Treatment of Obsessive Compulsive Disorder

Martin E. Franklin¹ and Edna B. Foa²

¹Child/Adolescent OCD, Tics, Trichotillomania and Anxiety Group, Department of Psychiatry, University of Pennsylvania School of Medicine, and ²Center for the Treatment and Study of Anxiety, Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia, Pennsylvania 19104; email: marty@mail.med.upenn.edu

Annu. Rev. Clin. Psychol. 2011. 7:229-43

The *Annual Review of Clinical Psychology* is online at clinpsy.annualreviews.org

This article's doi: 10.1146/annurev-clinpsy-032210-104533

Copyright © 2011 by Annual Reviews. All rights reserved

1548-5943/11/0427-0229\$20.00

Keywords

cognitive behavior therapy, exposure plus response prevention, serotonin reuptake inhibitors, empirically supported treatments, dissemination

Abstract

Obsessive compulsive disorder (OCD) is characterized by the presence of intrusive, anxiety-provoking thoughts, images, or impulses along with repetitive behaviors or mental acts designed to reduce obsessional distress. OCD is associated with significant functional impairment, psychiatric comorbidity, and compromised quality of life. Fortunately, substantive progress has been made in the past several decades in the development and empirical evaluation of treatments for OCD across the developmental spectrum. The current review begins with a discussion of the clinical presentation of OCD and psychological theories regarding its etiology and maintenance. A detailed discussion follows of exposure plus response prevention, the psychosocial treatment that has garnered the most evidence for its efficacy. A summary of the extant treatment outcome literature related to exposure plus response prevention as well as cognitive therapies, pharmacotherapies, and combined approaches is then presented. Recommendations for future clinical and research directions are then provided.

Contents	
DEFINITION OF OBSESSIVE	
COMPULSIVE DISORDER	230
CLINICAL PRESENTATION	230
Prevalence and Course	230
Comorbidity	231
COGNITIVE AND BEHAVIORAL	
THEORETICAL MODELS	231
EMPIRICALLY SUPPORTED	
TREATMENTS	232
Exposure and Response	
Prevention	232
Review of Evidence Base	
for EX/RP Procedures	233
Cognitive Therapies	234
Serotonergic Medications	235
DISSEMINATION OF	
EMPIRICALLY SUPPORTED	
TREATMENTS	236
ADAPTATIONS OF EXPOSURE	
AND RESPONSE PREVENTION	
FOR USE WITH CHILDREN	
AND ADOLESCENTS	237
IMPLICATIONS OF THE	
RESEARCH LITERATURE	
FOR CLINICAL	
DECISION-MAKING	237
WHAT DO WE STILL NEED	
TO KNOW?	238

bsessive mpulsive disorder The Diagnostic and Statistical Manu Disorders Froughth Edition Text Region

Obsessive compulsive disorder (OCD): obsessions and/or compulsions of sufficient clinical severity (e.g., greater than one hour/day) and associated with functional impairment and/or distress

Obsessions: intrusive or unwanted thoughts, images, or impulses that provoke anxiety or distress The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision (DSM-IV-TR; Am. Psychiatr. Assoc. 2000) defines obsessive compulsive disorder (OCD) by the presence of recurrent obsessions and/or compulsions that interfere substantially with daily functioning. Obsessions are "persistent ideas, thoughts, impulses, or images that are experienced as intrusive and inappropriate and cause marked anxiety or distress" (p. 457). Common obsessions are repeated thoughts about contamination, causing harm to others, and doubting whether one locked the front door.

DEFINITION OF OBSESSIVE

Compulsions are "repetitive behaviors or mental acts the goal of which is to prevent or reduce anxiety or distress" (Am. Psychiatr. Assoc. 2000, p. 457). Common compulsions include hand washing, checking, and mental compulsions (e.g., repeated praying silently). A functional link between obsessions and compulsions is typically evident: for example, in the DSM-IV field trial on OCD, over 90% of participants reported that their compulsions aim to either prevent harm associated with their obsessions or to reduce obsessional distress (Foa et al. 1995). For example, the obsessional thought of an OCD patient that he/she might harm someone by neglecting to lock the door will give rise to anxiety or distress. Compulsively checking the door is a behavior that attempts to reduce distress and reassure the patient that the feared consequence will not occur. Therefore, if the patient does not demonstrate a clear relationship between the obsession and the compulsion (obsessions are distressing and compulsions aim at reducing this distress), another diagnosis should be considered.

In order to distinguish diagnosable OCD from the virtually ubiquitous occasional phenomena of unwanted thoughts and repetitive behaviors reported by the vast majority of individuals without OCD (Crye et al. 2010, Rachman & de Silva 1978), obsessions and/or compulsions must be found to be of sufficient severity to cause marked distress, be time-consuming, and interfere with daily functioning. If another Axis I disorder is present, the obsessions and compulsions cannot be restricted to the content of that disorder (e.g., preoccupation with food in the presence of eating disorders).

CLINICAL PRESENTATION

Prevalence and Course

The National Comorbidity Survey Replication Study involving over 9,000 adult participants in the United States estimated that the 12-month prevalence rate of OCD was 1.0% (Kessler et al. 2005); epidemiological studies with children

and adolescents suggest similar lifetime prevalence rates in these samples (e.g., Flament et al. 1990, Valleni-Basile et al. 1994). Slightly more than half of the adults suffering from OCD are female (Rasmussen & Tsuang 1986), whereas a 2:1 male to female ratio has been observed in several pediatric clinical samples (e.g., Hanna 1995, Swedo et al. 1989). Development of OCD is typically gradual, but more rapid onset has been reported in some cases. The course of OCD is most often chronic with some waxing and waning of symptoms, with patients reporting some responsiveness to external stressors as well. In rare pediatric cases, however, onset is very sudden (e.g., overnight) and associated with strep infection; treatment of the infection is then associated with substantial reduction of symptoms, but recurrence of infection is associated with symptom exacerbation [Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS), Swedo et al. 1998].

Comorbidity

OCD in adults usually co-occurs with other disorders, with unipolar depression and anxiety disorders being the most common comorbid conditions (e.g., Torres et al. 2006). A relationship between OCD and eating disorders has also been identified, in that approximately 10% of women with OCD had a history of anorexia nervosa (Kasvikis et al. 1986), as did more than 33% of those with bulimia (Hudson et al. 1987, Laessle et al. 1987). Because certain comorbidity patterns have been found to compromise treatment outcome (e.g., severe depression in adults, Abramowitz et al. 2000; externalizing disorders in youth, Storch et al. 2008) comorbid disorders should be screened for and taken into account during the treatment decision phase as well as during the treatment phase itself.

