no report of 1) baseline differences between conditions, 2) differential attrition between conditions, and 3) blinding of outcome assessment. Finally, the majority (72%) of studies were published in the United States. Tables 1 and 2 describe each study with respect to design characteristics, effect sizes, and are separated by early and late follow-up time points, respectively.

CBT Effect by Contrast Type, Outcome Type, and Follow-up Time Point

CBT in contrast to minimal treatment.—Primary study effect sizes were pooled by contrast type and within each subgroup, pooled effect sizes by frequency and quantity outcomes at early (i.e., 1 to 6 months) and late (i.e., 8 + months) follow-ups are provided. Studies with minimal, waitlist, or assessment only contrast conditions comprised a minority of the studies reviewed, and the pooled effect size for frequency outcomes was g = .58 (95% CI = .15, 1.01, p = .009; tau^2 = .11, Q > .05, I^2 = 59%; k = 4) at early follow-up and g = .44 (95% CI = .02, .86, p = .039; tau^2 = .00, Q > .05, I^2 = 0%; k = 2) at late follow-up. For quantity outcomes at early follow-up, the pooled effect size for two studies was moderate and significant (g = .67: 95% CI= .41, .98, p < .001; tau^2 = .00, Q > .05, I^2 = 0%; k = 2), but only one study provided late follow-up quantity data (Kivlahan, Marlatt, Fromme, Coppel, & Williams, 1990). Converting effect estimates to a percentile success rate (U^3; Cohen,

[3]An influential study was defined as any study that, if removed, would change the statistical significance of the pooled effect estimate.

1988), the data show 15 to 26% of CBT participants had better outcomes than the median of those in minimal treatment conditions. Analyses by minimal contrast showed no influential studies. Figure 2 shows some asymmetry in the plot of primary studies, by minimal contrast, but rank correlation analyses do not suggest bias due to publication status ($\tau = -.33$, $p = .497$).

CBT in contrast to non-specific therapy.—The second level of contrast included studies that compared CBT to some form of non-specific therapy such as treatment at usual, supportive therapy, or group drug counseling. The pooled effect size for frequency outcomes at early follow-up was small and statistically significant ($g = .18$: 95% CI = .02, .35, $p = .04$; $tau^2 = .03$, $Q > .05$, $I^2 = 45\%$; $k = 9$)6 with a success rate roughly 8% higher than the median within the contrast condition. However, the effect was non-significant at late follow-up ($g = .05$: 95% CI = −.09, .19, $p = .492$; $tau^2 = .00$, $Q > .05$, $I^2 = 0\%$; $k = 7$). For quantity outcomes at early follow-up, the pooled effect was moderate and significant for two studies ($g = .42$: 95% CI = .03, .81, $p = .034$; $tau^2 = .00$, $Q > .05$, $I^2 = 0\%$), and only one study provided late follow-up quantity data (Kivlahan et al., 1990). Analyses of CBT in contrast to a non-specific therapy showed three influential studies that, when removed, the overall effect size became non-significant ($g_{trimmed} \sim .07$ to .26). Figure 3 shows the plot of primary studies, by non-specific contrast, and does not suggest bias due to publication status ($\tau = .36$, $p = .059$).

CBT in contrast to another specific therapy.—The third level of contrast included studies that compared CBT to another specific therapy (e.g., Motivational Interviewing, Contingency Management). The pooled effect size for frequency outcomes was non-significant at early ($g = -.02$: 95% CI = −.12, .08, $p = .740$; $tau^2 = .01$, $Q > .05$, $I^2 = 14\%$; $k = 16$) and late ($g = -.04$: 95% CI = −.15, .08, $p = .507$; $tau^2 = .01$, $Q > .05$, $I^2 = 15\%$; $k = 8$) follow-ups. For quantity outcomes, pooled effects were also non-significant at early ($g = .01$: 95% CI = −.11, .12, $p = .956$; $tau^2 = .01$, $Q > .05$, $I^2 = 36\%$; $k = 8$) and late ($g = .01$: 95% CI = −.09, .11, $p = 887$; $tau^2 = .00$, $Q > .05$, $I^2 = 0\%$; $k = 5$) follow-ups. Analyses of CBT in contrast to another specific therapy showed no influential studies and no evidence of publication bias (Figure 4; $\tau = .00$, $p = .500$).

