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A Meta-Analysis of the Effect of Cognitive Bias Modification on Anxiety and Depression

Lauren S. Hallion and Ayelet Meron Ruscio
University of Pennsylvania

Cognitive biases have been theorized to play a critical role in the onset and maintenance of anxiety and depression. Cognitive bias modification (CBM), an experimental paradigm that uses training to induce maladaptive or adaptive cognitive biases, was developed to test these causal models. Although CBM has generated considerable interest in the past decade, both as an experimental paradigm and as a form of treatment, there have been no quantitative reviews of the effect of CBM on anxiety and depression. This meta-analysis of 45 studies (2,591 participants) assessed the effect of CBM on cognitive biases and on anxiety and depression. CBM had a medium effect on biases ($g = 0.49$) that was stronger for interpretation ($g = 0.81$) than for attention ($g = 0.29$) biases. CBM further had a small effect on anxiety and depression ($g = 0.13$), although this effect was reliable only when symptoms were assessed after participants experienced a stressor ($g = 0.23$). When anxiety and depression were examined separately, CBM significantly modified anxiety but not depression. There was a nonsignificant trend toward a larger effect for studies including multiple training sessions. These findings are broadly consistent with cognitive theories of anxiety and depression that propose an interactive effect of cognitive biases and stressors on these symptoms. However, the small effect sizes observed here suggest that this effect may be more modest than previously believed.

Keywords: cognitive bias modification, attention training, anxiety, depression, meta-analysis

Prominent cognitive theories propose that negative cognitive biases, or a tendency to preferentially process negatively valenced information, play a central role in the onset and maintenance of anxiety and depression (Beck, 1976, 2008; Beck & Clark, 1997; D. A. Clark, Beck, & Alford, 1999; D. M. Clark & Wells, 1995; Eysenck, 1992, 1997; Mathews & MacLeod, 2005; Rapee & Heimberg, 1997; Williams, Watts, MacLeod, & Mathews, 1997). Broadly, these theories posit that biases increase the frequency, intensity, or variety of negative thoughts, which in turn adversely affect emotions and related anxiety and depression symptoms[1] (D. A. Clark & Steer, 1996). Cognitive models of social anxiety disorder, for example, propose that socially anxious individuals selectively attend to negative aspects of their appearance and behavior (e.g., blushing) and to social threat cues (e.g., signs of boredom or frowns). The hypothesized downstream effects of these attention biases include negative self-evaluation, heightened arousal, and increased anxiety (D. M. Clark & Wells, 2005; Rapee & Heimberg, 1997). Psychotherapy interventions grounded in cognitive theory rely in large part on the assumption that cognitive biases are causally related to symptoms. In support of these theories, several decades of research have demonstrated an association between anxiety, depression, and an array of negative cognitive biases (Mathews & MacLeod, 2005).

Although negative attention biases characterize both anxiety and depression, the nature of the biases that characterize these symptoms differs. Clinical and subclinical levels of anxiety are characterized by preferential attention to threatening information (i.e., information that is perceived as potentially threatening to one's physical or psychological well-being; Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van IJzendoorn, 2007; Cisler & Koster, 2010; Heinrichs & Hofmann, 2001; Mathews, Mackintosh, & Fulcher, 1997; Mogg & Bradley, 1998). In the commonly used dot-probe task, a test of attention bias (MacLeod, Mathews, & Tata, 1986), anxious individuals respond more quickly to a probe when it replaces a threatening image (e.g., a spider or an angry face) than when it replaces a neutral or positive image. Conversely, depression is not generally associated with attention bias toward threat. Rather, clinical and subclinical levels of depression are associated with difficulty disengaging attention from mood-congruent (i.e., negative or sad) self-relevant stimuli, as well as with attentional avoidance of positive stimuli (Bradley, Mogg, & Lee, 1997; Mathews & MacLeod, 2005). Additionally, anxiety is associated with biases in the early, automatic stages as well as the later, strategic stages of attention (Bar-Haim et al., 2007), whereas depression is associated with biases only in the later stages (e.g., Gotlib et al., 2004; Joormann, 2004).

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Lauren S. Hallion and Ayelet Meron Ruscio, Department of Psychology, University of Pennsylvania.

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Correspondence concerning this article should be addressed to Lauren S. Hallion, Department of Psychology, University of Pennsylvania, 3720 Walnut Street, Philadelphia, PA 19104. E-mail: hallion@psych.upenn.edu

[1] The term *symptoms* is used throughout the article to refer to subclinical as well as clinical manifestations of anxiety and depression.

A related bias that is frequently linked to anxiety and depression is the tendency to interpret ambiguous stimuli in a negative manner. For example, when presented with an ambiguous sentence (e.g., "*They discussed the priest's convictions*"), anxious individuals are more likely than nonanxious controls to select a threatening interpretation ("*They discussed the priest's criminal record*" vs. the neutral "*They discussed the priest's strong beliefs*"; Eysenck, Mogg, May, Richards, & Mathews, 1991; Richards & French, 1992). Evidence for interpretation biases in depression has been less consistent. Whereas some studies have found that clinically depressed individuals preferentially encode threatening interpretations of ambiguous sentences or homophones (e.g., "die" vs. "dye"; Mogg, Bradbury, & Bradley, 2006), other studies have not found a negative interpretation bias in depression (Bisson & Sears, 2007).

Although a substantial body of research has demonstrated a correlational relationship between cognitive biases and symptoms, many cognitive theories assume a causal relationship (see MacLeod, Campbell, Rutherford, & Wilson, 2004, for a review). Cognitive bias modification (CBM) was originally developed as a way for researchers to experimentally manipulate cognitive biases, permitting an empirical test of the causal role of these biases in anxiety and depression. Most current CBM paradigms were adapted from established paradigms in experimental cognitive psychology. In a typical CBM paradigm, participants are exposed to an experimental contingency between negative emotional stimuli and the target response. For example, in the dot-probe task (MacLeod et al., 1986), two stimuli appear simultaneously on a computer screen. One stimulus is threatening (e.g., an angry face), and one is positive or benign (e.g., a smiling or neutral face). Immediately following the offset of these stimuli, one or two dots (the "probe") appear in the location of one of the stimuli. The participant is required to identify the number of dots (one or two) as quickly as possible. Faster responses to a probe that replaces a threatening stimulus suggest preferential attention toward threatening information. Attention biases can be trained using this paradigm by varying the frequency with which the probe replaces the threatening stimulus. In studies designed to reduce attention bias toward threat, the probe replaces the benign stimulus on 80%–100% of trials. Over the course of many trials, participants are expected to implicitly learn the association between the benign stimulus and the target response and to begin attending selectively to benign stimuli. The success of training is assessed by removing the contingency between the stimulus and probe and examining whether participants who have undergone training continue to demonstrate the trained bias.

Interpretation bias paradigms differ from attention bias paradigms in several respects. Whereas stimuli in attention modification paradigms are typically pictures or words, stimuli in interpretation bias paradigms are typically sentences or paragraphs. Additionally, whereas attention bias paradigms usually require participants to respond to a stimulus by pressing a button, interpretation bias modification paradigms frequently require participants to be generative. For example, in a common interpretation bias training paradigm, participants are presented with a series of ambiguous sentences. The valence of each sentence can be determined only from the last word of the sentence, which is presented as a word fragment that participants must solve (Mathews & Mackintosh, 2000). For instance, participants might be presented with the following ambiguous sentence: "As you get ready to go to a party, you think the new people you meet there will find you . . ." Participants in a positive training group are presented with a fragment that resolves the sentence positively ("fr_e_dly"), whereas participants in a negative training group are presented with a fragment that resolves the sentence negatively ("b_r_ng"). After completing each fragment, participants typically are asked a comprehension question that reinforces the interpretation (e.g., "Will you be liked by your new acquaintances?"). To assess the success of training, participants are presented with new sentences that remain ambiguous even after the word fragment is completed. For example, "As you give a speech at your friend's wedding, you notice some people in the audience starting to . . ." is followed by the word fragment "l_ _gh." Participants are then asked to disambiguate these sentences by selecting one of several different meanings. Participants who choose positively disambiguated sentences (e.g., "As you speak, people in the audience laugh appreciatively") are considered to have developed a positive interpretation bias, whereas those who choose negatively disambiguated sentences (e.g., "As you speak, people in the audience find your efforts laughable") are considered to have developed a negative interpretation bias.

Many early CBM studies tested the causal relationship between cognitive biases and symptoms by inducing negative cognitive biases in healthy populations and assessing the effects of these biases on symptoms (e.g., Mathews & Mackintosh, 2000). These studies tended to find that negative cognitive biases could be induced in otherwise healthy individuals and that these biases increased anxiety and depression. More recently, researchers have investigated whether reducing negative biases (or inducing positive biases) in clinical populations reduces symptoms of anxiety and mood disorders (e.g., Papageorgiou & Wells, 2000; A. Wells, White, & Carter, 1997). These investigations have produced mixed results. Many studies have successfully modified cognitive biases and found corresponding changes in symptoms. For example, T. T. Wells and Beevers (2010) found that in mildly to moderately depressed undergraduate students, CBM reduced attention bias toward negative stimuli, which led to a reduction in depression symptoms. However, other studies have successfully modified cognitive biases with no immediate changes in symptoms. For example, Browning and colleagues (2010) found that healthy volunteers who were trained either to attend to threatening stimuli or to avoid those stimuli did not differ in anxious or depressed affect after training. Still other studies have found an improvement in symptoms among mildly depressed participants treated with CBM even without demonstrable changes in the participants' cognitive biases (Baert, DeRaedt, Schact, & Koster, 2010).

In contrast to conventional paradigms wherein symptoms are assessed immediately posttraining, a subset of CBM studies have been guided by a diathesis-stress conceptualization of cognitive biases. This conceptualization frames negative cognitive biases as a latent vulnerability that has little or no effect on symptoms until the individual encounters a stressor (e.g., Beck, 1987; MacLeod et al., 2004). As such, negative cognitive biases would not be predicted to increase anxiety or depression in situations where no potentially negative information is available. Instead, biases should influence symptoms only in the context of a stressful situation that activates biases, which in turn increase the salience and impact of negative aspects of the situation (e.g., Beck, 2008).

