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Psychological approaches in the treatment of specific phobias: A meta-analysis ☆

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Abstract

Data from 33 randomized treatment studies were subjected to a meta-analysis to address questions surrounding the efficacy of psychological approaches in the treatment of specific phobia. As expected, exposure-based treatment produced large effects sizes relative to no treatment. They also outperformed placebo conditions and alternative active psychotherapeutic approaches. Treatments involving *in vivo* contact with the phobic target also outperformed alternative modes of exposure (e.g., imaginal exposure, virtual reality, etc.) at post-treatment but not at follow-up. Placebo treatments were significantly more effective than no treatment suggesting that specific phobia sufferers are moderately responsive to placebo interventions. Multi-session treatments marginally outperformed single-session treatments on domain-specific questionnaire measures of phobic dysfunction, and moderator analyses revealed that more sessions predicted more favorable outcomes. Contrary to expectation, effect sizes for the major comparisons of interest were not moderated by type of specific phobia. These findings provide the first quantitative summary evidence supporting the superiority of exposure-based treatments over alternative treatment approaches for those presenting with specific phobia. Recommendations for future research are also discussed.

Keywords: Specific phobia; Meta-analysis; Exposure treatment

Contents

☆ Portions of these data were previously presented at the 2005 annual meeting of the Association for Behavioral and Cognitive Therapies.

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1. Introduction

Specific phobia is characterized by a marked and persistent fear of a specific object or situation that causes significant life interference or distress (APA, 1994). With a lifetime prevalence of 12.5% (Kessler, Berglund, & Demler, 2005) specific phobia ranks as the most common anxiety disorder. Specific phobias are currently divided into four subtypes: situational (e.g., fears of enclosed spaces, flying), natural environment (e.g., fears of heights, storms, water), animal (e.g., fears of snakes, spiders, dogs), and blood/injection/injury (e.g. fears of dental or medical procedures, injections, seeing blood), with the animal and natural environment subtypes being more prevalent (Curtis, Magee, Eaton, Wittchen, & Kessler, 1998).

As with most anxiety disorders, specific phobias show a chronic course with low rates of spontaneous remission (Wittchen, 1988). Despite their circumscribed nature, specific phobia is associated with significant impairment. Wittchen, Nelosn, and Lachner (1998) found that young adults with a diagnosis of specific phobia reported severe impairment in their routine activities during the worst episode of their disorder. Specific phobias also represent a significant challenge to the medical field. For example, almost one-third of patients undergoing recumbent MRI (fMRI) are not able to complete the procedure due to severe claustrophobic reactions (Quirk,

Letendre, Ciottone, & Lingley, 1989; Thorp, Owens, Whitehouse, & Dewey, 1990). Similarly, blood-injury and injection phobias often result in avoidance of medical procedures (Kleinknecht, 1994), and avoidance related to dental phobia can lead significant dental health problems and reductions in quality of life (cf. Mehrstedt, Tonnies, & Eisentraut, 2002).

1.1. Treatment utilization

There is now compelling evidence suggesting that those suffering from specific phobias are hesitant to seek treatment despite the availability of effective interventions. Based on data from the ECA study, only 31% of those meeting DSM-III criteria for phobia sought treatment (Regier, Narrow, & Rae, 1993), and of those, only 43.4% sought specialty mental health services (Narrow, Darrel, Rae, & Manderscheid, 1993).

Several factors may contribute to this reluctance to seek treatment. First, many perceive their phobia as untreatable, or are unaware of effective and available treatments. Second, as many of the available treatments involve direct confrontation with the phobic target, those who are aware of the available treatments may be apprehensive to engage in them. More specifically, about 25% of phobic patients refuse exposure-based treatment due to fear of facing the feared object or situation (Marks, 1992; Marks & O'Sullivan, 1988). Likewise, Öst (1989) reported that 90% of the spider phobic participants in his study would have refused his single-session treatment if they were told in advance what the treatment entailed. Third, because of the situationally-bound nature of the fear inherent in specific phobia, avoidance of the phobic target may be easily achieved and, thus, serves as a disincentive for seeking treatment. Finally, some individuals may have experienced a failure in conducting self-administered exposure and have therefore concluded that they are unresponsive to this mode of treatment.

Despite the low proportion of phobia sufferers who seek treatment (Regier et al., 1993), specific phobia is among the most treatable of disorders. Those who seek treatment can choose from a number of different interventions, including (but not limited to) cognitive therapy, modeling, imaginal or virtual reality exposure, and direct *in vivo* exposure. Of all available therapies, exposure therapy is the most widely studied and often considered the first line of treatment for specific phobias (see Barlow, Raffa, & Cohen, 2002; Craske, 1999; Telch, 2004). A recent review article concludes that *in vivo* exposure may be the most efficacious treatment for specific phobias (Choy, Fyer, & Lipsitz, 2007). However, the authors also note that other modalities of exposure and cognitive restructuring may also be useful in treating certain subtypes of specific phobia.

1.2. Current treatments for specific phobia

1.2.1. Exposure approaches

1.2.1.1. In vivo exposure. *In vivo* exposure involves having patients come into direct contact with the feared stimulus, such as a live spider. *In vivo* exposure has been extensively researched for the treatment of specific phobias including, but not limited to, spiders (Hellstrom & Öst, 1995; Muris, Mayer, & Merckelbach, 1998; Öst, 1996; Öst, Ferebee, & Furmark, 1997; Öst, Salkovskis, & Hellstrom, 1991), snakes (Gauthier & Marshall, 1977; Hepner & Cauthen, 1975), rats (Foa, Blau, Prout, & Latimer, 1977), dogs (Rentz, Powers, Smits, Cougle, & Telch, 2003.), thunder and lightening (Öst, 1978a), water (Menzies & Clarke, 1993), heights (Baker, Cohen, and Saunders, 1973; Bourque & Ladouceur, 1980), flying (Beckham, Vrana, May, Gustafson, & Smith, 1990; Howard, Murphy, & Clarke, 1983; Öst, Brandberg, and Alm, 1997), enclosed spaces (Craske, Mohlman, Yi, Glover, & Valeri, 1995; Öst, Johansson, and Jerremalm, 1982; Powers, Smits, and Telch, 2004), choking (Greenberg, Stern, & Weilburg, 1988), dental fears (Gitin, Herbert, and Schmidt, 1996; Moore & Brodsgaard, 1994), blood (Öst, Fellenius, & Sterner, 1991), and balloons (Houlihan, Schwartz, Miltenberger, & Heuton, 1993).