Analysis of Heterogeneous Pooled Effect Sizes

Given variability in the types of contrast conditions within this sample of CBT efficacy trials, this meta-analysis did not provide a single pooled effect size to characterize the effect of CBT for adult AUD/SUD. Using a subgroup approach, two out of 10 pooled effect sizes were determined to have sufficient systematic heterogeneity to warrant moderator analyses. Of these two estimates (I^2 range = 45 – 59%), only one subgroup also contained a sufficient k number of studies to provide variability in the covariates examined.[4] For heterogeneous effects of CBT on early follow-up use frequency in contrast to a non-specific therapy ($g = .18$; $Q > .05$, $I^2 = 45\%$; $k = 9$), we used a meta-regression approach to conduct random effects moderator analysis. Within this approach, a priori covariates are considered, but residual heterogeneity is also expected and acceptable. Table 3 summarizes findings for the

[4]For heterogeneous effects of CBT on early follow-up use frequency in contrast to minimal treatment, the sample of primary studies was $k = 4$.

participant, implementation, and methodological models. These analyses yielded largely non-significant meta-regression estimates, with the exception of client age. For CBT effects on early follow-up use frequency in contrast to a non-specific therapy, older age was associated with smaller effect sizes ($b = -.072$, $p = .044$).

Discussion

To our knowledge, this is the first meta-analysis of CBT efficacy for adults with alcohol and other drug use disorders conducted in 10 years, despite ongoing research and utilization of the CBT approach. We pooled primary study effect sizes by contrast condition type (i.e., minimal, non-specific therapy, specific therapy), and then by consumption outcome type (i.e., frequency, quantity) and follow-up time point (i.e., early, late). For the most part (k = 8/10), these subgroup estimates showed acceptable homogeneity. This suggests that the selected variables were informative effect size modifiers for the sample of clinical trials reviewed. Meta-analyses of alcohol or other drug treatments generally show effect sizes in the small to moderate range (e.g., Bertholet, Daeppen, Wietlisbach, Fleming, & Burnand, 2005; Lundahl, Kunz, Brownell, Tollefson, & Burke, 2010; Prendergast et al., 2002) and this includes pharmacological treatments (e.g., Fullerton et al., 2014; Maisel, Blodgett, Wilbourne, Humphreys, & Finney, 2013; Streeton & Whelan, 2001). In the present study, a moderate-to-large and stable (i.e., across outcome type and follow-up time point) effect size was observed for CBT in contrast to no treatment or a minimal treatment comparison (15–26% success rate). However, the majority of trials in this review considered CBT in contrast to an active comparison, either a non-specific or a specific therapy. In other words, the measure of efficacy in the CBT literature has most often been how well CBT performs in reference to another form of therapy.

In this meta-analysis, we selected two types of treatment outcomes a priori. These were alcohol or other drug use frequency and quantity. The goal was to consider both abstinence and harm reduction, although quantity outcomes (k_{es} = 19) were reported less consistently than frequency outcomes (k_{es} = 47), and only one study explicitly targeted substance use moderation (Heather et al., 2000). The current study can thus provide only preliminary data on the broader question of whether CBT is particularly effective for certain types of clinical outcomes. For example, Irvin and colleagues (1999) reported effect sizes for secondary outcomes (e.g., self-efficacy or coping skills) that were more than twice the magnitude of those for substance use. In the present study, CBT effect sizes for quantity outcomes were larger than frequency outcomes, such as number of abstinent days. This pattern of findings held for minimal and non-specific therapy contrasts, but caution is warranted due to the relatively smaller number of primary studies contributing quantity outcome effect estimates.

CBT effects in relation to the timing of follow-up assessment were examined in the present review. Here, outcomes were pooled at 1- to 6-month follow-up (i.e., early) and at 8 months or later (i.e., late). Further, a minority of early follow-up (Carroll et al., 1991; Smout et al., 2010; Papas et al., 2011; Stephens, Roffman, & Simpson, 2000) and late follow-up (Dawe, Rees, Mattick, Sitharthan, & Heather, 2002; Thornton, Gottheil, Patkar, & Weinstein, 2003) studies provided effect estimates prior to 6 and 12 months, respectively. This underscores the nature of findings in the present study as maintenance of CBT effects at follow-up, rather

than initial efficacy at post-treatment. Studies using a no treatment or a minimal treatment contrast provide the optimal conditions for examining the durability of treatment effect. In these cases, studies reporting frequency outcomes demonstrated that CBT effects were quite durable with moderate effects at both early ($k = 5$) and late ($k = 4$) follow-ups. The cognitive-behavioral emphasis on relapse prevention suggests this is a treatment well-suited to abstinence maintenance and long-term functioning. When the contrast condition was a non-specific or specific therapy, then relative durability in contrast to another form of treatment is the effect measure. This relative durability was not demonstrated in the present review.