Diathesis-stress theories would predict that the effect of CBM on symptoms will be evident only upon exposure to a potentially stressful or negative situation. Researchers have begun to test this hypothesis by exposing participants to stressful situations and comparing the symptoms of those who have and have not previously received CBM. For example, MacLeod, Rutherford, Campbell, Ebsworthy, and Holker (2002) trained participants to attend to either emotionally negative or neutral words. All participants were subsequently asked to solve a series of extremely difficult anagrams, then were told that a video of their unusually poor performance would be used as a demonstration for a laboratory class. Although all participants reported an increase in anxiety and depression symptoms following this stressor, participants who had been trained to attend to neutral words experienced an attenuated increase compared with participants who were trained to attend to negative words. Using a more naturalistic stressor, Dandeneau, Baldwin, Pruessner, Baccus, and Sakellaropoulo (2007) provided attention training to undergraduate students in the 5 days preceding each participant's final exams. When anxiety and depression symptoms were assessed on the morning of the exams, participants who had been trained to attend to positive stimuli reported lower levels of stress, as well as lower state anxiety following the exams, compared with participants who had received sham training.

Despite the varied conceptualizations and mixed results of previous CBM research, the past decade has witnessed a surge of interest in CBM. CBM was recently featured in a special section of the *Journal of Abnormal Psychology* (Koster, Fox, & MacLeod, 2009), and an increasing number of studies and reviews have noted CBM's promise as an alternative or complementary intervention for anxiety and depression. Given the large number of patients who remain symptomatic after receiving first-line treatments for anxiety and mood disorders (Hofmann & Smits, 2008), the novel approach taken by CBM and its potential to assist patients who are not helped by conventional treatments has generated a groundswell of enthusiasm. Nevertheless, before substantial attention is invested in CBM, a systematic assessment of its effect on anxiety and depression is warranted. This assessment is particularly critical given the discrepancies apparent in the extant literature.

Several narrative reviews have examined the effect of CBM on anxiety and depression (Bar-Haim, 2010; Browning, Holmes, & Harmer, 2010; Mohlman, 2004; Yiend & Mackintosh, 2004). These reviews have generally suggested that cognitive biases can be modified and that reductions in negative cognitive biases lead to reductions in symptoms. However, all but one of these reviews (Yiend & Mackintosh, 2004) focused on attention bias modification, excluding other frequently used CBM paradigms such as interpretation modification. Additionally, narrative literature reviews have several important limitations that can be overcome using quantitative synthesis techniques such as meta-analysis (Rosenthal & DiMatteo, 2001). First, narrative reviews typically rely on statistical significance to determine the efficacy of an intervention, but statistical significance is determined in large part by sample size. By pooling the results of multiple studies, meta-analysis allows researchers to overcome the problem of low statistical power that plagues most psychopathology research. Second, when evaluating potential clinical interventions, it is insufficient to know whether there is a statistically significant difference between treatment and control groups; it is also important to estimate the size of the effect to determine its likely clinical significance. Meta-analysis provides an estimate of effect size. Third, narrative literature reviews are limited in their ability to evaluate the impact of various study characteristics on the efficacy of an intervention. Meta-analysis permits a statistical assessment of the extent to which hypothesized moderators influence the effect size. This advantage of meta-analysis may be particularly important for the CBM literature, which is characterized by substantial variability in study and sample characteristics.

To date, one meta-analysis has been published on the effects of CBM (Hakamata et al., 2010). This meta-analysis examined 12 studies that used the dot-probe paradigm to investigate the efficacy of attention bias training for reducing anxiety symptoms. The authors found a large effect of training on cognitive biases (d = 1.16) and a medium effect of training on anxiety symptoms (d = 0.61). Although this quantitative synthesis represents an important step forward in evaluating cognitive theories of anxiety, its narrow focus on the dot-probe paradigm and on anxiety symptoms excluded a large number of CBM studies from consideration. A more comprehensive quantitative review that encompasses interpretation as well as attention biases, and depression as well as anxiety symptoms, is needed to address unresolved questions about CBM and its underlying theory.

The Present Study

The present meta-analysis sought to establish whether CBM leads to changes in anxiety and depression and to identify factors that predict the degree of change. This research question was pursued with three goals in mind. The first goal was to evaluate the extent to which CBM successfully changes cognitive biases. The premise of CBM—that modifying cognitive biases will produce changes in anxiety and depression symptoms—hinges first on the assumption that CBM can modify the biases that contribute to these symptoms. Recent reviews have concluded that attention biases can be modified through training (Browning, Holmes, & Harmer, 2010; Hakamata et al., 2010). Little is known, however, about the extent to which other kinds of cognitive biases can be trained or whether it is accurate to claim that CBM, broadly construed, successfully changes biases.

The second goal was to evaluate the extent to which CBM changes symptoms of anxiety and depression. Evidence that CBM significantly influences these symptoms would provide important empirical support for models that propose a causal role for cognitive biases in anxiety and depression. Additionally, a small but growing number of clinical trials have examined the efficacy of CBM for treating these disorders in clinical samples (Amir, Beard, Burns, & Bomyea, 2009; Amir, Beard, Taylor, et al., 2009; Baert, DeRaedt, Schact, & Koster, 2010; Schmidt, Richey, Buckner, & Timpano, 2009). Evidence that CBM has a substantial impact on symptoms of these disorders would support the aggressive pursuit of CBM as a potentially valuable intervention, either alone or in combination with existing treatments.

A third, related goal was to investigate hypothesized moderators of the effects of CBM on cognitive biases and on symptoms. Previous research and theory has suggested several potential moderators of the relationship between CBM and changes in symptoms: change in cognitive bias, symptom construct, cognitive bias

targeted, clinical status of the study sample, control group, and number of sessions.

Change in Cognitive Bias

Because the CBM model is predicated on the assumption that changes in biases lead to changes in anxiety and depression, it is essential to establish whether a relationship in fact exists between changes in cognitive biases and subsequent changes in symptoms. Equally important, there is a need to identify the conditions under which CBM most effectively modifies cognitive biases.

Symptom Construct

There are several compelling reasons to consider depression and anxiety together when evaluating the effect of CBM on symptoms. Symptom measures of anxiety and depression correlate at high levels, especially when assessed by self-report in nonclinical samples (e.g., L. A. Clark & Watson, 1991; L. A. Feldman, 1993), as is the case in most CBM studies. For example, the correlation between scores on two commonly used self-report measures—the State-Trait Anxiety Inventory–Trait Version and the Beck Depression Inventory—approaches $r = .70$ in nonclinical samples (e.g., Storch, Roberti, & Roth, 2004; Watson & Kendall, 1989). Associations are also high among diagnosed anxiety and mood disorders, which co-occur frequently in clinical (Brown, Campbell, Lehman, Grisham, & Mancill, 2001) and community (Kessler, 1997; Kessler et al., 2003) samples. Anxiety and depression covary to such an extent that treatment outcome studies for anxiety disorders frequently include measures of depression symptoms, and vice versa (e.g., Stewart & Chambless, 2009; Weisz, McCarty, & Valeri, 2006). Anxiety and depression further share a common pathophysiology, including dysregulation of the hypothalamic–pituitary axis and serotonergic systems (e.g., Binder & Nemeroff, 2010) and are ameliorated by similar pharmacological (e.g., Höschl & Svestka, 2008; Keller, 2003; Ninan, 2003) and psychosocial (e.g., G. Feldman, 2007; Moses & Barlow, 2006; Olatunji, Cisler, & Deacon, 2010) treatments.

Nevertheless, evidence that certain cognitive biases are selectively associated with anxiety or depression raises the possibility that modification of these biases may have specific, rather than general, effects on symptoms. For example, selective attention to threat is considered specific to anxiety, whereas selective attention to sad stimuli is considered specific to depression (Bar-Haim et al., 2007; Hankin, Gibb, Abela, & Flory, 2010). By contrast, other cognitive biases, such as a tendency to interpret ambiguous scenarios as negative, appear to characterize both anxiety and depression (Mathews & MacLeod, 2005). Given the overlap in some cognitive biases that characterize anxiety and depression and the high covariation between the symptoms themselves, it is possible that training designed to modify only one cognitive bias could influence symptoms of both anxiety and depression.

Cognitive Bias Targeted

Cognitive theories of anxiety and depression often posit a role for both attention and interpretation biases in the onset and maintenance of these syndromes (e.g., Beck, Emery, & Greenberg, 1985; D. M. Clark, 1986; A. Wells, 1995). Therefore, one might predict that manipulating either attention or interpretation biases would have an effect on symptoms. However, the lack of previous systematic reviews leaves open the possibility that CBM may not be effective for changing certain types of biases (e.g., interpretation biases) or that symptoms may not be affected even when those biases are successfully modified.

Clinical Status of the Study Sample

The majority of CBM studies have been conducted using healthy or unselected samples. Unfortunately, studies designed to assess the effect of CBM on anxiety and depression may be constrained by the already-low symptom levels present in these samples, whereas studies using analogue or clinical samples may be better positioned to demonstrate a positive effect of CBM on symptoms. Conversely, one could plausibly predict a smaller effect of CBM in clinical samples if the intervention is not sufficiently powerful to modify severe symptoms or deeply entrenched biases. In order to consider CBM as a potential treatment for anxiety and mood disorders, it is essential to establish whether CBM reduces symptoms in clinical samples.

Control Group

The studies included in this meta-analysis typically employed one of two types of control group: either a group that received the opposite training relative to the treatment group (e.g., negative biases were reinforced) or a group receiving "no contingency" sham training (e.g., negative and positive biases were reinforced with equal frequency). Assuming that CBM influences anxiety and depression by modifying cognitive biases, studies wherein the treatment and control groups received opposite training would be expected to show greater differences in cognitive biases, and therefore greater differences in anxiety and depression, compared with studies wherein only the treatment group received contingency-manipulated training.