1.2.1.2. Systematic desensitization. Developed by Wolpe (1958, 1973), and based on his theory of "reciprocal inhibition", systematic desensitization consists of teaching the patient to relax the voluntary muscles during imaginal confrontation with the feared stimulus. The treatment consists of three distinct components: (a) training in progressive muscle relaxation (PMR); (b) constructing a fear hierarchy; and (c) desensitization proper — consisting of repeated imaginal presentations of the feared stimulus in a graduated fashion while having the patient engage in progressive muscle relaxation (PMR).

1.2.1.3. Imaginal exposure. Imaginal exposure consists of having the patient imagine a confrontation with the feared stimulus. Unlike systematic desensitization, which dedicated a number of sessions to relaxation training, modern imaginal exposure approaches tend to omit the relaxation component.

1.2.1.4. Virtual reality and computer assisted exposure. These approaches allow the patient to confront computer-generated representations of the phobic target. The two variants of this general approach include virtual reality (VR; Rothbaum et al., 1995; Rothbaum, Hodges, Smith, Lee, & Price, 2000) and computer-aided vicarious exposure (CAVE; Dewis et al., 2001). In VR, the patient interacts with a virtual representation of the phobic stimulus, while wearing headphones and a head-tracking device. The virtual environment is thought to create a sufficient "sense of presence" (Rothbaum et al., 2000, p. 1021) to allow the patient to emotionally process the exposure to the stimulus. CAVE uses less advanced technology than VR; the client interfaces with a computer, guiding a virtual person through interactions with the phobic object.

1.2.1.5. Eye movement desensitization and reprocessing (EMDR). Originally developed by Shapiro (1989) for the treatment of post-traumatic stress disorder (PTSD), EMDR involves presenting imagery instructions related to the traumatic memory while the patient engages in rapid eye movements. The patient is instructed to focus on a disturbing image, memory, emotion, or cognition, while the therapist moves a finger across the patient's visual field and the patient tracks the finger's movement. One aim of the treatment is to change the cognitions regarding the trauma from negative to more positive (see Shapiro, 1989, 1995 for protocol details). Adapted for specific phobias, the imaginal exposure component consists of imagining confrontation with the phobic target, rather than a traumatic event, as in PTSD.

Although proponents of EMDR consider this treatment to be a unique modality in its own right, the therapy undoubtedly contains exposure as a central element. In fact, some have argued that the effects of this technique are attributable entirely to the imaginal exposure component (Pitman et al., 1996; Renfrey & Spates, 1994).

1.2.1.6. Applied tension (AT). AT has been primarily used in the treatment of blood or injection phobias. Patients are exposed to blood/injury stimuli in a graduated fashion while being instructed to tense their muscles in order to raise their blood pressure, thereby preventing fainting in the presence of blood or injections (Öst et al., 1991; Öst, Sterner, & Fellenius, 1989).

1.2.1.7. Applied relaxation (AR). Like AT, applied relaxation (AR) consists of teaching patients a specific skill (in this case, progressive muscle relaxation, PMR) and then teaching them to use that skill during confrontation with the feared stimulus. Patients are provided with ample training in PMR and exposure is often introduced much later into treatment (e.g., Öst, Lindahl, Sterner, & Jerremalm, 1984).

1.2.2. Alternative approaches to exposure therapies

1.2.2.1. Cognitive therapy (CT). CT has also been studied as a treatment for specific phobia (Craske & Rowe, 1997) either alone (Booth & Rachman, 1992; de Jongh et al., 1995; O'Donahue & Szymanski, 1993) or in combination with exposure-based treatments (Kamphuis & Telch, 2000; Koch, Spates, & Himle, 2004). In CT, patients are taught to identify and alter their faulty threat appraisals maintaining the phobic reaction. Examples of CT techniques include cognitive restructuring (de Jongh et al., 1995) and guided threat reappraisal (Kamphuis & Telch, 2000; Sloan & Telch, 2002).

1.2.2.2. Progressive muscle relaxation (PMR). PMR consists of teaching patients to tense and release specific muscle groups in an effort to train patients to relax themselves. Although some researchers use the terms AR and PMR interchangeably, AR by definition includes an exposure component, while PMR does not. Although PMR has been studied more extensively in the treatment of other anxiety disorders, several studies have examined the efficacy of PMR in the treatment of specific phobia (e.g., Öst et al., 1982; Gilroy, Kirkby, Daniels, Menzies, & Montgomery, 2000).

1.3. Aims of this meta-analysis

Our overarching objective was to provide a quantitative meta-analysis of the efficacy research on psychosocial treatments for specific phobia. Based on the available published studies, the following efficacy-related questions lent themselves to

meta-analytic investigation: (a) Are psychosocial treatments efficacious relative to no treatment or placebo treatments? (b) Are treatments that include exposure to the phobic target more effective than treatments that do not? (c) Are placebo "treatments" more effective than no treatment? (d) Are *in vivo* exposure treatments more effective than treatments that use an alternative modality of exposure (e.g., imaginal, computer-aided)? (e) Are exposure treatments that include adjunctive cognitive techniques more effective than exposure treatments alone? (f) Are multiple treatment sessions more effective than one treatment session? and (g) Are there factors that reliably predict (moderate) treatment outcome? Our decision to synthesize the data in a qualitative meta-analysis to address these questions was based on several factors. First, single studies do not provide definitive evidence on which to influence policy or practice (Hedges & Olkin, 1985). Second, findings addressing these questions are often contradictory. For example, some studies show cognitive augmentation strategies clearly enhance exposure treatment (e.g. Kamphuis & Telch, 2000; Sloan & Telch, 2002) while others do not (Szymanski & O'Donahue, 1995). Finally, narrative/qualitative reviews may inflate type II errors (Cooper & Rosenthal, 1980).

1.3.1. Psychosocial treatments: are they effective?

A first step toward assessing the utility of psychosocial treatments for specific phobias is to tease apart the relative contributions of treatment and non-specific factors. There is considerable variance in the effect sizes of psychosocial treatments relative to placebo and no-treatment controls.

1.3.2. Exposure treatments: are they effective?

Because exposure treatments represent the most widely studied treatment of specific phobia, a sufficient number of studies were available to separately examine their efficacy relative to (a) no treatment; (b) a placebo control; and (c) psychotherapies that do not include an exposure component. Moreover, several studies manipulated parameters of exposure treatment to evaluate ways to enhance its efficacy. We examined three such parametric comparisons: (a) one vs. five sessions of *in vivo* exposure; (b) *in vivo* exposure treatments vs. alternative exposure modalities (e.g., imaginal exposure, VR, CAVE); and (c) exposure plus cognitive techniques vs. exposure alone. The decision to test these comparisons was based entirely on the availability of studies. For example, there were too few studies to compare self-directed vs. therapist-directed exposure.