COGNITIVE AND BEHAVIORAL THEORETICAL MODELS

Several cognitive behavioral theories about the development and maintenance of OCD

symptoms have been put forward. Dollard & Miller (1950) adopted Mowrer's two-stage theory (1939, 1960) to explain the development and maintenance of fear/anxiety and avoidance in OCD. Mowrer's theory maintains that a neutral event stimulus (conditioned stimulus; CS) comes to elicit fear when it is repeatedly presented together with an event that by its nature causes pain/distress (unconditioned stimulus; UCS). The CS can be mental events, such as thoughts, and/or physical entities, such as bathrooms and trash cans. After fear/anxiety/distress to the CS is acquired, escape or avoidance behaviors are developed to reduce the anxiety. In OCD, the behavioral avoidance and escape take the form of repeated compulsions or rituals. Like other avoidance behaviors, compulsions are maintained because they indeed reduce the distress. Not only does Mowrer's theory adequately explain fear acquisition, it is also consistent with observations of how rituals are maintained. In a series of experiments, Rachman and colleagues (Hodgson & Rachman 1972, Roper & Rachman 1976, Roper et al. 1973) demonstrated that obsessions increase obsessional distress and compulsions reduce this distress. This conceptualization of a functional relationship between obsessions and compulsions influenced the definitions of OCD in the DSM-III (Am. Psychiatr. Assoc. 1980) and its successors.

Foa & Kozak (1986) proposed that OCD is characterized by erroneous cognitions. First, OCD sufferers assign a high probability of danger to situations that are relatively safe. For example, an individual with OCD will believe that if he touches public doorknobs without thoroughly washing his hands afterward, the germs on the doorknob will cause serious disease to him and/or to people whom he touched with his dirty hands. Second, individuals with OCD exaggerate the cost of the bad things that they think can happen. For example, contracting a minor cold is viewed as a terrible thing. For others with more abstract fears, the fear responses are associated with mistaken meaning rather than with a particular stimulus. For example, some patients who are disturbed by

Compulsions:

behaviors or mental acts engaged in purposefully to reduce obsessional distress or the likelihood of a feared outcome

Comorbidity:

psychiatric conditions that co-occur with an index disorder Exposure plus
response prevention
(EX/RP): form of
CBT involving
prolonged
confrontation with
feared stimuli and
encouragement to
refrain from
compulsions or other
forms of avoidance

perceived asymmetry and who reduce their distress by rearranging objects do not fear the objects themselves, nor do they anticipate disaster from the asymmetry. Rather, they are upset by their view that certain arrangements of stimuli are "improper." Foa & Kozak (1986) further suggested that individuals with OCD conclude that in the face of lack of evidence that a situation or an object is safe, they are dangerous, and therefore OCD sufferers require constant evidence for safety. For example, in order to feel safe, an OCD sufferer requires a guarantee that the dishes in a given restaurant are extremely clean before eating in this restaurant. People without OCD, on the other hand, conclude that if they do not have evidence that a situation is dangerous, then it is safe. Thus, a person without OCD would eat from the dishes in the restaurant unless he has clear evidence that they are dirty.

Cognitive accounts of OCD have also been proposed, with the most influential of these being the model posited by Salkovskis (1985). According to this model, intrusive, obsessional thoughts are stimuli that may provoke certain types of negative automatic thoughts. In particular, an exaggerated sense of responsibility and self-blame are the central themes in the OCD belief system. Neutralization, in the form of behavioral or cognitive compulsions, can be understood as an attempt to reduce this sense of responsibility and to prevent blame. Salkovskis further proposed that five dysfunctional assumptions characterize obsessive-compulsives and differentiate them from persons without OCD: (a) Having a thought about an action is like performing the action; (b) failing to prevent (or failing to try to prevent) harm to self or others is the same as having caused the harm in the first place; (c) responsibility is not attenuated by other factors (e.g., low probability of occurrence); (d) not neutralizing when an intrusion has occurred is similar or equivalent to seeking or wanting the harm involved in that intrusion to actually happen; (e) one should (and can) exercise control over one's thoughts (Salkovskis 1985, p. 579). Thus, although the obsession may be ego-dystonic, the automatic thought that it elicits will be ego-syntonic. This model suggests that treatment of OCD should largely focus on identifying the erroneous assumptions and modifying the automatic thoughts.

By extension, treatments that are based on each of the aforementioned theoretical models ought to result in a particular emphasis in therapy that is tied to the mechanism by which the OCD is thought to be maintained; for example, the more behavioral conceptualizations would lead therapists to strongly emphasize reduction of all forms of passive and active avoidance to permit learning to occur, whereas the cognitive theories should yield a particular focus in treatment on challenging the underlying belief system in order to affect symptom change. Theories that blend cognitive and behavioral elements, such as Foa and Kozak's Emotional Processing Theory (Foa & Kozak 1986), would be convergent with the use of cognitive and behavioral treatment strategies to provide the patient with corrective information about the world and about his own fear responses. The treatments described below flow from these prevailing conceptual models, yet evidence for the efficacy of these interventions provides only partial support for the theoretical foundations upon which they were built.

EMPIRICALLY SUPPORTED TREATMENTS

Exposure and Response Prevention

Brief description of procedures. The psychosocial intervention that has garnered the most empirical support is exposure and response prevention (EX/RP), which has been studied around the world for the past 40 years and has proven to be a remarkably efficacious and durable treatment for patients with OCD across the developmental spectrum (Abramowitz et al. 2005, Natl. Inst. Health Clin. Excell. 2005, Rosa-Alcazar et al. 2008). Current EX/RP treatments, which are based largely on the blended theoretical model proposed by Foa & Kozak (1986), typically include prolonged exposure to obsessional cues,

procedures aimed at blocking rituals, and informal discussions of mistaken beliefs that are often conducted in anticipation of exposure exercises. Exposures are most often done in real-life settings (in vivo) and involve prolonged contact with specific feared external (e.g., contaminated surfaces) or internal (e.g., images of having sex with religious figures) stimuli that the patient reports as distressing. When the patient also reports specific consequences that they fear would occur if they refrained from rituals or other forms of avoidance, these fears can also be addressed via imaginal exposure, which often consists of creating very detailed image scripts and then listening to or reading these scripts repeatedly until they are perceived as less anxiety provoking. Following from the theory of Foa & Kozak (1986), in vivo and imaginal exposure exercises are designed specifically to prompt obsessional distress; it is believed that repeated, prolonged exposure to feared thoughts and situations will provide information that disconfirms mistaken associations and evaluations held by the patients and thereby promotes habituation (Foa & Kozak 1986). Exposure is typically conducted gradually, with situations provoking moderate distress confronted before more upsetting ones. Exposure practices are routinely assigned for completion between sessions, and patients are also asked to refrain from rituals to the extent possible; complete ritual abstinence is the stated goal, and patients are reminded that the negative reinforcement provided by ritualizing maintains fear, whereas refraining from rituals promotes its dissipation.