Number of Sessions

Treatments for anxiety and depression typically require a number of sessions before improvements in symptoms can be detected (e.g., Forde et al., 2005). Therefore, cognitive bias training that occurs over multiple sessions might be predicted to have a larger and more stable effect on cognitive biases (and therefore on symptoms) than would training that is completed in a single session. Evidence that multiple sessions produce greater change in symptoms than does a single session would not necessarily provide evidence of causality; however, a dose–response relationship would be consistent with causal accounts. In addition, examining this potential moderator is important clinically for establishing the number of sessions required to produce a sizeable and stable effect on symptoms.

Method

Literature Search

Relevant studies were identified through a search of the PsycINFO, PubMed, and MEDLINE databases through October

2010 using combinations of the keywords *cognitive bias modification, attention* bias modification, interpretation bias modification, attention training*, and *bias training*, paired with *anx**, *depress**, and *dysphori**. The same databases were also searched using the names of researchers who frequently publish in the CBM field. The reference sections of all eligible articles and relevant journals were hand-searched for potentially eligible studies. In an effort to reduce bias that might result from publication bias (or the "file drawer problem"), unpublished doctoral dissertations were included if they fulfilled all other study eligibility criteria. This led to the inclusion of one doctoral dissertation (Wadlinger, 2009). Additionally, nine researchers who frequently publish in this research area (three or more studies) were contacted and asked whether they had any unpublished studies pertinent to the research question. No researchers supplied unpublished studies that met the eligibility criteria.

Selection of studies. The search procedure led to the identification of 106 records. These titles were reviewed, and 88 potentially relevant abstracts were obtained and screened using the inclusion criteria described in the next sections. From these abstracts, 73 articles were identified as containing potentially eligible studies. The full text of these articles was obtained and reviewed. The following criteria were then applied to select studies for the meta-analysis:

Design. The study included at least one experimental group in which a cognitive bias (e.g., attention to threat) was modified, as well as at least one control group. If training was provided to the control group, the training was designed to be inert (i.e., sham or no-contingency training) or to have the opposite effect relative to training for the trained group (e.g., to induce a negative bias). Single case series were not eligible for inclusion. This criterion led to the exclusion of 13 records.

Method of bias modification. Cognitive biases were directly targeted through training. Studies that manipulated cognitive biases using a method other than direct training (e.g., mood induction, psychotropic medication, associative conditioning using punishment) were not eligible. This criterion led to the exclusion of two records.

Symptom assessment. The study assessed anxiety- or depression-relevant symptoms (including anxious or depressed mood) using clinician-administered, self-report, physiological, and/or behavioral measures at least once after training. A list of the anxiety and depression measures used by each study is included in Table 1. This criterion led to the exclusion of six records.

Sample. The study used a psychologically healthy adult sample or an unselected sample from a generally healthy adult population (e.g., undergraduate students), or selected adult participants exclusively on the basis of anxiety or mood disorder diagnosis (e.g., diagnosis of generalized anxiety disorder) or other anxiety- or depression-related measures (e.g., low, moderate, or high trait anxiety). Studies that employed different or additional inclusion criteria (e.g., diagnosis of attention-deficit/hyperactivity disorder or schizophrenia, history of alcohol dependence, high preexisting levels of a particular cognitive bias) were not eligible. This criterion was applied to ensure that the results of this meta-analysis could be generalized to the populations most likely to receive CBM as an intervention for anxiety or depression. This resulted in the exclusion of six records.

Additional interventions. CBM was the sole treatment administered to all participants during the study. Studies that included another active treatment (e.g., mindfulness training), either in addition to CBM or as the primary comparison condition, were excluded. Studies in which all participants were currently receiving psychological treatment provided outside the context of the study were also excluded. This criterion was applied in order to reduce any obscuring or attenuation of the CBM effect size that might occur as a result of including another active treatment and to ensure that any observed changes in symptoms were in fact attributable to CBM. This resulted in the exclusion of two records.

Study stimuli. Training stimuli included positive or negative emotionally relevant stimuli (e.g., facial expressions, strongly valenced words) or anxiety- or depression-specific stimuli (e.g., pictures of spiders in spider phobia studies). Studies that trained attention toward or away from another specific class of stimuli (e.g., food, body shape, cigarettes, alcohol) were not eligible. This criterion led to the exclusion of three records.

Available data. Sufficient data were provided to calculate an effect size comparing the treatment and control groups on symptoms after training. Effect sizes were determined using group means and standard deviations; t, F, or chi-square values from between-group analyses; precise p values and degrees of freedom from between-group analyses; or other effect size values (e.g., correlation coefficients) reported in the text (Borenstein, Hedges, Higgins, & Rothstein, 2005; Lipsey & Wilson, 2001). When these data were not reported in the text of the article, the authors were contacted and additional data were requested. Data were requested for 20 studies and were received for 10 of those studies. If data necessary to calculate an effect size were not received, the study was excluded. This resulted in the exclusion of two records.

These selection criteria resulted in the inclusion of 39 articles, which included 45 eligible studies with 2,591 participants.

Coding System and Coding Decisions

Two sets of effect sizes were compiled. The first set (the posttest data set) included all studies that assessed symptoms immediately after training ($k = 43$). The second set (the stressor data set) included only those studies that assessed symptoms after participants were exposed to a stressor ($k = 20$), whether laboratory-induced ($k = 18$) or naturalistic ($k = 2$). The latter data set was compiled to test the hypothesis that the effects of CBM on anxiety and depression are apparent only in interaction with stress.

A standardized coding system was applied to every study. All study coding was completed by the first author. To minimize the potential for bias, the author coded eligible studies in two waves, first coding study characteristics, moderator variables, and the effect size for change in cognitive bias for all studies and then coding the anxiety and depression effect sizes. To assess interrater reliability, an advanced graduate student independently coded a randomly selected sample of eligible studies (33% of studies) using the standardized coding system. Reliability for the anxiety and depression effect size data and the categorical moderator data was calculated using intraclass coefficient and Cohen's kappa (Cohen, 1960), respectively. Reliability was .99 for the symptom measures and .92 for the moderators, indicating a high level of agreement.

Table 1
Study Characteristics and Weighted Mean Effect Sizes

Study	Sample	*N*	Bias	Control group	Anxiety measures	Depression measures	Posttest *g*	Poststress *g*
Amir, Beard, et al. (2009)	GAD diagnosis	29	A	No contingency	STAI-S/T, PSWQ, WDQ, HRSA	BDI-II, HAM-D	0.56	
Amir, Beard, Taylor, et al. (2009)	Social phobia diagnosis	44	A	No contingency	STAI-T, SPAI-SP, LSAS	BDI-II, HAM-D	0.38	
Amir et al. (2010)	Analogue social phobia	57	I	No contingency	STAI-S, behavioral		−0.08	
Amir et al. (2008)	Analogue social phobia	94	A	Negative	STAI-S		0.15	0.35†
Baert et al. (2010, Study 1)	Analogue MDD	48	A	No contingency	POMS (tension), MASQ (AA, GDA)	BDI-II, RRS, POMS (depression, anger, fatigue, vigor), MASQ (AD, GDD)	−0.15	
Baert et al. (2010, Study 2)	MDD diagnosis	35	A	No contingency	POMS (tension), MASQ (AA, GDA)	BDI-II, RRS, POMS (depression, anger, fatigue, vigor), MASQ (AD, GDD)	0.27	
Beard & Amir (2008)	Analogue social phobia	27	I	No contingency	STAI-T, SPAI-SP	BDI-II	0.51	
Browning, Holmes, Murphy, et al. (2010)	Healthy adults	53	A	Negative	STAI-S, anxiety VAS, relaxed VAS	Sadness VAS, happiness VAS	−0.19	
Dandeneau et al. (2007, Study 2b)	Unselected undergraduates	147	A	Other	POMS (anxiety)	Rosenberg SES	0.01	
Dandeneau et al. (2007, Study 3a)	Unselected undergraduates	25	A	Other	STAI-S, PSS	Rosenberg SES		1.05**
Dandeneau et al. (2007, Study 3b)	Telemarketers	23	A	Other	PSS	Rosenberg SES	0.52	
Eldar & Bar-Haim (2010)	High and low TA	60	A	No contingency	STAI-S		0.37	
Hayes et al. (2010)	High worriers	40	A	No contingency	Anxiety VAS, behavioral (×2)	Depression VAS	0.11	0.50
Hazen et al. (2009)	High worriers	23	A	No contingency	STAI-T, PSWQ	BDI-II	0.58†	
Hirsch et al. (2009)	High worriers	40	I	No contingency	Anxiety VAS, behavioral	Depression VAS	0.35	0.14
Hirsch et al. (2007)	Unselected undergraduates	24	I	Negative	STAI-S, other self-report		0.53	0.82*
Holmes & Mathews (2005, Study 2)	Community volunteers (20 > STAI-T < 45)	43	I	Negative	STAI-S/T		0.10	
Hoppitt et al. (2010)	Unselected community volunteers	95	I	Negative	STAI-S		−0.07	0.03
Johnson (2009)	Unselected undergraduates	109	A	No contingency	Other self-report	Other self-report	−0.17	0.08
Koster et al. (2010)	Unselected undergraduates	48	A	No contingency	STAI-T	BDI-II	−0.01	
Krebs et al. (2010)	Low and moderate worriers	64	A	Negative	Anxiety VAS, behavioral	Depression VAS	−0.31	0.07
Lang et al. (2009)	Unselected community volunteers	48	App	Neutral	Behavioral	PANAS-NA	0.43	0.22
Lange et al. (2010, Study 1)	Moderate TA	68	I	Negative	STAI-S, LSAS		0.05	
Lange et al. (2010, Study 2)	Moderate TA	39	I	Negative	STAI-T, LSAS		0.07	
Li et al. (2008)	Analogue social phobia	22	A	No contingency	SIAS, SPS, FNES		0.24	
Mackintosh et al. (2006, Study 2)	Healthy community volunteers	40	I	Negative	STAI-S		0.76*	0.57†
MacLeod et al. (2002, Study 1)	Moderate TA	64	A	Negative	Anxiety VAS	Depression VAS	−0.02	0.34
MacLeod et al. (2002, Study 2)	Moderate TA	64	A	Negative	Anxiety VAS	Depression VAS	0.07	0.60*