Perhaps the most important message of this meta-analysis, in concert with others in the literature, is that contrast condition matters when intervention effect magnitude is of interest (e.g., Wampold, 1997; Wampold, Minami, Baskin, & Tierney, 2002). We suggest that future meta-analyses label effect sizes for exactly what they are, that is, effect sizes in contrast to no treatment, assessment only, or other minimal treatment versus effect sizes in relation to another form of treatment. In the present study, estimates of effect were sizable only among the seven studies contrasting CBT with a minimal comparison. When non-specific therapies or usual care were the contrast, the pooled effect size was small to non-significant. In this review, non-specific contrasts were typically either treatment as usual (e.g., Bowen et al., 2014; McKay et al., 2010; Morgenstern, Blanchard, Morgan, Labouvie, & Havaki, 2001; Papas et al., 2011) or conditions designed to account for non-specific therapy factors (e.g., supportive therapy [Burtscheidt, Wölwer, Schwarz, Strauss, & Gaebel, 2002]; didactic education [Kivlahan et al., 1990]). Modest relative efficacy in contrast to these conditions underscores how little we know about the specificity of CBT ingredients when delivered to populations with alcohol or other drug use disorders. A view of Supplemental Table 1 supports this point where non-specific contrasts were quite variable, but often involved addiction information, mutual support, and 12-step program involvement. These are established elements of community-based care and confer benefit in their own right (SAMHSA, 2017).

Non-significant pooled effects, in contrast to a specific therapy, across outcome types and follow-up time point was observed in the present study. As noted above, this type of contrast characterized the majority of studies reviewed despite the known phenomenon of limited evidence for differential efficacy between specific therapies (i.e., *the dodo bird effect*). While it is beyond the scope of this work, an important question for future research is - how or why this phenomenon continues to occur? A range of explanations have been offered including common factors and specific, yet equally effective, factors (e.g., Magill, Kiluk, McCrady, Tonigan, & Longabaugh, 2015), and it could be a combination of both. Such questions are complex, but highly significant for future clinical training, intervention refinement, and community program implementation.

Limitations

The limitations of this study may reflect some of the key trade-offs in meta-analysis. Specifically, our primary goal was to derive valid, random effects estimates characterized by effect modifiers. In other words, the study sought to avoid combining “apples and oranges”

(Wilson, 2000). The trade-off was that some effect estimates were comprised of a small number of primary studies, which could result in underpowered moderator analysis if heterogeneity was present in these pooled effects. In this study, two of 10 subgroup effect sizes showed greater than 40% systematics heterogeneity, and one of these two subgroups had a sufficient number of studies to allow multivariate moderator analysis. CBT frequency outcomes at early follow-up, in contrast to a non-specific therapy, showed smaller effect sizes among studies with older samples. In summary, the derived effect sizes showed minimal heterogeneity, and when heterogeneity was observed, moderator analyses revealed few significant moderating factors possibly due to low statistical power.

Additional limitations are perhaps more conceptual than statistical, but nevertheless reflect potential challenges to the validity of meta-analytic studies of intervention efficacy. The first concerns fidelity and other sources of variability in what comprised the sample of CBT interventions. As noted, we sought homogeneity in how CBT was defined via inclusion of face-to-face CBT not combined with another intervention, whether psychosocial or pharmacological. However, reporting of therapist training (44%), supervision frequency and/or methods (70%), and fidelity (7%) was variable in the sample of studies. As such, the quality of CBT-delivery cannot be assured. Second, study results should be considered in the context of the ongoing debate about what constitutes an optimal outcome in randomized clinical trials with substance use disorders. We selected consumption measures, and favored biological assay variables, but equally meaningful are use consequences and improvements in overall functioning (Kiluk, Fitzmaurice, Strain, & Weiss, 2019). Further, optimal outcomes could vary as a function of intervention modality, including specific ingredients and purported mechanisms of action (Donovan et al., 2012). Therefore, the degree to which the outcomes presented in this review reflect an *ideal endpoint* or merely one kind of endpoint for measurement of CBT efficacy should be considered.