Review of Evidence Base for EX/RP Procedures

Exposure versus response prevention versus EX/RP. An experiment conducted by Foa and colleagues (1984) highlighted the importance of employing both exposure and response prevention in the treatment of OCD. Patients with washing rituals were randomly assigned to treatment by exposure only (EX), ritual prevention only (RP), or their combination (EX/RP); patients in each condition were found to be

improved at both post-treatment and followup, but EX/RP was clearly superior to the single-component treatments on almost every symptom measure at both assessment points. These findings clearly suggest that exposure and ritual prevention should be implemented concurrently; accordingly, it is important to convey this information to patients, especially when they are experiencing difficulty refraining from rituals or engaging effectively in exposure exercises during and between sessions.

Implementation of response prevention.

Instructions and encouragement to refrain from ritualizing and avoidance are strongly emphasized in EX/RP, and the success of these efforts is integral to good outcome (Foa et al. 2002). The therapist can support the patient in doing so providing encouragement and suggestions about alternatives to ritualizing and also by training family members in how best to respond when the patient either reports that he is struggling with urges to ritualize or when he is already in the midst of engaging in rituals. More extensive coaching of family members is typically needed when the patient is a child or adolescent as well as when reassurance from family members is a central form of relief from obsessional distress.

Gradual versus abrupt exposures. Although no differences in outcome were detected in a study comparing patients who confronted the most distressing situations from the start of therapy to those who confronted less distressing situations first, patients themselves reportedly preferred the more gradual approach (Hodgson et al. 1972). Given that patient motivation and agreement with treatment goals is a core element of successful EX/RP, situations of low-to-moderate difficulty are usually confronted in treatment first, followed by several intermediate steps before the most distressing exposures are attempted. Thus, exposure typically proceeds at a pace that is acceptable to the patient, and no exposure is intentionally attempted without the patient's approval. That said, we do find that clinically

Cognitive therapy:

psychotherapy that focuses on techniques such as rational argument and behavioral experiments in an attempt to modify mistaken beliefs it is important to set an ambitious agenda with respect to the treatment hierarchy and to try to address the most difficult items early enough in treatment that these items can be confronted repeatedly before the end of the acute phase of EX/RP.

Use of imaginal exposure. An early randomized study provided empirical support for a treatment protocol that included imaginal exposure, in vivo exposure, and ritual prevention compared to a program that did not include imaginal exposure (Foa et al. 1980, Steketee et al. 1982), yet a second study in a separate lab failed to find such an additive effect (de Araujo et al. 1995). These protocols varied from each other procedurally (e.g., 90-minute versus 30-minute imaginal exposures respectively), and thus the source of these studies' inconsistencies cannot be identified. That said, in our clinical work we have found imaginal exposure to be helpful for patients who report that disastrous consequences will result if they refrain from rituals. Because many patients' feared consequences cannot be readily translated into in vivo exposure exercises (e.g., turning into another person), imaginal exposure allows the patient an opportunity to confront these feared thoughts directly. Also, the addition of imagery to in vivo exposure may circumvent the cognitive avoidance strategies used by patients who try intentionally not to permit elaboration of the consequences of exposure while confronting feared situations in vivo. For patients who only report distress as a consequence to refraining from rituals and avoidance behaviors, imaginal exposure exercises may be superfluous.

Session frequency. Although the optimal frequency of exposure sessions has yet to be established, it appears that good outcomes can be achieved using a weekly, twice weekly, or intensive treatment format. Intensive exposure therapy programs that have achieved excellent results (e.g., Foa et al. 2005, Franklin et al. 2000) typically involve daily sessions over the course of approximately one month, but favorable outcomes have also been achieved with

more widely spaced sessions (e.g., Abramowitz et al. 2003, de Araujo et al. 1995, Storch et al. 2007, Warren & Thomas, 2001). Clinically, we have found that less frequent sessions may be sufficient for highly motivated patients with mild-to-moderate OCD symptoms who readily understand the importance of daily exposure homework; those with more severe symptoms or those with other impediments to compliance with EX/RP tasks are typically offered intensive treatment to reduce the likelihood of attenuated outcome.

Cognitive Therapies

Out of the cognitive conceptualizations of OCD came several treatment protocols that emphasized the primacy of maladaptive beliefs and their amelioration in the reduction of OCD symptoms. Several of the earliest forms of these treatments did not fare especially well (Emmelkamp et al. 1988, 1990); van Balkom et al. (1998) later found that six weeks of cognitive therapy without behavioral experiments and EX/RP without discussion of disastrous consequences led to OCD symptom reductions of 20% and 23%, respectively, which, benchmarked against EX/RP outcomes from other labs, was generally less robust than had been found previously. In contrast, more recent studies in which treatment was based on OCD-specific cognitive models (Cottraux et al. 2001; McLean et al. 2001; Whittal et al. 2005, 2008) found clinically significant and equivalent symptom reductions for more cognitively oriented protocols and EX/RP, respectively, although procedural overlap in the form of behavioral experiments in the cognitive conditions makes their findings somewhat difficult to interpret from a purely theoretical standpoint. Vogel and colleagues' randomized augmentation study demonstrated that the addition of cognitive procedures following a course of EX/RP did yield further improvement (Vogel et al. 2004), but this design did not address the question of whether full integration of cognitive and behavioral techniques yields better outcome than each technique alone. More recently, Whittal and colleagues (2010) failed to find a difference between a cognitive therapy that included behavioral elements and stress management training for individuals with obsessions not accompanied by prominent overt compulsions; substantial and lasting benefits were observed in both groups at the end of the acute phase of the trial and at a 12 months post-treatment assessment.

Serotonergic Medications

Efficacy. The use of serotonergic medications in the treatment of OCD has received a great deal of attention in the past two decades. In controlled trials with adults, clomipramine (CMI) has been found consistently superior to placebo (e.g., DeVeaugh-Geiss et al. 1989, Foa et al. 2005); similar results have been obtained with the selective serotonin reuptake inhibitors (SS-RIs) fluvoxamine (Greist et al. 1995), fluoxetine (e.g., Tollefson et al. 1994), sertraline (e.g., Greist et al. 1995), and paroxetine (e.g., Zohar et al. 1996). Accordingly, each of these medications has been approved by the Food and Drug Administration as treatments for adult OCD. In youth with OCD, CMI (DeVeaugh-Geiss et al. 1992) and the SSRIs fluvoxamine (Riddle et al. 2001), sertraline (March et al. 1998), and fluoxetine (Geller et al. 2001) were found superior to placebo in multicenter Food and Drug Administration registration trials. The pediatric OCD pharmacotherapy literature is consistent with the much larger adult literature in revealing (a) little PBO effect, (b) a typical 30% to 40% reduction in OCD symptoms, and (c) clinically significant residual symptoms on average even after a medication trial of adequate dose and duration. Moreover, amelioration of symptoms is typically maintained only as long as the drug is continued (e.g., Simpson et al. 2004). Studies that have employed slower drug taper schedules have yielded less substantial relapse rates than have those with faster taper schedules, but the follow-up data nevertheless converge to suggest that maintenance treatment is necessary in order to sustain achievements attained with pharmacotherapy alone (Dougherty et al. 2002).