(table continues)

Table 1 (*continued*)

Study	Sample	*N*	Bias	Control group	Anxiety measures	Depression measures	Posttest *g*	Poststress *g*
Mathews et al. (2007)	High TA	39	I	Other	STAI-S/T		0.52†	
Murphy et al. (2007)	Analogue social phobia	44	I	No contingency	STAI-S, other self-report		0.64*	0.65*
Najmi & Amir (2010)	Analogue OCD	52	A	No contingency	STAI-S, MOCI, other self-report, behavioral		—	0.03
Reese et al. (2010)	Analogue spider phobia	41	A	No contingency	Anxiety VAS, SPQ, SUDS, behavioral	Sadness VAS	0.19	0.08
Salemink & van den Hout (2010)	Moderate TA	82	I	Negative	Anxiety VAS		−0.17	
Salemink et al. (2007a)	Unselected undergraduates	118	I	Negative	STAI-S		0.35†	—
Salemink et al. (2007b)	Unselected undergraduates	81	I	Negative	STAI-S/T		0.23	
Schmidt et al. (2009)[a]	Diagnosed generalized social phobia	36	A	No contingency	STAI-T, SPAI, LSAS, BSPS	BDI-II	2.10**	
See et al. (2009)	Singaporean students studying overseas	40	A	No contingency	STAI-T			0.64*
Standage et al. (2009)	Unselected undergraduates	48	I	Negative	Anxiety VAS	Depression VAS	0.20	−0.00
Steinman & Teachman (2010)	High anxiety sensitivity	50	I	No contingency	ASI, BBSIQ, PANAS-FS, behavioral		0.16	0.19
Teachman & Addison (2008)	Analogue spider phobia	61	I	No contingency	PANAS-FS	PANAS-Sadness	0.12	
Wadlinger (2009) [Dissertation]	Unselected undergraduates	69	A	Neutral	STAI-S/T	PANAS-NA, CES-D, self-report other	0.03	
Wells & Beevers (2010)	Mild to moderate depression symptoms	31	A	No contingency	BAI	BDI-II	0.33	
Wilson et al. (2006)	Unselected undergraduates	48	I	Negative	Anxiety VAS	Depression VAS	−0.35	0.24
Yiend et al. (2005, Study 1)	Low TA, no psychiatric history	20	I	Negative	STAI-S/T		0.72†	
Yiend et al. (2005, Study 2)	Low TA, no psychiatric history	24	I	Negative	STAI-S/T		0.15	
Yiend et al. (2005, Study 3)	Low TA, no psychiatric history	19	I	Negative	STAI-S		0.22	

Note. Dashes indicate missing data. GAD = generalized anxiety disorder; A = attention bias; STAI-S/T = State-Trait Anxiety Inventory (state/trait version); PSWQ = Penn State Worry Questionnaire; WDQ = Worry Domains Questionnaire; HRSA = Hamilton Rating Scales for Anxiety; BDI-II = Beck Depression Inventory–II; HAM-D = Hamilton Rating Scales for Depression; SPAI-SP = Social Phobia and Anxiety Inventory–Social Phobia; LSAS = Liebowitz Social Anxiety Scale; I = interpretation bias; behavioral = behavioral task; MDD = major depressive disorder; POMS = Profile of Mood States; MASQ (AA, GDA, AD, GDD) = Mood and Anxiety Symptoms Questionnaire (Anxious Arousal, General Distress Anxiety, Anhedonic Depression, General Distress Depression, respectively); RRS = Ruminative Responses Scale; VAS = Visual Analogue Scale; Rosenberg SES = Rosenberg Self-Esteem Scale; PSS = Perceived Stress Scale; TA = trait anxiety; App = appraisals; PANAS-NA/FS = Positive and Negative Affect Scale–Negative Affect/Fear Scale; SIAS = Social Interaction Anxiety Scale; SPS = Social Phobia Scale; FNES = Fear of Negative Evaluation Scale; OCD = obsessive–compulsive disorder; MOCI = Maudsley Obsessive-Compulsive Inventory; SPQ = Spider Questionnaire; SUDS = Subjective Units of Distress; BSPS = Brief Social Phobia Scale; ASI = Anxiety Sensitivity Inventory; BBSIQ = Brief Body Sensations Interpretation Questionnaire; CES-D = Center for Epidemiologic Studies Depression Scale; BAI = Beck Anxiety Inventory.

[a] Outlier.

† $p < .10$. * $p < .05$. ** $p < .01$.

Additional coding decisions were made as follows:

1. In studies that included multiple treatment groups (e.g., attend-to-positive and attend-to-benign) in addition to a control group, a weighted mean of the treatment groups' scores was computed. This decision was made on the grounds that there is little theoretical or clinical justification for distinguishing the relative benefits of attention to one particular form of nonnegative information (i.e., benign vs. positive information).

2. When a study assessed symptoms more than once posttraining, the effect size was calculated using the scores obtained closest to the completion of training. For studies that assessed symptoms following a stressor, the effect size was calculated using the scores obtained closest to the onset of the stressor.

3. One study included two control groups, one of which received sham (i.e., no contingency) training and one of which was a waiting list control. In this case, only the sham training control group was used to calculate effect sizes. This decision was made with the goal of reducing effect size inflation attributable to nonspecific effects of the training procedures.

4. Only one study (Lang, Moulds, & Holmes, 2009) targeted a cognitive bias other than attention or interpretation. This cognitive bias (appraisals) was recoded as an interpretation bias for the purpose of examining the moderating effect of bias targeted.[2]

Independence of effect sizes. In order to meet the statistical assumption of independence of effect sizes, several steps were taken to ensure that each study contributed only one effect size to each set of analyses (Lipsey & Wilson, 2001). First, when data drawn from a single sample were reported in more than one article, the studies were considered statistically dependent and the most complete study report was used to compute a single effect size for the study. Additionally, because many studies assessed symptoms both after a stressor and immediately following training, stressor effect sizes were coded and analyzed in a separate data set so that both effect sizes could be used. Finally, many studies included several symptom measures at each assessment point. When multiple symptom measures were collected, a single arithmetic mean of all relevant effect sizes was included in the analyses.[3] In order to examine any differences in the effects of CBM on anxiety compared with depression, additional data sets were compiled, one of which solely included anxiety measures and one of which solely included depression measures, for each of the two data sets.

Treatment of missing data. If a study was missing the necessary data to calculate an effect size for one or more symptom measures but at least one effect size could be calculated on the basis of the study report or on data provided on request by the study's authors, the average of all calculable effect sizes was used. Two otherwise eligible articles (seven studies) did not have sufficient data to calculate any effect size after taking these steps and were excluded from the analyses. Sensitivity analyses were computed to assess for potential biasing effects of missing data on the effect size estimate. For both sets of effect sizes, a conservative adjusted effect size estimate was computed by imputing an effect size of $g = 0.00$ for studies that were excluded solely because of missing data, as well as for the symptom measures for which no data could be obtained in studies included in the meta-analysis (Cooper, 2010; Lipsey & Wilson, 2001).

Study quality. Studies were coded on several quality indicators (random assignment to condition, blinding of participants and experimenters, the conservativeness of the control condition, and attrition rate). However, several factors led to our decision not to use these quality scores as weights in the present analyses. First, due to the experimental nature of the CBM literature, there was little variability among the studies on a number of important methodological attributes. For example, although randomization to experimental condition was not required, only $k = 2$ studies reported that participants were not randomized to experimental condition. Second, several quality-related variables were not applicable (e.g., attrition rate for studies that included only one training session) or were not described (e.g., blinding of experimenters) for a majority of study reports. Third, other quality-related variables (e.g., blinding of participants) were intentionally manipulated in several studies. For example, Krebs, Hirsch, and Mathews (2010) compared the effect of minimal versus explicit instructions (i.e., blind vs. open-trial) on the extent to which CBM changed cognitive biases and symptoms. Studies that systematically evaluated the effect of these conventions in order to inform practice and theory were not considered less rigorous than studies that chose to follow these conventions. The final quality-related variable considered here (type of control condition) was assessed independently as a potential moderator.

Meta-Analytic Procedures

All effect sizes were coded such that a positive effect size reflected lower anxiety and depression in the treatment group relative to the control group. Hedges's g (Hedges, 1981) was employed for all analyses. The conventions typically used to interpret Cohen's d can be applied to Hedges's g: An effect size of 0.2 is considered small, 0.5 is considered moderate, and 0.8 is considered large (Cohen, 1988). Weighted mean effect sizes, heterogeneity analyses, and moderator analyses were conducted using Comprehensive Meta-Analysis, Version 2.2.046 (Borenstein et al., 2005). For both data sets, Hedges's g was computed using the following formula:

$$g = c_m\left[\frac{M_T - M_c}{SD_p}\right],$$

where the pooled standard deviation is defined as

$$SD_p = \sqrt{\frac{(n_T - 1)SD_T^2 + (n_c - 1)SD_c^2}{(n_T + n_c - 2)}}$$

and where c_m, a correction for small sample bias, is defined as

$$c_m = 1 - \frac{3}{4(n-1)}.$$

[2] The results did not differ significantly when the Lang et al. (2009) study was excluded from this analysis.

[3] Exceptions to this coding decision occurred when a study provided data for global outcome measures that assessed both anxiety and depression, in addition to individual anxiety and depression measures. In this case, the global effect size measure was excluded from effect size calculations. This coding decision was made in order to facilitate testing of the outcome construct (anxiety vs. depression) as a potential moderator of the effect size.