1.3.3. Alternatives to exposure therapy: are they effective?

Several studies of *truly* non-exposure psychosocial treatments were located, which allowed us to estimate their efficacy relative to no treatment. However, there were too few studies to compare non-exposure treatments to placebo.

1.3.4. Are placebo treatments effective in the treatment of specific phobias?

Several studies included both a credible placebo control groups (PL) and a wait-list control group (WL) thus allowing for an estimate of the placebo response in the treatment of specific phobia.

1.3.5. Effect size moderators

The available studies were markedly heterogeneous on a number of dimensions, such as specific phobia subtype, treatment dose, and level of therapist involvement. For those comparisons showing statistically significant heterogeneity, we examined whether these factors significantly influenced estimates of treatment efficacy.

1.3.5.1. Number of treatment sessions. Unlike many other disorders, specific phobias can display significant symptom reduction in doses as low as a single session (e.g., Hellstrom, Fellenius & Öst, 1996). An understanding of the dose–response relationship can be useful for both treatment planning and for a theoretical understanding of fear reduction.

1.3.5.2. Type of phobia treated. Treatments for specific phobia have been studied for each of the four subtypes. None of the studies meeting inclusion criteria investigated treatment efficacy across more than one specific phobia subtype. Choy et al. (2007) concluded that certain treatments may be more effective for some subtypes of specific phobia than others. However, these conclusions were qualitative and were based on a very small number of studies. Consequently, we included phobia type as a putative moderator of treatment outcome.

1.3.5.3. Therapist involvement. There were too few studies directly comparing self-guided vs. therapist-guided exposure. However, because of the public health significance associated with self-administered treatment delivery, we examined this exposure parameter as a putative moderator of treatment efficacy.

1.3.5.4. Date of publication. Based on past research suggesting that date of publication may influence effect sizes of randomized treatment studies (Abramowitz, 1997), we examined whether date of publication moderated the effect sizes for the comparisons of interest.

2. Methods

2.1. Selection of studies

We began by searching all published reports of randomized treatment studies of psychosocial interventions for specific phobia. Several methods were used to locate published studies. First, we conducted searches of PsycINFO 1840 to August, 2007, PsycARTICLES 1894 to August, 2007, PubMed 1966 to August, 2007, MedLine 1966 to August, 2007, and Cumulative Index to Nursing and Allied Health Literature 1982 to August, 2007, using the following keywords: *specific phobia*, *simple phobia*, *specific phobia treatment*, *simple phobia treatment*, *phobia treatment*, *acrophobia*, *claustrophobia*, *snake phobia*, *spider phobia* and *blood phobia*. These searches were limited to peer-reviewed, English language journals, with only adult participants. Searches never "exploded" these keywords into more general terminology. This search initially yielded 988 articles. We then examined the abstracts of these 988 articles, and identified 46 articles that provided descriptions consistent with the study inclusion criteria (see below). Next, we examined the reference sections of the 46 articles and selected an additional 14 articles that appeared to qualify for inclusion. These 60 articles were reviewed independently by two of the authors (KW and JH) to ensure that they met all of the above inclusion criteria. Twenty-three of these 60 studies were eliminated because they failed to meet one or more of the following inclusion criteria: (a) studied an adult population either meeting full DSM criteria for specific phobia, or meeting all criteria except for the distress/interference criterion; (b) randomized participants to two or more treatment conditions in which at least one active treatment was compared to one or more of the following conditions: wait-list control, placebo control, or another active treatment; and (c) included outcome indices that assessed target phobic symptoms as opposed to general psychological adjustment or dysfunction. Of the 37 remaining studies, eight studies did not report statistics that would allow for the calculation of effect sizes. After attempting to contact the authors, we were successful in obtaining the necessary data for four of the eight studies.[2] The remaining four studies were excluded, thus leaving a total of 33 published studies.

2.2. Classification of treatments

2.2.1. Exposure treatments

Treatments were classified as exposure treatments if they included direct or indirect confrontation with the feared stimulus. Such treatments included *in vivo* exposure, *in vivo* exposure with safety behavior availability, *in vivo* exposure with safety behavior utilization, imaginal exposure, active imaginal exposure, systematic desensitization, EMDR, VR, VR with a tactile component, CAVE, negative practice, therapist-assisted flooding, flooding by tape recording, and guided mastery.

Although some authors labeled their treatments as something other than exposure, we considered these treatments exposure if they included any exposure component. For example, an AR treatment that included sessions in which participants used AR skills in the presence of the phobic object was classified as exposure, as was a treatment called "cognitive restructuring" that incorporated cognitive restructuring during confrontation with the phobic object. Further, EMDR was classified as exposure because it includes an imaginal exposure component.

2.2.2. Non-exposure treatments

These were defined as any treatment presumed to be active (i.e., not a placebo) that did not include confrontation with the phobic target as a procedural element. These included relaxation, PMR (which some authors, perhaps inaccurately, refer to as AR with no exposure component), tension techniques alone, and cognitive therapy.

2.2.3. Placebo treatments (PL)

Treatments were classified as *Placebo* if all of the conditions were met: (a) the treatment was labeled as "placebo" in the original article; (b) participants were provided a credible rationale as to how the intervention would assist them in

[2] Data were provided by Beckham, Bourque, De Jongh, Muhlberger, and Gotestam, although data provided by Gotestam was unfortunately insufficient due to lack of data for the control group.

helping them overcome their phobia; and (c) included no procedural component known to be actively efficacious in the treatment of specific phobia. Placebo conditions included pleasant imagery, watching a nature film and being told (deceptively) that the film contained subliminal messages which would reduce fear of the target object, free association about childhood events,[3] information sessions about phobias, and pulsed audio–photic stimulation (APS).[4]

2.3. Classification of outcome measures

Outcome measures were classified into one of the following three domains: Behavioral, physiological, and questionnaire. Behavioral outcome measures were classified as those derived from a behavioral approach test (BAT). These included self-reported fear ratings while participants approached the phobic target and level of behavioral approach (e.g., number of steps completed during the BAT). Measures assessing psychophysiological responding were classified as physiological measures. They included: heart rate, heart rate reactivity, skin conductance, systolic and diastolic blood pressure. Questionnaire measures varied greatly and depended on the particular phobic target under study. However, questionnaires were only included if they directly measured fear or avoidance of the phobic target (e.g., Acrophobia Questionnaire). Questionnaires tapping general functioning (e.g., mood or personality) were excluded.