Relative and combined efficacy of EX/RP and pharmacotherapy. Although there is clear evidence that pharmaceutical treatment with serotonergic medications and EX/RP are each effective for OCD, studies directly comparing their relative and combined efficacy have generally been methodologically complex and yielded equivocal findings (Foa et al. 2002). The largest and perhaps most definitive of these studies in adult OCD examined the relative and combined efficacy of CMI and intensive EX/RP; post-treatment findings indicated that each of the active treatments was superior to placebo, EX/RP was superior to CMI, and the combination of the two treatments (COMB) was not superior to EX/RP alone (Foa et al. 2005); relapse was more evident following treatment discontinuation in the CMI group than in either of the treatments that included intensive EX/RP (EX/RP, COMB; Simpson et al. 2004). However, the design used in this study may not have optimally promoted an additive effect for CMI because the intensive portion of the EX/RP program was largely completed before patients reached their maximum dose of CMI. In addition, combined treatment effects may be more evident when intensive EX/RP is not used (Foa et al. 2002). Convergent with that view, an additive effect for combined treatment was found in a randomized controlled study examining the efficacy of weekly cognitive behavior therapy (CBT), sertraline, and their combination in pediatric OCD (Pediatr. OCD Treat. Study Team 2004). However, examination of site effects in that trial indicated that the CBT monotherapy effect at one site was very large, and no additive effect for combined treatment was found at this site.

Despite the absence of definitive empirical support for combined treatment over EX/RP alone, many continue to advocate combined procedures as the treatment of choice for OCD. In OCD subspecialty clinical practices, it is quite common to encounter patients presenting for EX/RP treatment who are already receiving serotonin reuptake inhibitors (SRIs), perhaps because of disparities in the availability of these treatments in most communities. Uncontrolled

Serotonin:

neurotransmitter evident in the brain that has been implicated at least in part in the pathophysiology of mood and anxiety disorders including OCD

SSRIs: selective serotonin reuptake inhibitors

COMB: combined treatment with exposure plus response prevention and medication

CBT: cognitive behavior therapy

SRIs: serotonin reuptake inhibitors

Open trial: treatment study in which patients were provided with a specific psychotherapy or pharmacotherapy without random assignment to condition

Randomized controlled trial (RCT): treatment study that includes random assignment to condition(s) and repeated assessment of outcome; considered the gold standard for establishing treatment efficacy

examinations of EX/RP treatment outcome for adults (Franklin et al. 2002) and youth (Franklin et al. 1998, Piacentini et al. 2002) treated in OCD fee-for-service clinics found no posttreatment differences between patients who received EX/RP alone and those who were on SRI medication when receiving EX/RP. From these data, as well as from the randomized trials that have examined relative and combined efficacy of EX/RP and pharmacotherapy, we can surmise that concomitant pharmacotherapy is not required for every patient to benefit substantially from EX/RP, but also that concomitant pharmacotherapy does not appear to inhibit EX/RP treatment response. Concomitant pharmacotherapy is often used in clinical practice to manage comorbid symptoms known to negatively impact EX/RP outcomes, such as depression and ADHD; optimal sequencing of these treatments has yet to be established empirically, however.

Pharmacotherapy and CBT augmentation strategies for SRI partial responders. The common problem of residual OCD symptoms and associated impairment even in SRI responders has promoted interest in developing augmentation strategies that would yield further symptom improvements. There is evidence from randomized trials in adult OCD patients who evidenced a partial response to SRIs that augmentations with the neuroleptic medications risperidone (McDougal et al. 2000) and quetiapine (Denys et al. 2004) were superior to augmentation with placebo, although the generally unfavorable side effect profile for neuroleptics complicates the assessment of the risk to benefit ratio for this treatment regimen. A recent randomized trial indicated that augmentation with twice-weekly CBT involving EX/RP yielded greater improvements and retention of gains than augmentation with stress management training for SRI partial responders (Foa 2010, Simpson et al. 2008), which is convergent with the open trial data in adults and in youth described above. Here again, however, the issue of limited availability of EX/RP

arises: Many of the empirically informed treatment recommendations for OCD (e.g., March et al. 1997) suggest that EX/RP should be used, yet the absence of therapists properly trained in its procedures curtail the extent to which it can be used, either as an initial treatment or as an augmentative treatment for SRI partial responders.

DISSEMINATION OF EMPIRICALLY SUPPORTED TREATMENTS

Much has been learned in the past several decades regarding the efficacy of treatments for OCD in adults and in youth, and this information now guides us in providing empirically informed treatment recommendations for the patients who seek our help. However, critics of the methods used to examine treatment efficacy have raised concerns about whether the findings from randomized trials designed specifically to emphasize internal validity have done so at the expense of generalizability to more typical clinical patients and psychotherapy practice settings (e.g., Westen et al. 2004). Many of these criticisms have been responded to in spirited and engaging academic debates (e.g., Crits-Christoph et al. 2005, Weisz et al. 2005), yet the prevailing question of how well these treatments hold up outside the academic research remains important to examine. Several studies have indicated that excellent EX/RP outcomes are not limited to highly selected randomized controlled trial (RCT) samples (Franklin et al. 2000) and can be achieved in OCD subspecialty private practice settings (Rothbaum & Shahar 2000, Warren & Thomas 2001) as well as in community agencies by supervised therapists who are not themselves OCD experts (e.g., Nakatani et al. 2009, Valderhaug et al. 2007). Thus, there now appears to be reason for encouragement regarding the applicability of EX/RP for complex cases and the transportability of this treatment bevond the academic context.

The effectiveness studies conducted thus far in OCD may serve as the building blocks for

the development of the needed infrastructure to disseminate EX/RP into the many communities where those with OCD cannot access it. The problem of limited CBT access, however, certainly is not specific to OCD: Shafran and colleagues (2009) have emphasized that empirically supported treatments for many disorders are rarely available in community settings and, even when they are, they are often delivered suboptimally. In order to facilitate the use of empirically supported CBT protocols in routine practice, these authors suggest that (a) treatment developers should state explicitly how existing trials address comorbidity; (b) clinicians should have easy access to training in diagnostic assessment and outcome measures; (c) effectiveness studies should provide adequate training and supervision for therapists when studying how well treatments work in routine clinical populations; (d) CBT trials and effectiveness studies should be analyzed for therapist effects and should establish the effects of levels of training on outcome; (e) reliable assessment of competence should be conducted; (f) more research should be conducted on methods of disseminating treatment procedures; (g) mechanisms of efficacious action should be studied more closely; and (b) methods to examine which patients require more intensive contact should be established.