Weighting of studies. Because studies with larger samples provide a more precise estimate of the effect size of interest, each study was weighted by the reciprocal of its squared error (an estimate of within-study variance) and tau-squared (an estimate of between-study variance). The standard error of *g* was calculated using the following formula:

$$SE_g = c_p\left(\sqrt{\frac{1}{N} + \frac{d^2}{2N}}\right)\left(\sqrt{2(1 - \rho)}\right)$$

Because few studies included the pretest–posttest correlation (ρ) for the eligible symptom measures, the test–retest reliabilities reported in the manuals or original articles for each measure were imputed (Lipsey & Wilson, 2001). If the pretest–posttest correlation could not be identified, a conservative estimate of ρ = .70 was imputed (e.g., Hofmann, Sawyer, Witt, & Oh, 2010).

Outliers. Final effect sizes ≥3 *SD* above or below the weighted mean effect size estimate in each data set were identified as outliers. One outlier was identified in the posttest data set (Schmidt et al., 2009). Sensitivity analyses were conducted to assess the robustness of findings when including versus excluding this outlier. The results excluding the outlier are presented here, on the grounds that outliers can be argued to estimate a different population mean than the mean estimated by the remaining effect sizes (Lipsey & Wilson, 2001). Except where noted, the results presented here did not differ significantly when the outlier was included.

Homogeneity of effect sizes. In meta-analysis it is necessary to test the assumption that the effect sizes included in each data set estimate the same population mean (Cooper, 2010; Lipsey & Wilson, 2001). Both data sets were tested for homogeneity of effect sizes using the *Q* statistic (Hedges & Olkin, 1985) and the I^2 statistic (Cooper, 2010; Higgins & Thompson, 2002; Huedo-Medina, Sanchez-Meca, Marin-Martinez, & Botella, 2006; Lipsey & Wilson, 2001). A significant *Q* statistic suggests that the distribution of effect sizes around the mean is greater than would be predicted from sampling error alone. The I^2 statistic quantifies the degree of heterogeneity by estimating the percentage of the variance that is attributable to between-studies variability (as opposed to within-studies sampling error), with percentages of I^2 = 25, 50, and 75 indicating low, moderate, and high degrees of heterogeneity, respectively (Higgins & Thompson, 2002). Some heterogeneity was expected for the current set of analyses in light of the notable variability between studies on such characteristics as the clinical status of the study sample, the cognitive biases targeted for training, and the number of training sessions administered.

Publication bias. Publication bias presents a serious concern for researchers conducting meta-analysis. Because studies with small effect sizes or nonsignificant results are less likely to be published, these studies are often systematically excluded from meta-analyses, leading to an inflated estimate of the overall effect size. Several steps were taken to reduce the potential impact of publication bias. As noted, unpublished doctoral dissertations were considered for inclusion. Additionally, authors who publish frequently in the field were contacted and unpublished studies were requested. Finally, when the necessary data to calculate an effect size were missing from published articles, authors were contacted and the missing data were requested.

Prior to each set of analyses, publication bias was assessed in two ways. First, a funnel plot, which plots the standard error for each study (determined by the study's sample size) against the study's effect size, was created and visually examined for both data sets. Studies with larger sample sizes provide more reliable estimates of the veridical effect size and therefore should cluster around the mean and toward the top of the plot, whereas smaller studies tend to show considerably more variability and therefore should scatter more widely around the mean and toward the bottom of the plot, leading to an inverted funnel shape. In the presence of publication bias, the plot becomes asymmetrical, typically with fewer small-sample-sized studies than would be predicted falling below the mean effect size. Second, Duval & Tweedie's (2000) trim-and-fill procedure was applied for both data sets. This procedure calculates the likely number of missing studies on the basis of the asymmetry in the funnel plot and produces an effect size and confidence interval that is adjusted to account for these missing studies. An important caveat to the present use of these procedures is that both funnel plots and the trim-and-fill procedure rely on the assumption of homogeneity of effect sizes. In heterogeneous data sets, the use of these techniques would violate the assumption and their results should be interpreted with particular caution.

Moderator analyses. Variables hypothesized a priori to be systematically associated with effect sizes were subjected to moderation analysis. Although the CBM model proposes that change in cognitive bias mediates the relationship between CBM and change in symptoms, the studies included here did not provide sufficient data on the relationship between extent of change in biases and extent of change in symptoms to permit mediation analysis. We therefore elected to investigate this relationship indirectly by evaluating the extent of bias change as a moderator of CBM's effect on symptoms (i.e., by examining whether studies that produced a larger change in cognitive biases also produced a larger change in symptoms). Additional moderator variables were also examined. We first examined the moderating effects of symptom construct (anxiety vs. depression), followed by the cognitive bias targeted (attention vs. interpretation). We then examined the clinical status of the study sample. The sample for each study was classified into one of the following three categories: (1) healthy or unselected sample, (2) analogue sample (i.e., diagnosed with an anxiety or mood disorder using a questionnaire) or sample with elevated anxiety or depression symptoms (e.g., high trait anxiety), or (3) clinical sample (i.e., diagnosed with an anxiety or mood disorder via clinical interview). Subsequently we examined the type of control group used (sham vs. negative training). Finally, we examined the number of training sessions administered. The distribution of number of training sessions was skewed, with the majority of studies administering only one training session. Therefore, studies were grouped into those that administered only one training session and those that administered more than one session. Categorical variables (except anxiety vs. depression) were tested using a mixed-effects meta-analytic categorical test, the meta-analytic equivalent of analysis of variance. In order to preserve independence of effect sizes, separate anxiety and depression effect size estimates were computed and compared. Continuous variables were tested as potential moderators using unrestricted maximum likelihood metaregression.

Results

Table 1 provides descriptive information for each study included in the meta-analysis. There was no evidence of heterogeneity in the effects of CBM on symptoms in either the posttest, $Q(43) = 37.19$, $p = .64$, $I^2 = 0.00$, or stressor, $Q(19) = 15.45$, $p = .63$, $I^2 = 0.00$, data sets. All analyses presented were conducted using a random effects model.

Extent and Moderators of Change in Cognitive Bias

A single weighted mean effect size was calculated, using the formula and parameters specified earlier, to compare the experimental and control groups in each study on the extent to which they displayed the targeted cognitive bias after training. CBM had a medium effect on cognitive biases ($g = 0.49$, 95% CI = [0.36, 0.63]), $Q(33) = 110.68$, $p < .001$, $I^2 = 70.18$. When sensitivity analyses were conducted by imputing an effect size of $g = 0.00$ for all missing effect sizes, the effect remained significant ($g = 0.41$, [0.27, 0.56]), $Q(44) = 140.46$, $p < .001$, $I^2 = 67.96$. Although there was some evidence of publication bias, the effect size estimate was significant, although smaller, after trim-and-fill analyses ($g = 0.34$, [0.15, 0.54], $Q = 139.50$). This finding should be interpreted with caution, however, because the homogeneity assumption of the trim-and-fill procedure was violated for this set of effect sizes.

Before examining the relationship between change in bias and change in symptoms, we tested potential moderators of the extent to which CBM successfully modified biases. CBM was significantly more effective at modifying interpretation biases ($k = 25$, $g = 0.81$, 95% CI = [0.59, 1.03]) than attention biases ($k = 15$, $g = 0.29$, [0.11, 0.50]), $Q(1) = 10.71$, $p = .001$. After imputing g = 0.00 for missing values, the point estimates for change in interpretation ($g = 0.56$, [0.35, 0.80]) and attention ($g = 0.16$, [0.05, 0.27]) were reduced but remained significantly different from each other, $Q(1) = 10.33$, $p = .001$. The effect of CBM on cognitive biases did not vary as a function of clinical characteristics of the sample, $Q(2) = 1.59$, $p = .45$; number of training sessions administered, $Q(1) = 0.27$, $p = .60$; or type of control group employed, $Q(1) = 1.02$, $p = .31$. These hypothesized moderators remained nonsignificant after sensitivity analyses, all $Q(1–2) \leq 3.52$, all $p \geq .17$.

Extent and Moderators of Change in Symptoms

CBM had a small but significant effect on symptoms in the posttest ($g = 0.13$, 95% CI = [0.05, 0.21], $p < .001$), $Q(42) = 37.19$, $p = .64$, and stressor ($g = 0.23$, [0.11, 0.34], $p < .001$), $Q(19) = 25.45$, $p = .69$, data sets. When sensitivity analyses were conducted by imputing an effect size of $g = 0.00$ for all missing effect sizes, the effect sizes for the posttest ($g = 0.11$, [0.03, 0.18]), $Q = 37.11$, $p = .93$, and stressor ($g = 0.21$, [0.10, 0.32]), $Q = 15.49$, $p = .69$, data sets remained small but significant.

Although these overall effect sizes did not differ significantly from one another, the findings for the posttest data set were qualified by strong evidence of publication bias. The funnel plots were asymmetric for this data set, suggesting missing studies with effect sizes below the mean (see Figure 1) and hinting that the observed effect sizes may reflect inflated estimates of the true effect of CBM on symptoms. The trim-and-fill procedure trimmed 12 studies from the posttest data set, reducing the effect size estimate to nonsignificance ($g = 0.05$, 95% CI [–0.04, 0.14]), $Q = 112.50$. In contrast, the stressor data set did not show evidence of publication bias (see Figure 2), and the trim-and-fill procedure did not identify any studies to be trimmed from this data set. These findings supported the small but robust effect of CBM on symptoms following exposure to a stressor.