2.4. Statistical analyses

Effect sizes were calculated using Comprehensive Meta-analysis Software (CMA) Version 2 (Biostat; Borenstein, Hedges, Higgins, & Rothstein, 2005). Effect sizes for each measure were calculated for the 11 treatment comparisons of interest. Effect sizes for each comparison were categorized as falling into one of three outcome domains described above (i.e., behavioral, physiological, or questionnaire).[5] Multiple measures in a given domain were averaged to form one composite effect size for that domain. For each study, we computed effect sizes for one or more comparisons of interest. When studies included multiple groups that were suitable for comparison, multiple effect sizes were obtained. For example, a study comparing three types of exposure therapy with a wait-list condition would generate three separate "exposure vs. wait-list" effect sizes.

For each comparison of interest, we obtained a separate effect size for each available outcome assessment domain: behavioral and/or questionnaire and/or physiological (though very few included physiological outcomes). For studies including more than one measure in a given domain, separate effect sizes were calculated for each measure and were then averaged to form a pooled behavioral or questionnaire effect size. Analyses for each comparison used only these pooled behavioral, questionnaire, and physiological outcomes. For example, a study assessing outcome using three questionnaire measures and two behavioral measures would yield two effect sizes — a composite questionnaire and a composite behavioral effect size.[6]

Analyses were run using fixed-effects models. For each comparison of interest, separate analyses were conducted for each assessment domain,[7] and an overall composite effect size was calculated by pooling the behavioral, questionnaire, and physiological effect sizes. Cohen's *d* was selected as the index of effect size, with *d*-values of 0.2, 0.5, and 0.8 representing small, medium, and large effects, respectively. Effect sizes for each outcome were weighted by sample size in the analysis of each comparison. Weighting of sample size was done in order to minimize the risk that a small, outlying sample would exert a disproportionate influence over the final effect size for a comparison (Rosenthal, 1991). For each comparison, we calculated the statistical significance (*p*-value) of the effect size, the within-

[3] Participants were provided with the rationale that these events (e.g., first day of school) were related to the target fear.

[4] Participants who underwent the APS placebo condition were provided with the rationale that the device will relax them. The APS device has been used as a credible placebo in studies of specific phobia (Powers et al., 2004; Wolitzky & Telch, in press) and social phobia (Smits, Powers, & Telch, 2005).

[5] These categories were selected in accordance with the tripartite model of anxiety (Turner & Michelson, 1984), which emphasizes the importance of evaluating self-report, behavioral, and physiological data in order to comprehensively assess anxiety.

[6] Because some studies included multiple modalities of outcome measures while others included only one, the outcome analyses for different modalities were calculated from slightly different collections of studies.

[7] For example, all "exposure vs. placebo pooled questionnaire measures" comparisons from various studies were selected.

comparison heterogeneity index (Q), and the p-value for the heterogeneity index. We also calculated the: (a) standard error (SEd), (b) variance, and (c) 95% confidence intervals (CI) for the effect sizes. These statistics, reported in Table 2, provide information on the stability, significance, and range of the true effect size.

Moderator analyses were performed by using the random-effects SPSS macro-developed by Lipsey and Wilson (2001). Studies were appropriately dummy-coded for each putative moderator.

3. Results

3.1. Characteristics of the final sample of studies

This final sample of 33 studies were published from 1977–2004 and included 90 treatments administered to 1193 participants. The mean sample size across studies was 36.15. The average length of treatment was 3.04 sessions, with 46% of the studies examining only one session of treatment. The average length of follow-up was 168 days, with follow-up assessment periods ranging from 2 weeks to 14 months. Table 1 shows the studies included in the meta-analysis for each of the 11 a priori comparisons. Table 2 shows the summary statistics for each comparison.

Table 1
Comparisons by study

Study	All vs. WL	Exp vs. WL	Non-Exp vs. WL	Exp vs. Plac	WL vs. Plac	Exp vs. Non-Exp	IVE vs. Non-IVE	1 vs. 5	Exp vs. Exp+CT
Bates, McGlynn, Montgomery, and Mattke (1996)	x	x							
Beckham et al. (1990)	x	x							
Booth and Rachman (1992)						x			
de Jongh et al. (1995)	x		x						
Emmelkamp et al. (2002)							x		
Foa et al. (1977)	x	x		x	x				
Garcia-Palacios, Hoffman, Carlin, Furness and Botella (2002)	x	x							
Gauthier and Marshall (1977)	x	x		x	x				
Gilroy et al. (2000)						x	x		
Heading et al. (2001)	x	x					x		
Hecker (1990)							x		
Hellstrom, Fellenius, and Öst (1996)						x		x	
Hoffman, Garcia-Palacios, Carlin, Furness, and Botella-Arbona (2003)	x	x							
Kamphuis and Telch (2000)									x
Kirsch, Tennen, Saccone, and Cody (1983)	x	x		x	x				
Koch et al. (2004)									x
Muhlberger, Herrmann, Wiedemann, Ellgring, and Pauli (2001)						x			
Muris and Merkelbach (1997)	x	x							
O'Donahue and Szymanski (1993)	x		x						
Öst (1978a,b)	x	x							
Öst et al. (1982) physiological responders	x	x	x			x			
Öst et al. (1982) behavioral responders	x	x	x			x			
Öst et al. (1991)						x			
Öst, Hellstrom, & Kaver (1992)								x	
Öst et al. (1997)								x	
Öst, Alm, Brandberg, and Breitholtz (2001)						x		x	
Pendleton and Higgins (1983)	x	x	x			x			
Powers et al. (2004)	x	x		x	x				
Rentz et al. (2003)							x		
Rothbaum et al. (1995)	x	x							
Rothbaum et al. (2000)	x	x					x		
Sloan and Telch (2002)									x
Szymanski and O'Donahue (1995)	x	x	x	x	x	x			
Williams, Turner, and Peer (1985)	x	x					x		