ADAPTATIONS OF EXPOSURE AND RESPONSE PREVENTION FOR USE WITH CHILDREN AND ADOLESCENTS

CBT for adults and youth is actually quite similar formally, provided that adjustments are made to increase developmental sensitivity. For example, the rationale for treatment and instructions for EX/RP are cast in developmentally appropriate language, and response prevention is typically more gradual with youth who may be less inherently motivated to participate in treatment. Importantly, the goals of CBT are identical across the developmental spectrum: teaching the patient to

confront (rather than avoid and ritualize) anxiety-evoking thoughts and situations.

As has typically been the case with all pediatric anxiety and mood disorders, the building of the CBT outcome literature in pediatric OCD began with age-downward extension of the protocols found efficacious with adults, followed by publication of single case studies, case series, and open clinical trials. Collectively, the published uncontrolled evaluations (e.g., Franklin et al. 1998, March et al. 1994, Piacentini et al. 2002, Wever & Rey 1997) vielded remarkably similar and encouraging findings across settings and cultures: At posttreatment, the vast majority of patients were responders, with statistically significant, clinically meaningful, and durable reductions in OCD symptoms. This pilot work set the stage for randomized studies evaluating the efficacy of CBT, the first of which was published in the late 1990s (deHaan et al. 1998); since then a number of other RCTs have followed (Barrett et al. 2004, Bolton & Perrin 2008, Freeman et al. 2008, Pediatr. OCD Treat. Study Team 2004, Storch et al. 2007, Williams et al. 2010), and their collective outcomes further underscore that CBT involving EX/RP is an efficacious treatment for children and adolescents with OCD (Abramowitz et al. 2005, Barrett et al. 2008).

IMPLICATIONS OF THE RESEARCH LITERATURE FOR CLINICAL DECISION-MAKING

The extant literature reviewed above offers guidance for clinicians regarding the likelihood of patients' responsiveness to the various treatments for OCD and thus should be referenced and emphasized during the discussion of treatment alternatives with patients seeking professional assistance. At the same time, findings from studies that have examined the efficacy and effectiveness of treatments are based on aggregated data and thus do not provide certainty for individual outcomes; this point must also be acknowledged openly. In the case of adult OCD, there is greater confidence

Empirically supported treatments: treatments found efficacious in several scientifically credible studies of efficacy

regarding the expected responses to EX/RP and SRI pharmacotherapy, as dozens of studies conducted around the world have contributed to the knowledge base about these treatments. Although the treatment outcome literature has grown substantially in pediatric OCD over the past decade, the number of studies, associated sample sizes, and methodological quality of the studies published to date leave many important questions still unanswered. In the case of both the adult and pediatric literatures, however, the data on prediction of treatment response have generally yielded divergent and sometimes even inconsistent findings. Such investigations have generally been hampered by relatively small sample sizes within specific treatment conditions; these sample size issues may directly flow from the reality that OCD is a relatively low-base-rate disorder, which makes efficient collection of large samples impractical. Accordingly, it may be the case that efforts to identify predictors (factors that are generally associated with differential treatment response) and moderators (factors that are associated with differential response to specific treatments) will only advance if databases from sites conducting similar treatments on similar samples can be collapsed, as the current generation of treatment trials has typically been powered to examine treatment outcome but not prediction or moderation. Such an undertaking would require standardization of assessment batteries, data collection methods, and, to the extent possible, treatment delivery, and would necessitate cooperative efforts previously unattempted in our field. However, these difficult steps may well be necessary if we are ever to improve our precision in answering the most fundamental question still to be answered, which is which treatments will work best for which patients with which characteristics? Child psychiatry has already made some positive steps in this direction (Child & Adolescent Psychiatry Trials Network, CAPTN; Shapiro et al. 2009), though such efforts have not been attempted in OCD specifically as yet.

WHAT DO WE STILL NEED TO KNOW?

Studies examining the relative versus combined efficacy of EX/RP and medications thus far have failed to clarify which patients actually need both treatments, nor have they shed sufficient light on the issue of optimal treatment sequencing for initial treatments. More research is also needed on the issue of managing partial and nonresponse to the available treatments: Only one such study in adults has been published (Simpson et al. 2008), with findings anticipated soon from a recently completed multicenter RCT in pediatric partial responders to SRI (Freeman et al. 2009). The relative efficacy of augmentation with CBT versus with an atypical neuroleptic is currently being examined in a multicenter trial (E. Foa & B. Simpson, principal investigators), which will provide patients and providers with more definitive information about the risk to benefit ratio of each approach. The effect of OCD subtype on treatment outcome also needs to be examined in larger studies, as insufficient sample sizes and method variance across studies have resulted in inconsistent findings regarding subtype by treatment effects. The time is also ripe for developing treatment innovations to target specific mediators known to affect EX/RP outcomes, such as family dysfunction and certain comorbidity patterns. Recent findings on the efficacy of Acceptance and Commitment Therapy for OCD (Twohig et al. 2010) raise an even broader question about the mechanism that underlies effective treatment for OCD, in that a weekly treatment founded on the framework of relational frame theory that did not include any in-session exposure yielded substantial and clinically significant changes in OCD symptoms that were clearly superior to what was achieved with a psychosocial control condition (relaxation). No issue facing the field, however, is as daunting and important as the dissemination crisis, since failure to improve access to care is a threat to the relevance of all of the psychological treatments of established efficacy for OCD.

SUMMARY POINTS

- 1. The first-line treatment of choice for OCD is cognitive-behavioral therapy (CBT) incorporating exposure and response prevention (EX/RP).
- 2. Serotonin reuptake inhibitors (SRIs) have also proven efficacious, though partial response appears to be the norm.
- 3. In adults, CBT has been shown to be at least as effective as pharmacotherapy; data from children support the same conclusion.
- 4. Different treatment strategies may be indicated for adult patients with severe depression or child/adolescent patients with PANDAS or an externalizing disorder.
- 5. Developmental adaptations to EX/RP are needed, but EX/RP appears to be efficacious with younger patients as well as with adults.
- 6. Cognitive therapies also appear to be efficacious for OCD in adults; less information is available about their applicability to children and adolescents, though preliminary evidence suggests that they may also be effective with younger samples.
- 7. Future research is required to determine whether CBT and pharmacotherapy can be used to augment each other when monotherapy proves ineffective or partially effective.
- 8. Dissemination of empirically supported psychotherapies such as EX/RP is a critically important next step for the field.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

LITERATURE CITED

Abramowitz JS, Foa EB, Franklin ME. 2003. Exposure and ritual prevention for obsessive-compulsive disorder: effects of intensive versus twice-weekly sessions. 7. Consult. Clin. Psychol. 71:394–99

Abramowitz JS, Franklin ME, Street GP, Kozak MJ, Foa EB. 2000. Effects of comorbid depression on response to treatment for obsessive-compulsive disorder. *Behav. Ther.* 31:517–28

Abramowitz JS, Whiteside SP, Deacon BJ. 2005. The effectiveness of treatment for pediatric obsessive-compulsive disorder: a meta-analysis. *Behav. Ther.* 36:55–63

Am. Psychiatr. Assoc. 1980. Diagnostic and Statistical Manual of Mental Disorders. Washington, DC: Am. Psychiatr. Assoc. 3rd ed.