Relationship between change in cognitive bias and symptoms. Having found an association between CBM and change in biases as well as between CBM and change in symptoms, we tested whether change in cognitive bias moderated the effect of CBM on symptoms. Within both data sets, metaregression was

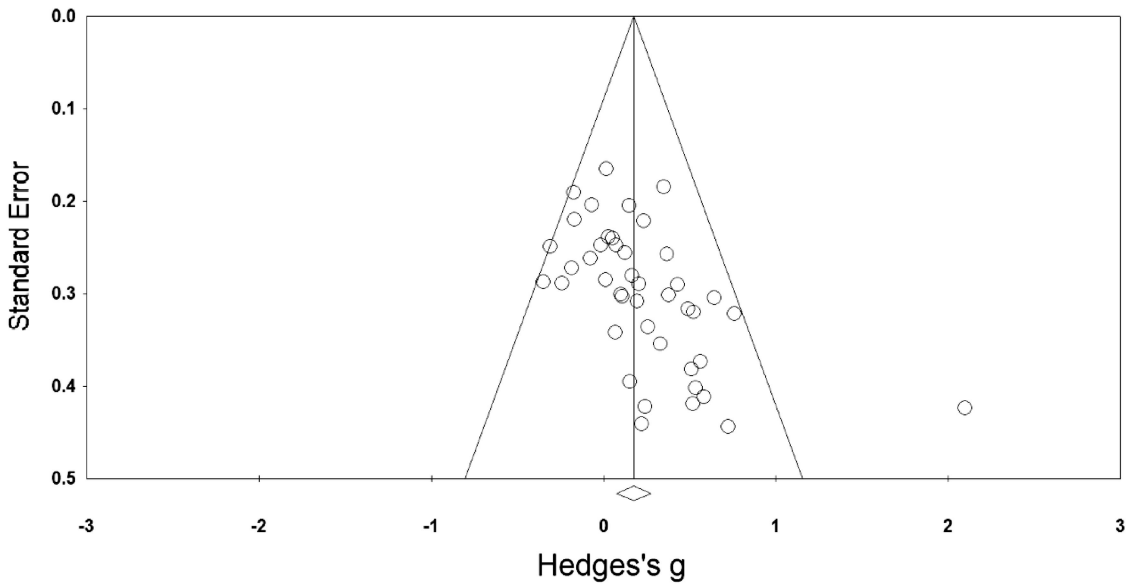

Figure 1. Posttest funnel plot of publication bias: Standard Error × Hedges's *g*.

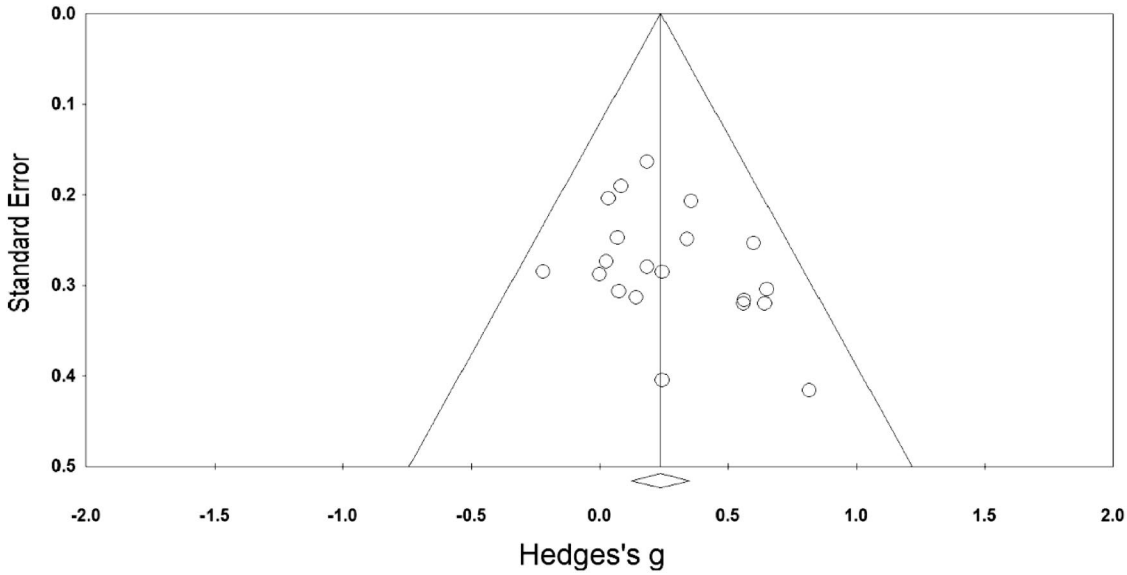

Figure 2. Stressor funnel plot of publication bias: Standard Error × Hedges's *g*.

used to examine the relationship between the effect size for change in cognitive bias and the corresponding effect size for change in symptoms. Change in cognitive bias did not significantly moderate the effect of CBM on symptoms in either data set. Whereas the posttest data set showed a marginally significant trend toward moderation that may have been significant with greater statistical power (slope = 0.16, $p = .052$), $Q(1) = 3.10$, $p = .08$, there was no evidence in the stressor data set that changes in cognitive biases were associated with changes in symptoms (slope = –0.18, $p = .24$), $Q(1) = 1.37$, $p = .24$. Neither slope was significant after sensitivity analyses (slope = 0.20, $p = .15$, and slope = –0.31, $p = .48$, respectively).

Symptom construct. The effect of CBM on anxiety was small but significant in both data sets: posttest ($g = 0.13$, 95% CI = [0.05, 0.22]), $Q(40) = 45.96$, $p = .24$, and stressor ($g = 0.28$, [0.16, 0.41]), $Q(17) = 16.01$, $p = .52$ (see Table 2). After imputing $g = 0.00$ for missing values, the effect sizes became smaller but remained significant (posttest $g = 0.12$, [0.04, 0.20]; stressor $g = 0.22$, [0.11, 0.33]). The effect on depression was somewhat smaller and was not statistically significant in either data set: posttest ($g = 0.06$, [–0.05, 0.18]), $Q(22) = 19.74$, $p = .60$, or stressor ($g = 0.12$, [–0.05, 0.29]), $Q(9) = 6.93$, $p = .65$. The pattern of results was similar after sensitivity analyses imputing $g = 0.00$ (posttest $g = 0.04$, [–0.06, 0.14]; stressor $g = 0.12$, [–0.05, 0.29]). However, the

Table 2
Moderation Analyses for Categorical Variables

	Posttest data set					Stressor data set				
Variable[a]	*k*	*g*	95% CI	*z*	*Q*	*k*	*g*	95% CI	*z*	*Q*
Outcome measure[b]										
Anxiety	41	0.13	[0.05, 0.22]	3.08		18	0.28	[0.16, 0.41]	4.41	
Depression	23	0.06	[−0.05, 0.18]	1.05		10	0.12	[−0.05, 0.29]	1.44	
Bias targeted					$Q(1) = 1.72$, $p = .19$					$Q(1) = 0.12$, $p = .73$
Attention	20	0.08	[−0.04, 0.19]	1.27		11	0.25	[0.11, 0.40]	3.47	
Interpretation	22	0.19	[0.07, 0.30]	3.12		9	0.21	[0.01, 0.41]	2.05	
Participant clinical status					$Q(2) = 2.73$, $p = .26$					$Q(1) = 0.08$, $p = .78$
Healthy/unselected	24	0.09	[−0.01, 0.19]	1.70		13	0.22	[0.08, 0.36]	3.08	
Elevated symptoms/analogue	15	0.17	[0.03, 0.32]	2.31		7	0.28	[0.07, 0.48]	2.64	
Clinical diagnosis	3	0.39	[0.01, 0.77]	2.03						
Control group					$Q(1) = 1.20$, $p = .27$					$Q(1) = 0.23$, $p = .64$
No contingency (50%)	18	0.18	[0.05, 0.32]	2.60		8	0.22	[0.03, 0.41]	1.94	
Attend to negative	19	0.08	[−0.04, 0.20]	1.33		9	0.28	[0.12, 0.45]	3.30	
No. of training sessions					$Q(1) = 2.17$, $p = .14$					$Q(1) = 1.03$, $p = .30$
One	30	0.10	[0.01, 0.19]	2.13		18	0.22	[0.10, 0.34]	3.69	
More than one	12	0.26	[0.07, 0.44]	2.70		2	0.49	[−0.003, 0.98]	1.95	

[a] One outlier (Schmidt et al., 2009) was excluded from the analyses presented here. [b] Anxiety and depression effect sizes were analyzed separately to preserve the independence of effect sizes.

effect sizes for anxiety and depression did not differ significantly from one another, either at posttest or poststressor. Anxiety and depression measures consequently were combined for all remaining analyses.

Cognitive bias targeted. The bias targeted for training (attention or interpretation) was not significantly associated with the magnitude of effect sizes in either data set, posttest $Q(1) = 2.11$, $p = .15$; stressor $Q(1) = .07$, $p = .80$. This result was not altered by sensitivity analyses, both $Q(1) \leq .82$, both $p \geq .37$.

Clinical status of the study sample. Clinical status did not moderate the effect of CBM on symptoms in either data set, posttest $Q(2) = 2.70$, $p = .26$; stressor $Q(1) = 0.08$, $p = .78$. These results were not altered by sensitivity analyses, both $Q(1–2) \leq 3.89$, both $p \geq .14$. Notably, only four studies used clinical samples, one of which was an outlier (Schmidt et al., 2009). Excluding the outlier, these studies yielded a medium effect size ($g = 0.39$) in the posttest data set. When the outlier was included, the effect became large ($g = 0.79$) but the moderation analysis remained nonsignificant. No studies in the stressor data set used clinical samples.

Control group. Studies that trained the control group to engage in a negative cognitive bias were predicted to show larger effect sizes than did studies that provided the control group with sham (i.e., no contingency) training. The effect was nonsignificant but in the predicted direction in the stressor data set, $Q(1) = 0.23$, $p = .64$. However, the opposite pattern was observed in the posttest data set, where the negative-training control group yielded a nonsignificantly smaller effect ($g = 0.09$) than did the sham training control group ($g = 0.18$), $Q(1) = 1.18$, $p = .28$. These results were not altered by sensitivity analyses, both $Q(1) \leq 1.50$, both $p \geq .22$.