Table 2
Statistical information by comparison

Comparison	*K*	# out	*N*	*d*	SEd	Var.	95% C.I.	*Q* (*df*)
All Tx vs. WL C	20	58	1217	1.03***	0.06	0.004	0.91–1.16	(57) 142.95***
All Tx vs. WL B	14	27	589	1.09***	0.09	0.008	0.91–1.27	(27) 39.42†
All Tx vs. WL Q	14	30	614	1.02***	0.09	0.008	0.85–1.19	(29) 97.65***
Exp vs. WL C	18	45	961	1.05***	0.07	0.005	0.91–1.20	(44) 121.08***
Exp vs. WL B	13	22	499	1.16***	0.10	0.01	0.96–1.36	(21) 34.52*
Exp vs. WL Q	12	22	448	1.00***	0.11	0.01	0.79–1.20	(21) 79.26***
Non-Exp vs. WL C	6	12	298	0.98***	0.13	0.02	0.73–1.23	(11) 21.51*
Non-Exp vs. WL B	3	4	104	0.81***	0.22	0.05	0.37–1.24	(3) 2.33
Non-Exp vs. WL Q	5	8	194	1.07***	0.16	0.03	0.76–1.38	(7) 18.24**
Exp vs. Plac C Post	5	14	316	0.48***	0.12	0.01	0.25–0.72	(13) 12.75***
Exp vs. Plac B Post	5	9	216	0.42**	0.15	0.02	0.13–0.70	(8) 7.46
Exp vs. Plac Q Post	2	5	100	0.61**	0.21	0.04	0.20–1.02	(4) 4.69
Exp vs. Plac C FU	2	7	210	0.80***	0.15	0.02	0.50–1.09	(6) 7.04
Exp vs. Plac B FU	2	4	130	0.68***	0.20	0.04	0.30–1.07	(3) 4.90
WL vs. Plac C	5	7	146	0.57***	0.18	0.03	0.23–0.92	(6) 15.82
WL vs. Plac B	5	5	107	0.82***	0.21	0.04	0.41–1.22	(4) 9.79
WL vs. Plac Q	2	2	39	−0.03	0.33	0.12	−0.66–0.61	(1) 1.27
Exp vs. Non-Exp C Post	10	27	80	0.44***	0.08	0.007	0.28–0.60	(24)37.36†
Exp vs. Non-Exp B Post	5	10	236	0.38**	0.13	0.02	0.12–0.63	(9) 11.89
Exp vs. Non-Exp Q Post	9	15	330	0.51***	0.11	0.01	0.28–0.73	(14) 24.09*
Exp vs. Non-Exp C FU	7	17	461	0.35***	0.10	0.01	0.16–0.54	(16) 31.21*
Exp vs. Non-Exp B FU	3	6	168	0.33*	0.16	0.03	0.02–0.64	(5) 15.48**
Exp vs. Non-Exp Q FU	5	9	261	0.36**	0.14	0.02	0.08–0.63	(8) 12.40
IVE vs. Non-IVE C Post	7	11	357	0.38***	0.11	0.01	0.17–0.58	(10) 16.54
IVE vs. Non-IVE B Post	5	6	220	0.37**	0.14	0.02	0.11–0.64	(5) 7.04
IVE vs. Non-IVE Q Post	5	5	155	0.38*	0.17	0.03	0.06–0.70	(4) 9.50
IVE vs. Non-IVE C FU	6	9	306	0.20†	0.12	0.01	−0.03–0.43	(8) 6.96
IVE vs. Non-IVE B FU	4	5	187	0.20	0.15	0.02	−0.09–0.49	(4) 3.95
IVE vs. Non-IVE Q FU	4	4	119	0.20	0.19	0.03	−0.16–0.57	(3) 3.01
1 vs. 5 C Post	4	7	194	0.11	0.14	0.02	−0.17–0.39	(6) 1.98
1 vs. 5 Q Post	4	4	110	0.23	0.19	0.04	−0.14–0.61	(3) 0.76
1 vs. 5 P Post	2	2	62	0.003	0.25	0.07	−0.50–0.50	(1) 0.08
1 vs. 5 C FU	4	7	194	0.18	0.15	0.02	−0.10–0.47	(6) 6.89
1 vs. 5 Q FU	4	4	110	0.35†	0.19	0.04	−0.03–0.74	(3) 4.41
1 vs. 5 P FU	2	2	62	0.19	0.30	0.09	−0.40–0.78	(1) 0.09
Exp vs. Exp+CT C Post	5	9	200	0.17	0.13	0.02	−0.09–0.44	(8) 5.63
Exp vs. Exp+CT B Post	5	5	125	0.25	0.18	0.03	−0.11–0.60	(4) 0.70
Exp vs. Exp+CT Q Post	3	3	75	0.22	0.23	0.06	−0.24–0.68	(2) 3.10
Exp vs. Exp+CT C FU	3	5	125	0.14	0.18	0.03	−0.21–0.49	(4) 3.73
Exp vs. Exp+CT B FU	3	3	75	0.15	0.23	0.06	−0.31–0.61	(2) 2.91

Key: statistics: *K* = number of studies; # out = number of outcomes in comparison; *d* = Cohen's *d* effect size; SEd = standard error of the effect size; Var. = variance; 95% C.I. = 95% confidence intervals; *Q* = heterogeneity index; *$p<0.05$; **$p<0.01$; ***$p<0.001$; †$p<0.10$.
Abbreviations: Q = questionnaires; B = behavioral measures; P = physiological measures; C = composite (all measures); Exp = exposure; Tx = treatments; WL = wait-list controls; Plac = placebo; Post = post-treatment assessment; FU = follow-up; Exp+CT = exposure augmented with a cognitive technique; Non-Exp = non-exposure treatment.

3.2. Efficacy of active treatments relative to no treatment

Twenty studies compared one or more active treatments to a wait-list or no-treatment control condition. Not surprisingly, this comparison revealed that phobic participants receiving active treatment showed marked benefit relative to untreated participants. The magnitude of this effect was large, $d=1.03$, $p<0.001$, with significant heterogeneity in the effect sizes across studies $Q(57)=142.95$. The magnitude of this effect was similar for both questionnaires $d=1.02$ ($p<0.001$) and behavioral measures, $d=1.09$ ($p<0.001$). This effect size can be interpreted as showing that the average participant undergoing treatment in these studies is better off than approximately 84% of those randomized to no treatment. As is typical with studies assessing wait-list control groups, too few studies included

follow-up measures to permit the systematic evaluation of longer-term outcome. It should also be noted that in 18 of the 20 studies included in this comparison, the active treatment comprised an exposure-based treatment.

3.3. Efficacy of exposure treatments

3.3.1. Exposure treatments vs. wait-list

Eighteen studies compared one or more exposure treatments to a wait-list control condition. This comparison yielded a large overall effect size, $d=1.05$, $p<0.001$, and significant heterogeneity across studies, $Q(44)=121.08$. Effect sizes for this comparison were similar for behavioral ($d=1.16$) and questionnaire ($d=1.00$) measures (all p's<0.001).