Conclusion

In conclusion, this meta-analysis shows CBT efficacy, in contrast to no or minimal treatment, was moderate and durable over follow-up. Consistent with a number of evidence-based addictions therapies, CBT effect sizes were small to non-significant in contrast to non-specific and specific therapies, respectively. The majority of derived effects were homogeneous, suggesting that the selected subgroup variables (i.e., contrast type, outcome type, and follow-up time point) were informative modifiers of CBT effects[5].

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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[5]Please see Supplemental Information for PRISMA Checklist.

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Public Health Significance:

This meta-analysis provides an up-to-date summary of treatment efficacy in Cognitive Behavioral Therapy (CBT) for alcohol or other drug use disorders. CBT is effective for these conditions with outcomes roughly 15–26% better than average outcomes in untreated, or minimally treated, controls.

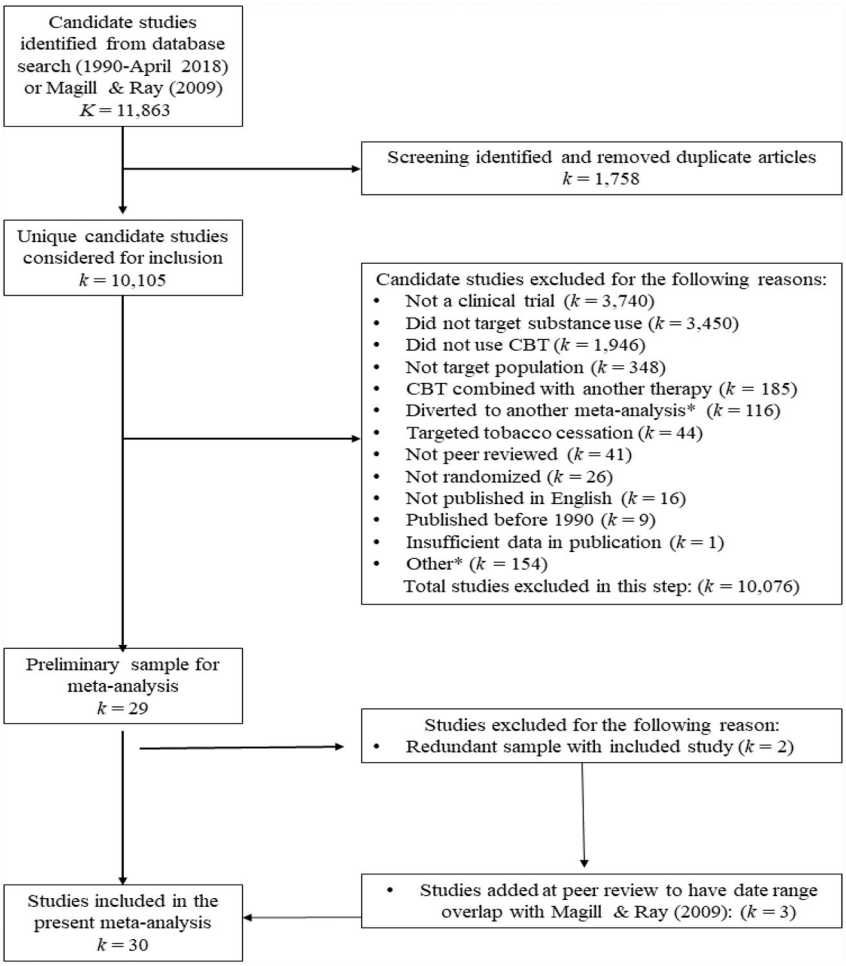

Figure 1.

Flow of primary study inclusion.

Notes. K/k is defined as number of groups. CBT = cognitive behavioral therapy * E.g., dual diagnosis population; couples or self-help format; ineligible control condition.