Am. Psychiatr. Assoc. 2000. Diagnostic and Statistical Manual of Mental Disorders. Washington, DC: Am. Psychiatr. Assoc. 4th ed., text rev.

Barrett PM, Farrell L, Pina AA, Peris TS, Piacentini J. 2008. Evidence-based psychological treatments for child and adolescent obsessive-compulsive disorder. J. Clin. Child Adolesc. Psychol. 37:131–55

Barrett PM, Healy-Farrell L, March JS. 2004. Cognitive-behavioral family treatment of childhood obsessive-compulsive disorder: a controlled trial. 7. Am. Acad. Child Adolesc. Psychiatry 43:46–62

Bolton D, Perrin S. 2008. Evaluation of exposure with response-prevention for obsessive compulsive disorder in childhood and adolescence. J. Behav. Ther. Exp. Psychiatry 39:11–22

Cottraux J, Note I, Yao SN. 2001. A randomized controlled trial of cognitive therapy versus intensive behavior therapy in obsessive compulsive disorder. *Psychother. Psychosom.* 70:288–97

Crits-Christoph P, Wilson GT, Hollon SD. 2005. Empirically supported psychotherapies: comment on Westen, Novotny, and Thompson-Brenner (2004). Psychol. Bull. 131:412–17

- Crye J, Laskey B, Cartwright-Hatton S. 2010. Non-clinical obsessions in a young adolescent population: frequency and association with metacognitive variables. *Psychol. Psychother*. 83:15–26
- DeAraujo LA, Ito LM, Marks IM, Deale A. 1995. Does imaginal exposure to the consequences of not ritualising enhance live exposure for OCD? A controlled study: I. Main outcome. *Br. 7. Psychiatry* 167:65–70
- de Haan E, Hoogduin KA, Buitelaar JK, Keijsers GP. 1998. Behavior therapy versus clomipramine for the treatment of obsessive-compulsive disorder in children and adolescents. J. Am. Acad. Child Adolesc. Psychiatry 37:1022–29
- Denys D, de Geus F, van Megen HJGM, Westenberg HGM. 2004. A double-blind, randomized, placebocontrolled trial of quetiapine addition in patients with obsessive-compulsive disorder refractory to serotonin reuptake inhibitors. *J. Clin. Psychiatry* 65:1040–48
- DeVeaugh-Geiss J, Landau P, Katz R. 1989. Treatment of OCD with clorimipramine. *Psychiatry Ann.* 19:97–101
- DeVeaugh-Geiss J, Moroz G, Biederman J, Cantwell D, Fontaine R, et al. 1992. Clomipramine hydrochloride in childhood and adolescent obsessive-compulsive disorder—a multicenter trial. J. Am. Acad. Child Adolesc. Psychiatry 31:45–49
- Dollard J, Miller N. 1950. Personality and Psychotherapy: An Analysis in Terms of Learning, Thinking, and Culture. New York: McGraw-Hill
- Dougherty DD, Rauch SL, Jenike MA. 2002. Pharmacological treatments for obsessive compulsive disorder. In *A Guide to Treatments That Work*, ed. PE Nathan, JM Gordon, pp. 387–410. New York: Oxford Univ. Press. 2nd ed.
- Emmelkamp PMG, de Haan E, Hoogduin CAL. 1990. Marital adjustment and obsessive-compulsive disorder. Br. J. Psychiatry 156:55–60
- Emmelkamp PMG, Visser S, Hoekstra RJ. 1988. Cognitive therapy versus exposure in vivo in the treatment of obsessive-compulsives. *Cogn. Ther. Res.* 12:103–14
- Flament M, Koby E, Rapoport JL, Berg CJ, Zahn T, et al. 1990. Childhood obsessive compulsive disorder: a prospective follow-up study. *J. Child Psychol. Psychiatr. Allied Discipl.* 31:363–80
- Foa EB. 2010. CBT augmentation of SRI treatment of OCD: results of follow-up. In *How do treatments for anxiety disorders benefit patients in the long run?* Symposium presented at 30th annu. Meet. Anxiety Disord. Assoc. Am., Baltimore, MD
- Foa EB, Franklin ME, Moser J. 2002. Context in the clinic: How well do CBT and medications work in combination? *Biol. Psychiatry* 51:989–97
- Foa EB, Kozak MJ. 1986. Emotional processing of fear: exposure to corrective information. *Psychol. Bull.* 99:20–35
- Foa EB, Kozak MJ, Goodman WK, Hollander E, Jenike MA, Rasumussen SA. 1995. DSM-IV field trial: obsessive-compulsive disorder. *Am. J. Psychiatry* 152:90–96
- Foa EB, Liebowitz MR, Kozak MJ, Davies SO, Campeas RE, et al. 2005. Treatment of obsessive compulsive disorder by exposure and ritual prevention, clomipramine, and their combination: a randomized, placebo-controlled trial. *Am. J. Psychiatry* 162:151–61
- Foa EB, Steketee G, Grayson JB, Turner RM, Latimer P. 1984. Deliberate exposure and blocking of obsessive-compulsive rituals: immediate and long-term effects. *Behav. Ther.* 15:450–72
- Foa EB, Steketee G, Turner RM, Fischer SC. 1980. Effects of imaginal exposure to feared disasters in obsessivecompulsive checkers. Behav. Res. Ther. 18:449–55
- Franklin ME, Abramowitz JS, Bux DA, Zoellner LA, Feeny NC. 2002. Cognitive-behavioral therapy with and without medication in the treatment of obsessive-compulsive disorder. *Prof. Psychol. Res. Pract.* 33:162–68
- Franklin ME, Abramowitz JS, Kozak MJ, Levitt J, Foa EB. 2000. Effectiveness of exposure and ritual prevention for obsessive compulsive disorder: randomized compared with non-randomized samples. J. Consult. Clin. Psychol. 68:594–602
- Franklin ME, Kozak MJ, Cashman L, Coles M, Rheingold A, et al. 1998. Cognitive behavioral treatment of pediatric obsessive compulsive disorder: an open clinical trial. J. Am. Acad. Child Adolesc. Psychiatry 37:412–19
- Freeman JB, Choate-Summers ML, Garcia AM, Moore PS, Sapyta J, et al. 2009. The Pediatric Obsessive-Compulsive Disorder Treatment Study II: rationale, design and methods. *Child Adolesc. Psychiatry Ment. Health* 3:4

- Presents a cognitive behavioral account of the persistence of fear and the conditions necessary to promote habituation to feared stimuli.
- The first randomized controlled trial to examine the relative and combined efficacy of EX/RP and an SRI using an unambiguous study design.