3.3.2. Exposure treatments vs. placebo

Five studies compared an exposure treatment to a placebo treatment. This comparison yielded a moderate overall effect size at post-treatment and a large effect size at follow-up, $d=0.48$, $p<0.001$. Significant heterogeneity was observed at both post-treatment, $Q(13)=12.75$, and follow-up, $Q(6)=7.04$. Post-treatment effect sizes were somewhat higher for questionnaire measures ($d=0.61$) than for behavioral measures ($d=0.42$). The superiority of exposure treatment over placebo treatment was stronger at follow-up ($d=0.68$). These findings indicate that the average participant receiving exposure treatment is doing better than 75% of participants receiving placebo treatment at the follow-up assessment. However, these findings should be interpreted with caution, due to the small number of studies contributing to this comparison.

3.4. Efficacy of non-exposure treatments

Six studies compared a non-exposure treatment to a wait-list condition. This comparison yielded a large overall effect size, $d=0.98$, $p<0.001$, with significant heterogeneity across studies [$Q(11)=21.51$, $p<0.01$]. Effects were larger for questionnaire measures ($d=1.07$, $p<0.001$) than for behavioral measures ($d=0.81$, $p<0.001$). These findings indicate that treatments that do not involve an exposure component can still benefit those with specific phobia.

3.4.1. Efficacy of exposure treatments compared to non-exposure treatments

Ten studies directly compared one or more exposure treatments to a non-exposure treatment. As predicted, exposure treatment led to significantly greater improvement at both post-treatment, ($d=0.44$, $p<0.001$) and follow-up ($d=0.35$, $p<0.001$), with significant heterogeneity across studies, $Q(24)=37.36$ (post-treatment) and $Q(16)=31.21$ (follow-up). These findings were consistent across measurement domains, with moderate post-treatment effects sizes observed for behavioral, $d=0.38$, $p<0.001$, and questionnaire measures, $d=0.51$, $p<0.001$. A similar pattern was observed at follow-up, with effect sizes in the moderate range, albeit somewhat weaker (behavioral measures, $d=0.33$, $p<0.05$; questionnaire measures, $d=0.36$, $p<0.01$. Overall, these findings indicate that exposure-based treatments significantly outperform non-exposure treatments at both post-treatment and follow-up assessments, with the average exposure-treated participant outperforming approximately 64% of those receiving an active non-exposure treatment.

3.5. Efficacy of placebo treatments

Five studies included both a PL and a WL control group thus allowing for an assessment of specific phobic individuals' response to placebo treatment. This comparison yielded a moderate overall effect size in favor of placebo treatment, $d=0.57$, $p<0.01$, with significant heterogeneity across studies, $Q(6)=15.82$, $p<0.01$. Interestingly, larger effects were observed when examining behavioral outcomes, $d=0.82$ $p<0.001$. Only two of these studies reported questionnaire outcomes, with no significant differences in effect between PL and WLC. Overall, these findings indicate that placebo effects are significant, and similar in magnitude to the effect sizes favoring non-exposure treatments over no treatment.

3.6. Examination of exposure treatment variations

3.6.1. Efficacy of in vivo exposure compared to other exposure modalities

Seven studies compared *in vivo* exposure to an alternative mode of exposure to the phobic target. This comparison yielded a significant advantage of exposure conducted *in vivo* over alternative exposure modalities at post-treatment

$d=0.38$, $p<0.001$. However, at follow-up, the advantage of *in vivo* exposure was no longer significant, $d=0.20$, $p=0.09$. These findings were consistent across measurement domains. Effect sizes at post-treatment were in the moderate range for both behavioral, $d=0.37$, $p<0.01$, and questionnaire, $d=0.36$, $p<0.01$ indices, favoring *in vivo* exposure. However, effect sizes at follow-up were not significant for either of the assessment domains. Taken together, these data indicate that, while *in vivo* exposure may be more efficacious than other exposure modalities in the short-term, this advantage is no longer present at follow-up.

3.6.2. Efficacy of exposure plus cognitive techniques compared to exposure alone

Five studies were available for this comparison. Contrary to prediction, exposure augmented with cognitive procedures did not outperform exposure treatment alone. The overall composite effect size was not significant, and comparisons of the two treatments for each assessment domain separately revealed no significant advantage for combining exposure with cognitive techniques.

3.6.3. Efficacy of multiple-session exposure treatments relative to single-session treatments

Four studies were available for this comparison. The overall effect size was not significant at post-treatment, $d=0.11$, $p>0.10$ or at follow-up, $d=0.18$, $p<0.10$, with non-significant heterogeneity across studies. Examination of each assessment domain separately revealed an advantage of five sessions over 1 session for questionnaire outcomes at follow-up, $d=0.35$, $p=0.06$. These findings suggest that there may be some advantage of multiple-session over single-session exposure treatment for enhancing treatment outcome at follow-up. However, the small number of studies included in this comparison warrants caution.

3.7. Analyses of effect size moderators

Before testing the putative moderators, we first evaluated whether there was significant heterogeneity in the effect sizes for each outcome using the random-effects macro-designed by Lipsey and Wilson (2001). For each comparison, we tested for homogeneity of effects within each type of measure, and we found significantly heterogeneous effects only for the questionnaire measures in the exposure vs. WL comparison, $\chi^2(21, N=22)=77.32$, $p<0.001$ and the all treatments vs. WL comparison, $\chi^2(26, N=27)=85.90$, $p<0.001$. Therefore, we tested for moderators only for these comparisons.

Only one putative moderator was significant. An inverse variance weighted regression model indicated that an increase in the number of treatment sessions was associated with a larger effect size for the exposure vs. WL comparison, $B=0.41$, $z(19)=2.13$, $p<0.05$, and for the all treatments vs. WL comparison, $B=0.43$, $z(24)=2.58$, $p<0.01$. Type of phobia, date of publication, and degree of therapist involvement were not found to moderate treatment outcome.

4. Discussion

This meta-analysis included 33 RCTs investigating psychological treatments for specific phobia. Most (82%) investigated an exposure-based treatment, 60% included a follow-up assessment and 42% reported percentage of participants achieving clinically significant improvement. This meta-analysis yielded several interesting findings. First, the average participant receiving treatment was better off than approximately 85% of non-treated participants. This overall treatment effect size was larger than that reported for similar comparisons of treatments for depression, panic disorder, and GAD (Westen & Morrison, 2001), attesting to the efficacy of the treatments delivered in published treatment studies of specific phobias.