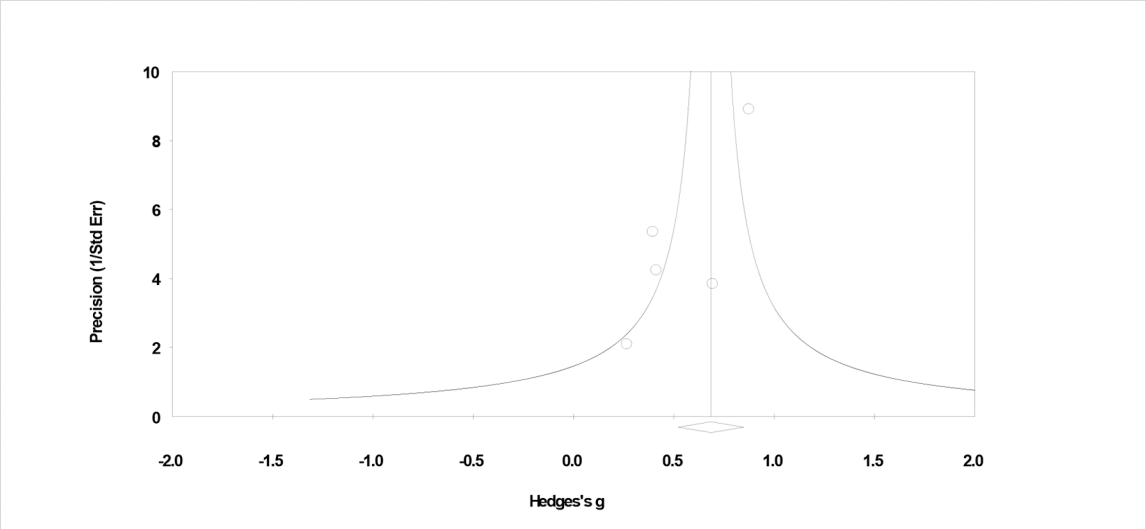

Figure 2.

Plot of assessment of publication bias.

Notes. Assessment of bias in CBT effect in contrast to a minimal condition. The plot shows some asymmetry, but the rank order correlation shows a non-significant relationship between precision and effect size ($\tau = -.33$, $p > .05$).

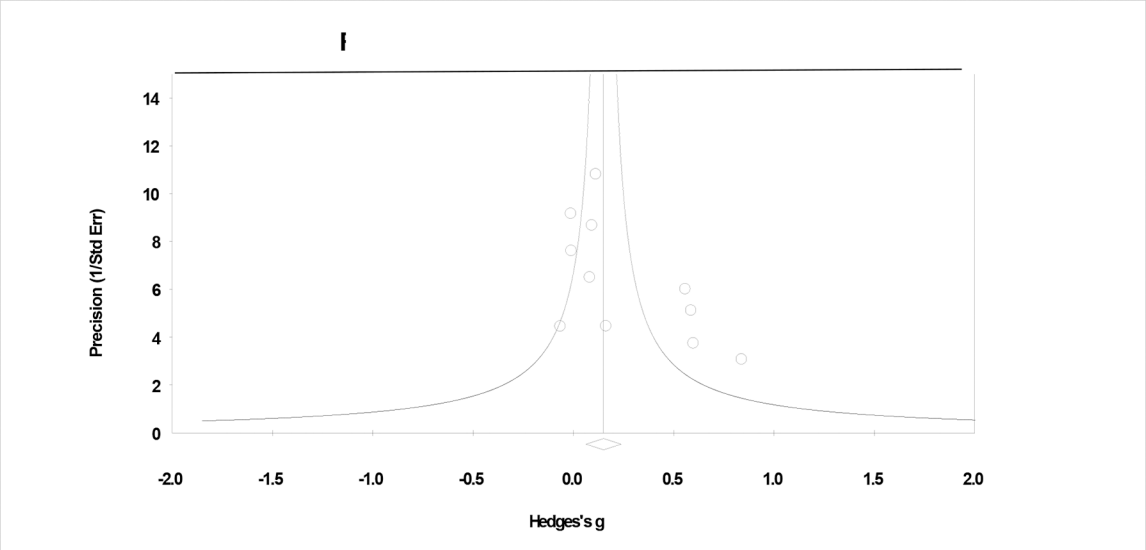

Figure 3.

Plot of assessment of publication bias.

Notes. Assessment of bias in CBT effect in contrast to a non-specific therapy. The plot shows symmetry and the correlation test shows a non-significant relationship between precision and effect size ($\tau = .36$, $p > .05$).