Number of sessions. There was no significant effect of number of training sessions on symptoms in the posttest data set, $Q(1) = 1.97$, $p = .16$. However, when sensitivity analyses that included the outlier (Schmidt et al., 2009) were conducted, studies that provided more than one training session had a significantly larger effect on symptoms ($g = 0.40$, 95% CI [0.14, 0.67]) compared with studies that provided only one training session ($g = 0.11$, [0.02, 0.20]), $Q(1) = 4.19$, $p = .04$. In the stressor data set, number of training sessions did not have a significant effect on symptoms ($g = 0.22$) for one training session versus ($g = 0.49$) for multiple training sessions, $Q(1) = 1.15$, $p = .28$. Sensitivity analyses imputing g = 0.00 for all missing values did not significantly alter the results in either data set, both $Q(1) \leq 2.01$, both $p \geq .16$.

Discussion

Effect of CBM on Symptoms and Cognitive Biases

Prior to correcting for potential publication bias, we found that CBM exerted a small, positive effect on anxiety and depression symptoms. In studies that assessed these symptoms immediately following training, this small effect size was reduced to nonsignificance when Duval and Tweedie's (2000) trim-and-fill procedure was applied to account for apparent publication bias. However, in studies that assessed symptoms after participants experienced a stressor (e.g., a threatening video or upcoming exam) following training, the effect was larger, albeit nonsignificantly, and more robust, with minimal evidence of publication bias. These results suggest that cognitive biases may exert their influence on anxiety and depression only through interaction with a stressor. This account is consistent with diathesis-stress cognitive models of anxiety and mood disorders, which propose that latent cognitive biases must be activated by a stressful life event before they will influence symptoms (e.g., Beck, 1996).

Nevertheless, even when symptoms were assessed in the context of a stressor, CBM had only a small effect on symptoms. There are several possible interpretations of this small effect size. First, cognitive biases may play only a small role in the development or maintenance of anxiety and depression. This interpretation is at odds with the proposal that cognitive biases play a central and primary role in the development of these symptoms (e.g., Beck & Clark, 2007) but is consistent with contemporary models that view cognitive biases as one of a multitude of potential causes of these symptoms (e.g., Hudson & Rapee, 2004). A second possibility is that biases do play a central role in anxiety and depression but that CBM produces insufficient change in biases to substantially influence these symptoms. A more dramatic change in biases than the medium effect observed here may be required for CBM to have a clinically significant effect on anxiety and depression. Consistent with this interpretation, we found that the extent of change in cognitive biases moderated the effect of CBM on symptoms at a level that approached statistical significance. A third possibility is that cognitive biases may have a gradual, rather than immediate, effect on symptoms that is not captured by current CBM paradigms. For example, it is possible that attending to threat for a brief period of time (e.g., in a single experimental session) has a minimal impact on anxiety but that attending to threat habitually over many weeks or months may have a cumulative adverse effect on symptoms. The relative dearth of follow-up data on the continued effects of CBM over time makes it difficult to rule out this interpretation. A fourth possibility relates to the fact that CBM paradigms typically target a single cognitive bias, despite research demonstrating that anxiety and depression are characterized by a broad range of cognitive biases that may produce an interactive or additive effect on symptoms (e.g., Hirsch, Clark, & Mathews, 2006). Whereas changes in a single cognitive bias may exert only a small effect on symptoms, targeting multiple types of biases that are relevant to these symptoms may produce a larger effect.

Unfortunately, the most commonly used CBM paradigms are inadequate to distinguish among these explanations. To provide a clearer picture of the role of cognitive biases in anxiety and depression, future research will need to strengthen training paradigms so that they produce a larger effect on biases. If larger changes in biases are found to produce larger, more robust changes in anxiety and depression, it would provide more compelling support for current cognitive theories. A clearer picture may also emerge after accounting for other potential influences on outcome. For example, few of the studies in the present analysis reported data on participant compliance with the training procedures—a potentially significant concern given the solitary and repetitive nature of most CBM paradigms. This leaves open the possibility that data from noncompliant or fatigued participants may have attenuated the effect size of CBM on biases and symptoms. Data on fatigue effects may be important not only for interpreting results but also for evaluating the clinical utility of CBM, because clinical populations may be expected to fatigue more quickly than

do healthy or nonclinical populations. Finally, longer term follow-up assessment would be valuable, because it would allow detection of any gradual changes in symptoms that CBM might produce, particularly in the context of naturally occurring life stressors.

Although the present findings provide some support for one proposed pathway through which CBM may have its effects (i.e., CBM alters biases, and biases interact with stress to influence anxiety and depression symptoms), the full model was not supported in either data set. As described earlier, the effect of CBM on symptoms was small-to-null in the posttest data set, and the extent to which biases changed through training did not significantly moderate the effect of CBM on symptoms (although there was a nonsignificant trend in this direction). CBM had a significant effect on anxiety and depression when those symptoms were assessed in the context of a stressor, but this effect was not moderated by the extent to which biases changed. One reason that the full model was not supported in the stressor data set may have been an inadequately powered test of moderation due to the relatively small sample combined with relatively low variability in symptom effect sizes. Importantly, our results do not preclude the possibility that within individual studies, participants who experienced the greatest bias change also experienced the greatest reduction in symptoms. Because many factors contribute to a study's overall effect size, this pattern could exist within each study without necessarily being reflected in the relationship between the overall effect size of various studies and the extent to which biases changed within those studies. Supporting this possibility, several studies in this meta-analysis found an association between the degree of bias change and the degree of symptom change in the treatment groups (e.g., Najmi & Amir, 2010; Reese, McNally, Najmi, & Amir, 2010), although other studies did not find this association (Johnson, 2009) and many studies did not report this relationship. Although individual studies may not be adequately powered to detect a significant relationship between change in biases and change in symptoms, routine reporting of the relationship within studies may allow future quantitative syntheses to test this important assumption.

The present findings are not entirely consistent with those reported in the recent meta-analysis by Hakamata and colleagues (2010). Whereas that analysis found a large effect of CBM on attention biases, the present study found only a medium effect on cognitive biases and a small effect on attention biases specifically. Additionally, Hakamata and colleagues reported a medium effect of attention training on anxiety symptoms, whereas the present study found a small effect of CBM on symptoms that was reduced to nonsignificance after correcting for publication bias. These discrepancies are not entirely surprising, because literature reviewed by Hakamata and colleagues focused on a more circumscribed research question than the one examined here and therefore considered only 12 studies. Our aim to provide a broad review of the CBM literature led us to synthesize a wider array of studies than those reviewed previously, yielding more variability in participant characteristics, training methodology, outcome measures and constructs, and study focus (experimental vs. intervention). For example, whereas Hakamata and colleagues focused strictly on studies that used the dot-probe paradigm, the present analysis included other attention training paradigms as well. Additionally, the present study included measures of depression as well as anxiety symptoms. Finally, previous reviews, including the Hakamata et al. meta-analysis, generally have not corrected for publication bias (e.g., by using Duval and Tweedie's, 2000, trim-and-fill procedure). Because the present findings suggest that publication bias is a significant concern in the CBM literature, careful consideration of this bias is warranted when synthesizing its results.

An important discovery that emerged from this broad review of studies is that CBM has a smaller effect on attention than interpretation biases. Although this difference may be at least partly explained by methodological differences between attention and interpretation bias modification paradigms (e.g., a greater requirement for generativity in interpretation paradigms), it also suggests a number of interesting theoretical explanations. One potential explanation is that attentional biases may be more resistant to change than are interpretation biases. Another is that CBM may be more effective for biases that are more susceptible to effortful control (i.e., late-stage biases) than for more automatic biases. The latter explanation requires further study, because it raises the possibility that demand characteristics could be playing a role in the modification of these biases. Finally, it is notable that although CBM was more effective for modifying interpretation than attention biases, the effect of CBM on symptoms did not differ as a function of whether interpretation or attention biases were modified. Why a larger change in interpretation biases did not correspond to a larger change in symptoms remains an important question for future research to address.

Potential Moderators of the Effect of CBM on Anxiety and Depression

The present study also investigated whether CBM has a differential effect on anxiety and depression. We found that CBM was associated with a small but reliable reduction in anxiety symptoms. One potential explanation of this small effect is that instead of attenuating negative cognitive biases, CBM procedures may simply train participants to avoid negative stimuli (Cisler & Koster, 2010; Koster, Baert, Bockstaele, & De Raedt, 2010). There is some evidence to suggest that anxious individuals orient toward threat in the early (automatic) stages of attentional processing but attend away from threat in the later (strategic) stages (Garner, Mogg, & Bradley, 2006; Koster, Crombez, Verschuere, Van Damme, & Wiersema, 2006; Mogg, Bradley, Miles, & Dixon, 2004). This tendency, known as the vigilance-avoidance pattern (Mogg & Bradley, 1998), has been proposed to impair habituation to feared stimuli, resulting in the maintenance or enhancement of anxiety (Cisler & Koster, 2010; Koster et al., 2010). With a few exceptions, attention training paradigms have typically presented stimuli for 500 ms or longer, a period that may be long enough to allow strategic avoidance to take place. Critics of CBM have argued that this may allow participants to attend automatically to stimuli in the early stages of attentional processing but to avoid threatening stimuli in the later stages of effortful processing (Koster et al., 2010; Mogg & Bradley, 1998). Because avoidance is thought to reduce anxiety in the short term, directing attention away from threatening material could account for the reduction in anxiety that is observed following CBM. The effects on anxiety would be expected to be temporary, however, because avoidance is predicted to increase or maintain anxiety in the long term (Borkovec,

Alcaine, & Behar, 2004). In contrast, if CBM operates by normalizing attentional processes, its effects on anxiety should be maintained over time. Only a few studies to date have attempted to determine whether CBM specifically alters early stage automatic biases. These studies have trained and tested attention to stimuli presented at short (e.g., 16–30 ms) durations. If CBM effectively trains automatic attention biases, differences between the training and control groups should be evident during both short and long exposure times. Whereas some studies have reported successful training of automatic biases using short exposure times of less than 30 ms (e.g., Mathews & MacLeod, 2002), other studies have found no significant effect of attention bias training at exposure times of 30 ms or less (e.g., Johnson, 2009; Koster et al., 2010; MacLeod et al., 2002). These mixed findings leave open the possibility that attention training does not change automatic attention biases to negative stimuli but instead teaches participants to avoid those stimuli in the later stages of attentional processing. Future research using shorter stimulus presentation paradigms will be essential to rule out this alternative explanation of the small positive effect of CBM on anxiety.