4.1. How effective is exposure treatment?

When comparing exposure treatments to no-treatment control groups, the effects sizes were larger than those found in meta-analyses investigating exposure treatment for social anxiety disorder (Gould, Buckminster, Pollack, Otto, & Yap, 1997) and panic disorder (Gould, Otto, & Pollack, 1995). However, they were somewhat smaller than the effect sizes reported for exposure and response prevention treatment of OCD (van Balkom et al., 1994). Our findings with respect to the relative superiority of exposure treatment to alternative treatments (both active and placebo) offer more compelling evidence in support of the efficacy of exposure treatments for specific phobia. Moreover, the magnitude of the effect sizes

(i.e., moderate to large) as well as their robustness across multiple domains of assessment and time periods provide greater confidence in the conclusion that exposure treatments represent the treatment of choice for specific phobias.

4.2. How strong is the placebo response for specific phobia treatment?

To our knowledge, this is the first review to examine the placebo response in specific phobia. Contrary to expectation, treatments classified as "placebo" showed a moderate effect size when compared to no treatment. However, it should be noted that there were too few comparisons to test whether placebo treatments outperform no treatment at follow-up. The nature of the placebo treatments varied considerably and included things such as the administration of pulsed audio/photic stimulation (Powers et al., 2004) to expectations of subliminal fear-reducing messages (Gauthier & Marshall, 1977). Based on these findings, it is suggested that treatment efficacy studies routinely include placebo treatments in order to provide a more stringent test of new presumed "active" treatments. The mechanisms through which placebo treatments exert their effects have yet to be studied. One possibility is that placebo treatments enhance treatment outcome expectations, which in turn motivate the phobic individual to engage in self-directed exposure. Unfortunately, the studies that did include a placebo vs. wait-list comparison did not assess participants' self-exposure between sessions. However, if placebo treatments exert their effects by motivating subjects to engage in self-directed exposure between sessions, one might expect the difference between exposure and placebo treatments to diminish at follow-up. Such was not the case; in fact the exposure vs. placebo treatment difference at follow-up exceeded that at post-treatment.

4.3. Efficacy of alternative treatments

Given the low rates of treatment seeking among specific phobia sufferers (Regier et al., 1993), efficacious alternatives to exposure treatment are needed for the sizeable group of patients who refuse to undergo treatments involving direct confrontation with the phobic target. Our review found six studies that compared a non-exposure treatment to either a wait-list condition ($N=5$) or a placebo control ($N=1$). Contrary to the widely-held belief that non-exposure treatments offer limited benefit to specific phobic sufferers (Choy et al., 2007), our findings revealed that phobic participants derive significant benefit from non-exposure treatments when compared with no treatment. However, the limited data available suggests that this benefit may simply reflect non-specific treatment effects given that the one study comparing a non-exposure treatment (cognitive restructuring) to a placebo treatment (Szymanski & O'Donahue, 1995) found no significant differences between them. This finding is also consistent with our data showing similar effect sizes for exposure vs. placebo ($d=0.48$) and exposure vs. non-exposure treatments ($d=0.44$). Taken together, these data suggest that the non-exposure treatments studied to date are probably no more efficacious than placebo treatment. It is interesting to note that effect sizes for non-exposure treatments were larger for questionnaire measures ($d=1.07$) than for behavioral measures ($d=0.81$). One interpretation may be that these alternative treatments are equivalent in demand characteristics to placebo conditions. This would help explain the discrepancy in questionnaire and behavioral data as questionnaires are easier to "fake" than are behavioral assessments. Nevertheless, the development of more potent alternatives to exposure treatment remains an important goal given the reluctance of many phobic individuals to undergo exposure treatment (Marks, 1992).

Not surprisingly, our findings revealed that the superiority of exposure treatments over "bona fide" alternative treatments diminishes at follow-up (yet remains statistically significant). However, these findings should be viewed with caution given the small number of comparisons and the biases introduced by subject attrition. One explanation for this finding is that phobic individuals who show short-term improvement regardless of the type of treatment they receive may be more apt to engage in naturalistic exposure during the follow-up period, thus moving them closer to the exposure-treated participants at follow-up.

Future research is needed on the longer-term effects of alternatives to exposure-based treatments. Unfortunately, the comparisons involving alternatives to exposure in this meta-analysis were too few to examine specific variants of non-exposure treatments. By collapsing the non-exposure treatments into one category, we do not intend to suggest that all non-exposure treatments are equally efficacious.

4.4. Are certain variations of exposure treatment more effective than others?

Telch (2004) has argued that not all exposure treatments are created equal. Indeed, studies that have manipulated parameters of exposure provide evidence suggesting that the way in which exposure treatment is conducted can

significantly impact treatment outcome (Kamphuis & Telch, 2000; Powers et al., 2004; Rowe & Craske, 1997a,b; Sloan & Telch, 2002; Telch et al., 2004; Wolitzky & Telch, in press).

One conclusion offered from several qualitative reviews is that *in vivo* exposure is more effective than other modes of exposure (Antony & Barlow, 2002; Choy et al., 2007). However, both of these reviews point to the lack of direct comparisons between *in vivo* exposure and alternative modes of exposure treatment. Our findings call into question this conclusion. While *in vivo* exposure outperformed alternative modes of exposure at post-treatment, the superiority of *in vivo* exposure was no longer present at follow-up. Additional analyses were performed to determine whether the lack of differences at follow-up were due to greater return of fear in the *in vivo exposure* treatment or greater improvement from the post-treatment to follow-up period for those receiving alternative modes of exposure treatment. Among those receiving *in vivo exposure*, four of the five studies reporting follow-up data showed no additional improvement from post to follow-up (Emmelkamp et al., 2002; Gilroy et al., 2000; Heading et al., 2001; Rentz et al., 2003) Only one study showed continued post to follow-up improvement for those receiving *in vivo* exposure (Rothbaum et al., 2000). In contrast, among those treated with an alternative mode of exposure, four of the five studies showed additional improvement from post to follow-up and one study showed maintenance of treatment gains.

These findings suggest that exposure treatment when conducted *in vivo* may lead to more rapid improvement relative to less direct forms of exposure treatment. However, the advantage of *in vivo* exposure is no longer present at follow-up due to continued improvement for those receiving less direct forms of exposure as opposed to greater return of fear among those receiving *in vivo* exposure. Although the lack of significant differences at follow-up may be due to continued naturalistic exposure during the post-treatment to follow-up period, none of the studies reported data on this variable. Another possibility is that a ceiling effect may have been present for those who received *in vivo* exposure. Even though those receiving non-*in vivo* exposure continued to improve from post to follow-up, their fear reduction did not surpass that of those receiving *in vivo* exposure. Further, these two possibilities are not mutually exclusive. Improvement for those in the *in vivo* exposure conditions may have reached a ceiling effect, while those receiving non-*in vivo* exposure modalities may have had more room for improvement, which they achieved via naturalistic exposure from the post to follow-up periods.