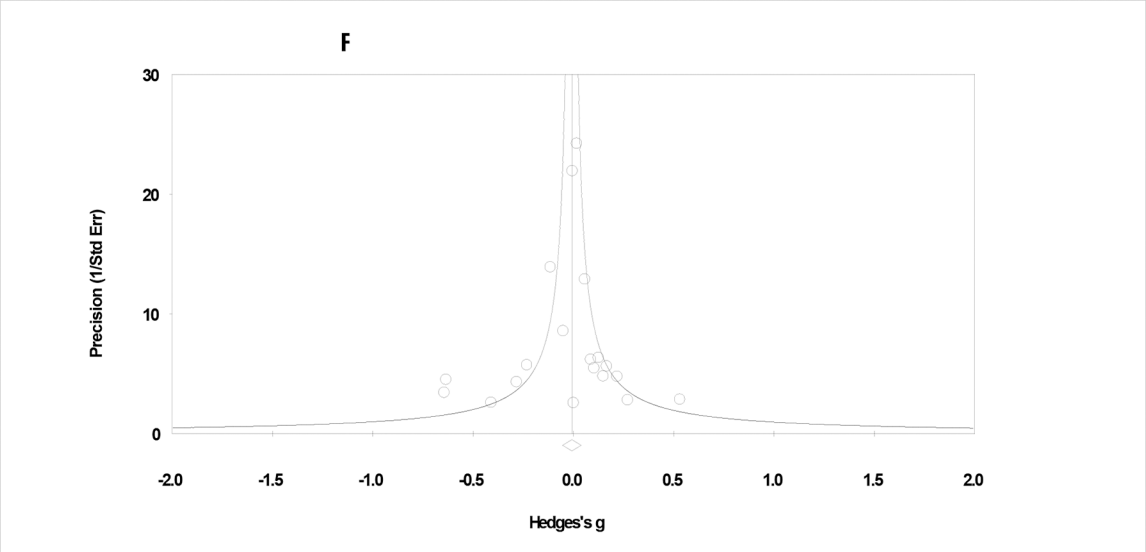

Figure 4.

Plot of assessment of publication bias.

Notes. Assessment of bias in CBT effect in contrast to a specific therapy. The plot shows symmetry and the correlation test shows a non-significant relationship between precision and effect size (τ = .00, p > .05).

Table 1.

CBT efficacy at early follow-up by type of contrast condition

First author (date)	N[1]	Treatment	Contrast	Drug	Follow-up month	Outcome[2]	Risk of Bias[3]	g(se)
Minimal/Waitlist Contrasts								
Kivlahan (1990)	43	alcohol skills training	assessment only	alcohol	4	drinks per week	L	.43(.36)
Lanza (2014)	50	cognitive behavioral therapy	waitlist	polydrug	6	rate abstinent	U	.27(.48)
McAuliffe (1990) - US	88	relapse prevention	referral	opiates	6	rate abstinent	L	.44(.26)
McAuliffe (1990) – HK	80	relapse prevention	referral	opiates	6	rate abstinent	L	.26(.32)
Stephens (2000)	291	relapse prevention	waitlist	marijuana	4	days used	L	1.01(.16)
Stephens (2000)	291	relapse prevention	waitlist	marijuana	4	use per day	L	.75(.16)
Non-Specific Therapy Contrasts								
Bowen (2014)	183	relapse prevention	TAU	polydrug	6	days used	L	.26(.16)
Burtscheidt (2002)	80	coping skills training	non-specific support group	alcohol	6	rate abstinent	U	.12(.26)
Kivlahan (1990)	43	alcohol skills training	didactic alcohol information	alcohol	4	drinks per week	L	.46(.37)
McKay (1997)	98	relapse prevention	group counseling	polydrug	6	days used	L	−.14(.20)
McKay (2004)	257	relapse prevention	group counseling	polydrug	6	rate abstinent	U	.10(.13)
McKay (2010)	75	relapse prevention	TAU	cocaine	6	rate relapse	U	.00(.31)
Monti (1997)	128	coping skills training	attention placebo	cocaine	6	days used	L	.59(.20)
Morgenstern (2001)	168	cognitive behavioral therapy	TAU	polydrug	6	days abstinent	L	.08(.15)
Papas (2011)	75	cognitive behavioral therapy	TAU	alcohol	3	days used	L	.72(.24)
Papas (2011)	75	cognitive behavioral therapy	TAU	alcohol	3	drinks per day	L	.40(.23)
Stephens (1994)	212	relapse prevention	social support group	marijuana	6	days used	U	.01(15)
Specific Therapy Contrasts								
Brown (2002)	133	relapse prevention	twelve-step facilitation	polydrug	6	days to first lapse	L	−.23(.17)
Budney (2006)	60	cognitive behavioral therapy	contingency management	marijuana	6	days used	L	.32(.30)
Carroll (1991)	42	relapse prevention	interpersonal psychotherapy	cocaine	1	rate abstinent	H	.53(.35)
Donovan (1988)	39	relapse prevention	interpersonal process group	alcohol	6	days drank	U	.27(.35)
Heather (2000)	91	behavioral self-control	moderation cue exposure	alcohol	6	days abstinent	L	−.23(.23)
Heather (2000)	91	behavioral self-control	moderation cue exposure	alcohol	6	drinks per day	L	.41(.23)
Kadden (1989)	96	coping skills training	interactional group therapy	alcohol	6	days abstinent	U	.00(.19)
Kadden (2001)	250	cognitive behavioral therapy	interactional group therapy	alcohol	6	days abstinent	U	.17(.18)