- Freeman JB, Garcia AM, Coyne L, Ale C, Przeworski A, et al. 2008. Early childhood OCD: preliminary findings from a family-based cognitive-behavioral approach. J. Am. Acad. Child Adolesc. Psychiatry 47:593– 602
- Geller DA, Hoog SL, Heiligenstein JH, Ricardi RK, Tamura R, et al. 2001. Fluoxetine treatment for obsessive-compulsive disorder in children and adolescents: a placebo-controlled clinical trial. J. Am. Acad. Child Adolesc. Psychiatry 40:773–79
- Greist JH, Choinard G, DuBoff E, Halaris A, Kim S, et al. 1995. Double-blind parallel comparison of three dosages of sertraline and placebo in outpatients with obsessive-compulsive disorder. Arch. Gen. Psychiatry 52:289–95
- Hanna GL 1995. Demographic and clinical features of obsessive compulsive disorder in children and adolescents. J. Am. Acad. Child Adolesc. Psychiatry 34:19–27
- Hodgson RJ, Rachman S. 1972. The effects of contamination and washing in obsessional patients. *Behav. Res. Ther.* 10:111–17
- Hodgson RJ, Rachman S, Marks IM. 1972. The treatment of chronic obsessive-compulsive neurosis: follow-up and further findings. Behav. Res. Ther. 10:181–89
- Hudson JI, Pope HG, Yurgelun-Todd D, Jonas JM, Frankenburg FL. 1987. A controlled study of anorexia nervosa and obsessive nervosa. Br. 7. Psychiatry 27:57–60
- Kasvikis YG, Tsakiris F, Marks IM, Basoglu M, Noshirvani HF. 1986. Past history of anorexia nervosa in women with obsessive-compulsive disorder. *Int. 7. Eat. Disord.* 5:1069–75
- Kessler RC, Chiu WT, Demler O, Walters EE. 2005. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. Arch. Gen. Psychiatry 62:617–27
- Laessle RG, Kia S, Fichter MM, Wittchen H, Pirke KM. 1987. Major affective disorder in anorexia nervosa and bulimia: a descriptive diagnostic study. *Br. J. Psychiatry* 151:785–89
- March JS, Biederman J, Wolkow R, Safferman A, Mardekian J, et al. 1998. Sertraline in children and adolescents with obsessive-compulsive disorder: a multicenter randomized controlled trial. 7AMA 280:1752–56
- March JS, Frances A, Kahn D, Carpenter D. 1997. Expert consensus guidelines: treatment of obsessive-compulsive disorder. *J. Clin. Psychiatry* 58(Suppl. 4):1–72
- March JS, Mulle K, Herbel B. 1994. Behavioral psychotherapy for children and adolescents with obsessive-compulsive disorder: an open trial of a new protocol-driven treatment package. *J. Am. Acad. Child Adolesc. Psychiatry* 33:333–41
- McDougal CJ, Epperson CN, Pelton GH, Wasylink S, Price LH. 2000. A double-blind, placebo-controlled study of risperidone addition in serotonin reuptake inhibitor-refractory obsessive-compulsive disorder. Arch. Gen. Psychiatry 57:794–801
- McLean PL, Whittal ML, Thordarson DS. 2001. Cognitive versus behavior therapy in the group treatment of obsessive-compulsive disorder. *J. Consult. Clin. Psychol.* 69:205–14
- Mowrer OH. 1939. A stimulus-response analysis of anxiety and its role as a reinforcing agent. *Psychol. Rev.* 46:553–65
- Mowrer OH. 1960. Learning Theory and Behavior. New York: Wiley
- Nakatani E, Mataix-Cols D, Micali N, Turner C, Heyman I. 2009. Outcomes of cognitive behaviour therapy for obsessive compulsive disorder in a clinical setting: a 10-year experience from a specialist OCD service for children and adolescents. *Child Adolesc. Mental Health* 14:133–39
- Natl. Inst. Health Clin. Excell. 2005. Obsessive compulsive disorder: core interventions in the treatment of obsessive-compulsive disorder and body dysmorphic disorder. London: NICE
- Pediatr. OCD Treat. Study Team. 2004. Cognitive-behavioral therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder: the Pediatric OCD Treatment Study randomized controlled trial. JAMA 292:1969–76
- Piacentini J, Bergman RL, Jacobs C, McCracken JT, Kretchman J. 2002. Open trial of cognitive behavior therapy for childhood obsessive-compulsive disorder. *7. Anxiety Disord*. 16:207–19
- Rachman S, DeSilva P. 1978. Abnormal and normal obsessions. Behav. Res. Ther. 16:233-48
- Rasmussen SA, Tsuang MT. 1986. Clinical characteristics and family history in DSM III obsessive-compulsive disorder. Am. J. Psychiatry 143:317–33

State-of-the-art epidemiology study examining OCD phenomenology and comorbidity.

Presents a step-by-step guide to clinical decision-making for adult and pediatric OCD.

The first randomized controlled trial to examine the relative and combined efficacy of EX/RP and an SSRI in children and adolescents.

Presents an influential cognitive model of the etiology of obsessions and compulsions; has influenced the development of cognitive therapies for OCD.

The first randomized controlled trial of Acceptance and Commitment Therapy for adult OCD; notably, no in-session exposure is conducted in the ACT protocol.