The finding that CBM did not have a reliable effect on depression symptoms also warrants consideration. This finding could be interpreted as suggesting that cognitive biases play a relatively minor role in depression and that CBM may have limited value as an intervention for depression symptoms. Qualifying this interpretation is the fact that many studies in this meta-analysis targeted biases that have been shown to be specific to anxiety (e.g., attention bias to threat) but few targeted biases that are specific to depression (e.g., attention bias for sad faces). Moreover, although CBM did not have a statistically significant effect on depression alone, the effect size for depression did not differ reliably from the significant effect size for anxiety. Because only a small number of studies assessed depression after training ($k = 10$), this finding may simply reflect inadequate statistical power to detect small differences in the effects of CBM on highly correlated anxiety and depression symptom measures. Alternatively, it may suggest one or more intriguing possibilities about how CBM modifies biases. First, it is possible that CBM paradigms operate by influencing nonspecific mechanisms shared by anxiety and depression. Certain negative cognitive biases, such as interpretation biases or strategic processing of generally negative self-relevant information, may have an adverse effect on emotions more generally. It is conceivable that CBM modified cognitive biases that are common to anxiety and depression (e.g., attention to negative stimuli) even when paradigms were designed to target more specific biases (e.g., attention to threat). Second, recent research has suggested that modifying one bias may have an indirect effect on other biases (Amir, Bomyea, & Beard, 2010; Hirsch et al., 2006). For example, Amir et al. (2010) found that modifying interpretation biases also influenced attention biases in socially anxious individuals. A more fine grained analysis of the effects of CBM on biases that are common (vs. specific) to anxiety and depression and the effects of modifying those biases on symptoms would be a valuable step toward a more informed understanding of the connections among cognitive biases and their respective roles in anxiety and depression.

The present meta-analysis also investigated the relationship between the number of training sessions a participant receives and the effectiveness of the intervention. In both data sets, studies with multiple training sessions yielded a nonsignificantly larger effect on symptoms than did studies with a single training session. This effect reached statistical significance when an outlier that provided multiple training sessions (Schmidt et al., 2009) was included in the analysis. The skewed distribution of number of training sessions led us to group studies into two categories for analysis, likely reducing statistical power (Streiner, 2002); it is possible that significant results would have been obtained (prior to sensitivity analyses), had greater variety in the number of training sessions permitted metaregression analyses. Alternatively, it is possible that the nonsignificantly larger effect associated with multiple training sessions was driven by other differences between studies that varied in number of sessions. For example, all of the studies that used a clinical sample provided more than one training session. However, because only a small proportion of the studies that provided multiple training sessions used clinical samples, it is unlikely that the observed effect of multiple training sessions is an epiphenomenon of clinical status. Although the present findings are suggestive, studies that experimentally manipulate the number of training sessions provided to participants would help establish whether there is a dose–response relationship between CBM and symptom change.

Also relevant from a clinical perspective is whether the efficacy of CBM varies as a function of clinical severity. The effect of CBM on symptoms did not differ significantly across studies with clinical (diagnosed) samples, analogue or elevated-symptom samples, and healthy or unselected samples. However, studies with clinical samples showed a medium effect of CBM on symptoms that was nonsignificantly larger than the small effect observed for studies with healthy or unselected samples. This effect became large but remained nonsignificant when an outlier with a clinical sample (Schmidt et al., 2009) was included. Unfortunately, interpretation of this moderation analysis is somewhat muddled by the fact that clinical status was confounded with control group in the studies analyzed. Specifically, the majority of studies with healthy or unselected samples used a "negative training" control group in which negative biases were induced, whereas the majority of studies with diagnosed or analogue/elevated-symptom samples used a sham training control group in which biases were not modified. All else being equal, studies with clinical samples might be expected to yield larger effect sizes than would studies with healthy or unselected samples, because the greater room for change in clinical samples allows for more dramatic posttest differences in biases between the treatment and control groups, along with greater consequent changes in symptoms. However, the confounding of control group with clinical status may have equalized the bias changes across groups, reducing the likelihood of an observable difference in effect size between the samples. Future research might resolve this issue by using multiple control groups (e.g., attend-to-positive and attend-to-negative) in studies with clinical samples.

Although the medium effect of CBM on symptoms in clinical samples is smaller than the large effect sizes typically observed for current empirically supported treatments of anxiety disorders (Stewart & Chambless, 2009) and major depressive disorder (Driessen, Cuijpers, Hollon, & Decker, 2010), it is in line with the effects of individual components of psychotherapy observed in dismantling studies (e.g., Borkovec & Ruscio, 2001; Resick et al., 2008). This finding, along with several unique features of CBM,

suggests that CBM could have promise as a complementary intervention administered in conjunction with traditional psychotherapy. Whereas traditional cognitive therapy targets cognitive biases that occur in the late stages of processing (e.g., by challenging cognitive distortions), CBM aims to intervene at the level of earlier, more basic and automatic cognitive biases (e.g., attention bias to threat). If both types of biases contribute to symptoms, and if CBM can in fact modify early attention biases, a conjoint approach involving modification of both types of biases may produce a larger effect on symptoms than would addressing either alone. Even in situations where CBM and cognitive therapy target similar biases (e.g., interpretation of ambiguous scenarios), some patients who do not respond to cognitive therapy might benefit from the dramatically different approach employed by CBM.

To date, only one study has examined the effects of adding CBM to psychotherapy (McEvoy & Perini, 2009). This study compared cognitive behavioral therapy (CBT) plus CBM (attention training) with CBT plus relaxation training. No significant effect of CBM was observed in this study. However, because the comparison condition included an additional active intervention, it is unclear whether CBT plus CBM would have been superior to CBT alone. This possibility warrants further study, because the administration of most CBM paradigms via computer makes their dissemination as a complementary treatment or early intervention relatively inexpensive. CBM has been administered via the Internet with some success (Salemink, van den Hout, & Kindt, 2009) and could also be administered by trained laypersons in a variety of settings. The possibility of administering CBM as a preventative intervention is particularly interesting in light of the present finding of a robust effect of CBM on symptoms following a stressor. This finding suggests that individuals who have negative cognitive biases but have not yet developed clinically significant anxiety or depression may benefit from receiving CBM, because it may reduce their susceptibility to anxiety or depression symptoms following stressful life events.

Limitations and Future Directions

The present study had several limitations. A common critique of meta-analyses is that they often lump together "apples and oranges" (Hunt, 1997). Combining depression and anxiety measures, or attention and interpretation biases, could be criticized on these grounds. To address this potential concern, the present study examined differences between these varying study features by conducting moderation analyses (Rosenthal & DiMatteo, 2001). A second limitation was the exclusion of the relatively few studies that selected participants on the basis of syndromes other than anxiety or depression, such as schizophrenia, alcohol dependence, or disordered eating. Because the cognitive biases that contribute to these disorders may differ from those that contribute to anxiety and mood disorders, CBM may have different effects on symptoms in these populations (e.g., McCusker, 2001). Finally, the present analysis was limited by several factors related to the current state of the CBM literature, including the paucity of data on participant compliance with the training procedures, the small number of studies that included clinical samples, the strong evidence of publication bias in the posttest data set, and the lack of follow-up data examining the long-term effects of CBM on biases and symptoms.

There are a number of ways in which future CBM research could address these limitations and spur the field forward. As noted earlier, future research would benefit substantially from the development of CBM paradigms that produce a larger effect on cognitive biases. There are several ways in which this might be achieved, each of which also has potentially important clinical or theoretical implications. For example, future research might examine whether modifying multiple cognitive biases (e.g., both attention and interpretation biases) within a single study has an additive effect in terms of symptom improvement. Alternatively, paradigms may be strengthened by the inclusion of additional training sessions or training trials per session, a wider variety of training stimuli, or more ecologically valid contexts to enhance the generalizability of training. Future studies could also systematically evaluate whether CBM is more effective when the paradigms are designed to target biases relevant to a specific disorder. For example, would CBM targeting attention to social threat cues versus negatively valenced cues more broadly be particularly effective for reducing social anxiety disorder? The finding that CBM is more effective when it targets multiple biases or disorder-relevant biases would have important implications for practice (e.g., how best to design CBM interventions) and research (e.g., as a method of experimentally testing the role of specific biases in a given disorder).

The present analysis suggests several other recommendations for future CBM studies that will help advance the field. The robust finding of a small effect of CBM on symptoms in the context of a stressor suggests that researchers should strongly consider including a laboratory or naturalistic stressor in future CBM investigations. Additionally, the promising finding of a medium effect of CBM on symptoms in clinical samples underscores the need for further research with clinical samples to determine whether CBM can be considered an effective intervention for anxiety and depression. Because the present analysis did not find an effect of CBM on depression, early efforts might focus most fruitfully on anxiety disorder samples. Similarly, although early CBM research primarily emphasized attention training, our finding that CBM is more successful for modifying interpretation biases suggests that interpretation training paradigms may provide productive avenues for future research. Researchers should take particular care, however, to demonstrate that demand characteristics are not responsible for observed effects of CBM on biases and symptoms. Finally, the strong evidence of publication bias uncovered here highlights the importance of disseminating even nonsignificant research findings, to increase confidence in any positive findings that may arise from future syntheses of the CBM literature.

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