First author (date)	N[1]	Treatment	Contrast	Drug	Follow-up month	Outcome[2]	Risk of Bias[3]	*g*(se)
Lanza (2014)	50	cognitive behavioral therapy	acceptance and commitment	polydrug	6	rate abstinent	U	−.41(.38)
Litt (2016)	193	cognitive behavioral therapy	network support therapy	alcohol	6	days abstinent	L	−.18(14)
Litt (2016)	193	cognitive behavioral therapy	network support therapy	alcohol	6	heavy use days	L	−.11(.14)
Maude-Griffin (1998)	128	cognitive behavioral therapy	twelve-step facilitation	cocaine	4	rate abstinent	L	.22(.21)
McKay (2010)	75	relapse prevention	contingency management	cocaine	6	rate relapse	U	−.22(.32)
P. MATCH (1997) - opt.	952	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	6	days abstinent	L	−.05(.08)
P. MATCH (1997) - opt.	952	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	6	drinks per day	L	.09(.08)
P. MATCH (1997) - aft.	774	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	6	days abstinent	L	.06(.09)
P. MATCH (1997) - aft.	774	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	6	drinks per day	L	−.06(.09)
Shakeshaft (2002)	295	cognitive behavioral therapy	brief intervention	alcohol	6	days heavy use	L	−.05(.12)
Sitharthan (1997)	42	cognitive behavioral therapy	cue exposure	alcohol	6	days used	L	−.61(.31)
Sitharthan (1997)	42	cognitive behavioral therapy	cue exposure	alcohol	6	drinks per day	L	−.66(.31)
Smout (2010)	104	cognitive behavioral therapy	acceptance and commitment	meth.	6	rate relapse	H	−.29(.50)
Smout (2010)	104	cognitive behavioral therapy	acceptance and commitment	meth.	6	grams per month	H	.16(.20)
Stephens (2000)	291	relapse prevention	motivational interviewing	marijuana	6	days used	L	.12(.15)
Stephens (2000)	291	relapse prevention	motivational interviewing	marijuana	6	use per day	L	.04(.15)

Notes. $k_{effect\ sizes}$ = 41. TAU = treatment as usual, US = United States, HK = Hong Kong, opt. = outpatient, aft. = aftercare, meth. = methamphetamine.

[1] If an arm of the trial did not contribute an effect contrast, the study-level sample size was adjusted.

[2] Negative outcomes such as days used or number of times used per day were reverse-scored such that a positive effect estimate would reflect a positive treatment outcome.

[3] Cochrane Risk of Bias Tool (Higgins et al., 2011), L = low risk, U = unclear risk, H = high risk.

[4] Active contrasts outcomes were pooled to provide a single estimate per outcome type and follow up for Project MATCH.

Table 2.