Our findings examining the effects of treatment dose deserve comment. Although many patients who undergo one-session treatment protocols show clinically significant improvement (e.g., Powers et al., 2004), our findings call into question the assertion that one session of exposure is the "treatment of choice" for specific phobias (Öst et al., 2001, p. 168). Rather, our findings showed that those treated with five sessions of exposure-based treatment were reporting moderately more improvement on self-report measures of phobic symptoms relative to those treated in just one session. Further probing of the differences at follow-up suggest that the superiority of multiple sessions vs. one session was not due to greater return of fear but rather continued improvement from the post to follow-up period for those receiving multiple sessions. These findings should be interpreted cautiously, as few studies were included in this comparison. However, our moderator analyses showing that number of treatment sessions predicted treatment response (with more sessions associated with better outcome) provides additional support for the value of multiple treatment sessions.

Does the addition of cognitive strategies enhance treatment outcome? The studies included in this comparison showed considerable heterogeneity in their findings, with some showing large and consistently positive effect sizes favoring exposure with a cognitive augmentation strategy (e.g., Kamphuis & Telch, 2000; Sloan & Telch, 2002) and others showing large positive effect sizes for some measures and large negative effect sizes for other measures (e.g., Szymanski & O'Donahue, 1995). These inconsistencies may be due in part to the heterogeneity of interventions that are labeled "cognitive".

4.5. Factors predicting treatment outcome

With the exception of treatment length, none of the other potential prognostic variables included in the moderator analyses were significantly associated with treatment outcome. These data suggest that the effect sizes for the major comparisons of interest were not significantly qualified by phobia subtype, level of therapist involvement, or date of publication. We were particularly surprised over the null findings with respect to type of phobia in light of Choy et al.'s (2007) conclusion that certain subtypes of specific phobia may respond more favorably to specific types of treatment (e.g., cognitive therapy is most helpful in claustrophobia). Note, that their conclusions were based on data from individual studies that report on the efficacy of a particular treatment approach for a particular type of phobia (e.g., applied tension for blood-injury phobia) and not controlled effect sizes from meta-analytic findings or reported treatment moderator effects within a controlled treatment trial comparing several distinct treatments.

4.6. Study limitations

The validity of the inferences drawn from meta-analytic investigations is partly a function of the number, quality, and limitations of the individual studies upon which each meta-analysis is based. In the process of reviewing the existing treatment literature of psychosocial treatments for specific phobia, several significant limitations of the individual studies became apparent. One significant limitation of virtually all the studies was the failure to report participants' use of self-guided exposure during the period between post-treatment and follow-up. A related limitation, also noted by Choy et al. (2007), is the failure of most studies to report whether their treatment protocol encouraged study participants to practice confronting their phobic targets between sessions. We suggest that future studies report on the level of self-guided exposure after the prescribed treatment protocol is over, and examine whether those who engaged in self-directed exposure between post-treatment and follow-up continued to improve or maintained gains more than those who did not. We would also like to suggest the need for the experimental investigation of the effects of explicit instructions for self-guided exposure on long-term treatment efficacy.

A second limitation of the studies reviewed was the failure of most studies to include drop-outs in the outcome analyses. Consequently, our effect sizes for the comparisons of interest are based on the subset of participants who completed treatment and thus one should not assume our findings generalize to intent-to-treat samples. A related issue is the failure of most studies to report the percentage of those who refused treatment, thus precluding the investigation of possible differences in *palatability* of various phobia treatments. It is recommended that future studies routinely report refusal rates to address this issue.

Third, It should also be noted that the number of studies testing a non-exposure treatment were too few to allow more fine grained-analyses examining the efficacy of exposure treatments vs. individual non-exposure alternative treatments such as cognitive therapy or relaxation treatments. Hence, our findings showing exposure treatments outperformed non-exposure alternative treatments should be interpreted with some degree of caution as should our finding showing that non-exposure treatments outperform no treatment. A similar limitation should be noted with respect to our findings on whether cognitive procedures enhance the efficacy of exposure treatments. Because of the small number of studies testing individual cognitive techniques, we were forced to use a lumping approach in which studies of any cognitive augmentation strategy were lumped together. Clearly, more studies are needed that examine alternatives to exposure-based methods. These should be studied in the context of a "stand alone" treatment as well as within the context of an exposure augmentation approach.

Finally, our selection of moderator variables was constrained by the type of information supplied consistently across studies. Potentially important moderators, such as trait anxiety, distress tolerance, and psychiatric comorbidity could not be evaluated because either no information was provided for these variables, information was not provided in a way that could be coded for moderator analysis, or there was very little variation across studies on the variable of interest (e.g., the majority of studies used a college-aged sample). The significant heterogeneity observed for several of the comparisons suggests other variables may be moderating treatment efficacy. Unfortunately, our analytical options were constrained by the data provided.

4.7. Conclusions

What conclusions can be drawn from this quantitative review of psychosocial treatments for specific phobia? First, our findings are consistent with other qualitative reviews (Barlow, Moscovitch, & Micco, 2004; Choy et al., 2007) indicating that exposure-based treatments appear to be the most potent and durable of the treatments currently subjected to empirical evaluation. Moreover, despite the brief duration of these treatments, the effect sizes relative to no treatment rank them as one of the most potent treatments for any psychiatric condition. Second, contrary to the assertion that one session of exposure treatment is as effective as multiple sessions, the data lead us to conclude that multiple exposure sessions are more effective than one session of exposure particularly at follow-up and suggest that clinicians should deliver treatment in multiple sessions to enhance long-term treatment gains. Third, our findings suggest that overall, non-exposure treatments do outperform no treatment, but the magnitude of this effect is about the same as that for placebo vs. no treatment, suggesting the possibility that the efficacy of non-exposure treatments are the result of non-specific factors such as expectancy effects. Fourth, our findings suggest that those presenting with specific phobia display a moderate placebo response rate and highlight the importance of controlling for non-specific treatment effects in future efficacy studies. Fifth, we found no evidence to support the recent conclusion of Choy et al. (2007) that acute

treatments for specific phobia have differential efficacy among the various phobia subtypes. Rather, our moderator analyses found no significant moderator effect of specific phobia subtype on treatment outcome. We conclude that gaps in the existing treatment literature do not allow this question to be answered at this time and further conclude that treatment research in specific phobia will advance considerably by the addition of studies that test multiple treatments with participants presenting with different phobia subtypes. Hopefully, data from studies like these will provide the basis for developing empirically informed treatment matching strategies for the future.

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