- Riddle MA, Reeve EA, Yaryura-Tobias JA, Yang HM, Claghorn JL, et al. 2001. Fluvoxamine for children and adolescents with obsessive-compulsive disorder: a randomized, controlled, multicenter trial. J. Am. Acad. Child Adolesc. Psychiatry 40:222–29
- Roper G, Rachman S. 1976. Obsessional-compulsive checking: experimental replication and development. Behav. Res. Ther. 14:25–32
- Roper G, Rachman S, Hodgson R. 1973. An experiment on obsessional checking. Behav. Res. Ther. 11:271–77
 Rosa-Alcázar AI, Sánchez-Meca J, Gómez-Conesa A, Marín-Martínez F. 2008. Psychological treatment of obsessive-compulsive disorder: a meta-analysis. Clin. Psychol. Rev. 28:1310–25
- Rothbaum BO, Shahar F. 2000. Behavioral treatment of obsessive-compulsive disorder in a naturalistic setting. Cogn. Behav. Prac. 7:262–70
- Salkovskis PM 1985. Obsessional compulsive problems: a cognitive-behavioral analysis. *Behav. Res. Ther.* 23:571–83
- Shafran R, Clark DM, Fairburn CG, Arntz A, Barlow DH, et al. 2009. Mind the gap: improving the dissemination of CBT. *Behav. Res. Ther.* 47:902–9
- Shapiro M, Silva SG, Compton S, Chrisman A, DeVeaugh-Geiss J, et al. 2009. The Child and Adolescent Psychiatry Trials Network (CAPTN): infrastructure development and lessons learned. Child Adolesc. Psychiatry Ment. Health 3:12
- Simpson HB, Foa EB, Liebowitz MR, Ledley DA, Huppert JD, et al. 2008. A randomized controlled trial of cognitive-behavioral therapy for augmenting pharmacotherapy in obsessive-compulsive disorder. Am. J. Psychiatry 165:621–30
- Simpson HB, Liebowitz MR, Foa EB, Kozak MJ, Schmidt AB, et al. 2004. Post-treatment effects of exposure therapy and clomipramine in obsessive-compulsive disorder. *Depress. Anxiety* 19:225–33
- Steketee GS, Foa EB, Grayson JB. 1982. Recent advances in the treatment of obsessive-compulsives. Arch. Gen. Psychiatry 39:1365–71
- Storch EA, Geffken GR, Merlo LJ, Mann G, Duke D, et al. 2007. Family-based cognitive-behavioral therapy for pediatric obsessive-compulsive disorder: comparison of intensive and weekly approaches. J. Am. Acad. Child Adolesc. Psychiatry 46:469–78
- Storch EA, Merlo LJ, Larson MJ, Geffken GR, Lehmkuhl HD, et al. 2008. Impact of comorbidity on cognitive-behavioral therapy response in pediatric obsessive-compulsive disorder. J. Am. Acad. Child Adolesc. Psychiatry 47:583–92
- Swedo SE, Leonard HL, Garvey M, Mittleman B, Allen AJ, et al. 1998. Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections: clinical description of the first 50 cases. Am. J. Psychiatry 155:264–71
- Swedo SE, Rapoport JL, Leonard HL, Lenane M, Cheslow D. 1989. Obsessive compulsive disorder in children and adolescents: clinical phenomenology of 70 consecutive cases. *Arch. Gen. Psychiatry* 46:335–41
- Tollefson GD, Rampey AH, Potvin JH, Jenike MA. 1994. A multicenter investigation of fixed-dose fluoxetine in the treatment of obsessive-compulsive disorder. *Arch. Gen. Psychiatry* 51:559–67
- Torres AR, Prince MJ, Bebbington PE, Bhugra D, Brugha TS, et al. 2006. Obsessive-compulsive disorder: prevalence, comorbidity, impact, and help-seeking in the British National Psychiatric Morbidity Survey of 2000. *Am. J. Psychiatry* 163:1978–85
- Twohig MP, Hayes SC, Plumb JC, Pruitt LD, Collins AB, et al. 2010. A randomized clinical trial of Acceptance and Commitment Therapy versus progressive relaxation training for obsessive-compulsive disorder. *J. Consult. Clin. Psychol.* 78:705–16
- Valderhaug R, Larsson B, Götestam KG, Piacentini J. 2007. An open clinical trial of cognitive-behaviour therapy in children and adolescents with obsessive-compulsive disorder administered in regular outpatient clinics. Behav. Res. Ther. 45:577–89
- Valleni-Basille LA, Garrison CZ, Jackson KL. 1994. Frequency of obsessive compulsive disorder in a community sample of young adolescents. J. Am. Acad. Child Adolesc. Psychiatry 33:782–91
- van Balkom AJLM, de Haan E, van Oppen P, Spinhoven P, Hoogduin KAL, et al. 1998. Cognitive and behavioral therapies alone versus in combination with fluvoxamine in the treatment of obsessive compulsive disorder. 7. Nerv. Mental Dis. 186:492–99
- Vogel PA, Stiles TC, Götestam KG. 2004. Adding cognitive therapy elements to exposure therapy for obsessive compulsive disorder: a controlled study. Behav. Cogn. Psychother. 32:275–90

- Warren R, Thomas JC. 2001. Cognitive-behavior therapy of obsessive-compulsive disorder in private practice: an effectiveness study. *J. Anxiety Disord.* 15:277–85
- Weisz JR, Weersing VR, Henggeler SW. 2005. Jousting with straw men: comment on Westen, Novotny, and Thompson-Brenner, 2004. Psychol. Bull. 131:418–26
- Westen D, Novotny CM, Thompson-Brenner H. 2004. The empirical status of empirically supported psychotherapies: assumptions, findings, and reporting in controlled clinical trials. *Psychol. Bull.* 130:631–63
- Wever C, Rey JM. 1997. Juvenile obsessive compulsive disorder. Aust. N. Z. J. Psychiatry 31:105-13
- Whittal ML, Robichaud M, Thordarson DS, McLean PD. 2008. Group and individual treatment of obsessive-compulsive disorder using cognitive therapy and exposure plus response prevention: a 2-year follow-up of two randomized trials. J. Consult. Clin. Psychol. 76:1003–14
- Whittal ML, Thordarson DS, McLean PD. 2005. Treatment of obsessive-compulsive disorder: cognitive behavior therapy versus exposure and response prevention. *Behav. Res. Ther.* 43:1559–76
- Whittal ML, Woody SR, McLean PD, Rachman S, Robichaud M. 2010. Treatment of obsessions: a randomized controlled trial. *Behav. Res. Ther.* 48:295–303
- Williams TI, Salkovskis PM, Forrester L, Turner S, White H, et al. 2010. A randomised controlled trial of cognitive behavioural treatment for obsessive compulsive disorder in children and adolescents. Eur. Child Adolesc. Psychiatry 19:449–56
- Zohar J, Judge R, OCD-Paroxetine-Study-Investigators. 1996. Paroxetine versus clomipramine in the treatment of obsessive-compulsive disorder. Br. J. Psychiatry 169:468–74



Volume 7, 2011

Contents

The Origins and Current Status of Behavioral Activation Treatments for Depression	
Sona Dimidjian, Manuel Barrera Jr., Christopher Martell, Ricardo F. Muñoz, and Peter M. Lewinsohn	1
Animal Models of Neuropsychiatric Disorders A.B.P. Fernando and T.W. Robbins	9
Diffusion Imaging, White Matter, and Psychopathology Moriah E. Thomason and Paul M. Thompson	3
Outcome Measures for Practice Jason L. Whipple and Michael J. Lambert	7
Brain Graphs: Graphical Models of the Human Brain Connectome Edward T. Bullmore and Danielle S. Bassett	3
Open, Aware, and Active: Contextual Approaches as an Emerging Trend in the Behavioral and Cognitive Therapies Steven C. Hayes, Matthieu Villatte, Michael Levin, and Mikaela Hildebrandt	1
The Economic Analysis of Prevention in Mental Health Programs Cathrine Mihalopoulos, Theo Vos, Jane Pirkis, and Rob Carter	9
The Nature and Significance of Memory Disturbance in Posttraumatic Stress Disorder Chris R. Brewin 20:	3
Treatment of Obsessive Compulsive Disorder Martin E. Franklin and Edna B. Foa 229	9
Acute Stress Disorder Revisited Etzel Cardeña and Eve Carlson	5
Personality and Depression: Explanatory Models and Review of the Evidence	
Daniel N. Klein, Roman Kotov, and Sara 7. Bufferd	9

Errata

An online log of corrections to Annual Review of Clinical Psychology articles may be found at http://clinpsy.annualreviews.org