CBT efficacy at late follow-up by type of contrast condition

First author (date)	N[1]	Treatment	Contrast	Drug	Follow-up month	Outcome[2]	Risk of Bias[3]	g(se)
Minimal/Waitlist Contrast Studies								
Kivlahan (1990)	43	alcohol skills training	assessment only	alcohol	12	drinks per week	L	.98(.38)
McAuliffe (1990) - US	88	relapse prevention	referral	opiates	12	rate abstinent	L	.35(.27)
McAuliffe (1990) - HK	80	relapse prevention	referral	opiates	12	rate abstinent	L	.60(.35)
Non-Specific Therapy Contrasts								
Bowen (2014)	183	relapse prevention	TAU	polydrug	12	days used	L	−.08(.16)
Burtscheidt (2002)	80	coping skills training	non-specific support group	alcohol	30	rate abstinent	U	.30(.46)
Jones (1982)	45	alcohol skills training	TAU	alcohol	12	days abstinent	U	.82(.46)
Jones (1982)	45	alcohol skills training	TAU	alcohol	12	mean consumption	U	.85(.46)
Kivlahan (1990)	43	alcohol skills training	didactic alcohol information	alcohol	12	drinks per week	L	.75(.38)
McKay (1997)	98	relapse prevention	group counseling	polydrug	24	days used	L	.09(.17)
McKay (2004)	257	relapse prevention	group counseling	polydrug	12	rate abstinent	U	.13(.13)
McKay (2010)	75	relapse prevention	TAU	cocaine	18	rate relapse	U	−.13(.32)
Stephens (1994)	212	relapse prevention	social support group	marijuana	12	days used	U	−.04(.15)
Thornton (2003)	291	Behavioral treatment	facilitative therapy	polydrug	9	ASI[5]	U	−.04(20)
Specific Therapy Contrasts								
Budney (2006)	60	cognitive behavioral therapy	contingency management	marijuana	12	days used	L	−.01(.29)
Dawe (2002)	100	behavioral self-control	moderation cue exposure	alcohol	8	days used	U	.03(.22)
Dawe (2002)	100	behavioral self-control	moderation cue exposure	alcohol	8	days heavy use	U	.22(.22)
Litt (2016)	193	cognitive behavioral therapy	network support therapy	alcohol	27	days abstinent	L	−.14(.14)
Litt (2016)	193	cognitive behavioral therapy	network support therapy	alcohol	27	days heavy use	L	−.03(.14)
McKay (2010)	75	relapse prevention	contingency management	cocaine	18	rate relapse	U	−.36(.33)
P. MATCH (1997) - opt.	952	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	15	days abstinent	L	−.05(.08)
P. MATCH (1997) - opt.	952	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	15	drinks per day	L	.09(.08)
P. MATCH (1997) - aft.	774	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	15	days abstinent	L	.09(.09)

First author (date)	N^1	Treatment	Contrast	Drug	Follow-up month	Outcome[2]	Risk of Bias[3]	*g*(se)
P. MATCH (1997) - aft.	774	cognitive behavioral therapy	motivational interviewing/twelve-step facilitation[4]	alcohol	15	drinks per day	L	−.11(.09)
Sandahl (2004)	49	cognitive behavioral therapy	psychodynamic therapy	alcohol	15	days abstinent	L	−.64(.29)
Stephens (2000)	291	relapse prevention	motivational interviewing	marijuana	16	days used	L	.06(.16)
Stephens (2000)	291	relapse prevention	motivational interviewing	marijuana	16	use per day	L	.02(.16)

Notes. $K_{effect\ sizes}$ = 26. TAU = treatment as usual, US = United States, HK = Hong Kong, opt. = outpatient, aft. = aftercare, meth. = methamphetamine.

[1] If an arm of the trial did not contribute an effect contrast, the study-level sample size was adjusted.

[2] Negative outcomes such as days used or number of times used per day were reverse-scored such that a positive effect estimate would reflect a positive treatment outcome.

[3] Cochrane Risk of Bias Tool (Higgins et al., 2011), L = low risk, U = unclear risk, H = high risk.

[4] Active contrasts outcomes were pooled to provide a single estimate per outcome type and follow up for Project MATCH.

[5] Addiction Severity Index (McLellan et al., 1980).

Table 3.

Study-level predictors of effect size heterogeneity

Model	Beta	*b*	*z*	*p*
Non-Specific, early frequency				
Participant Block				
Mean age of participants	−1.225	−.072	−2.017	.044
Percent female participants	.160	.003	.347	.729
Percent white participants	−.728	−.005	−1.314	.189
Primary drug (reference = drug)	.798	.547	1.636	.102
Substance use severity (reference = dependent)	−.470	−.248	−.969	.332
Q_E (5) = 4.874				
Q_R (3) = 1.183				
Implementation Block				
Treatment format (reference = group)	−.191	−.068	−.453	.651
Treatment length	−.708	−.018	−1.681	.093
Q_E (2) = 2.827				
Q_R (6) = 3.229				
Methodological Block				
Risk of bias (reference = low)	−.451	−.232	−1.110	.267
Q_E (1) = 1.232				
Q_R (7) = 4.824				

Notes. k = 9. Q_E and Q_R = chi-square explained and residual, respectively. Treatment length was measured in number of planned CBT sessions.