SERIES IN ANXIETY AND RELATED DISORDERS

CONCEPTS AND CONTROVERSIES IN OBSESSIVE-COMPULSIVE DISORDER

Edited by
Jonathan S. Abramowitz
and
Arthur C. Houts

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To Stacy, Emily, and Miriam, and in loving memory of
Morris "Grandpop" Abramowitz—J.S.A.

To Mary and Margaret—A.C.H.

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PREFACE

Few syndromes in psychopathology generate as much popular curiosity and clinical exploration as does obsessive-compulsive disorder (OCD). Since the 1970s, research on OCD has increased exponentially. Specific advances include an improved grasp of the heterogeneity of the disorder, identification of putative subtyping schemes, and the development of increasingly sophisticated theoretical models of the etiology and maintenance. Perhaps most importantly, research has led to advances in treatment; and whereas the first line therapies (cognitive-behavior therapy and serotonergic medication) are not entirely effective for every sufferer, they have transformed OCD from an unmanageable lifetime affliction into a treatable problem that need not reduce quality of life.

Despite the aforementioned advances, there have emerged a number of sharp disagreements concerning OCD. Differences have surfaced over phenomenological issues, etiological models, and approaches to treatment, and often occur (but not exclusively) along disciplinary lines between biologically oriented and cognitive-behaviorally oriented authorities. For example, medical approaches posit that abnormal biological processes cause OCD, whereas psychosocial formulations emphasize the role of learning and dysfunctional cognitions. Yet because theoretical conjecture and empirical findings from within each tradition are typically addressed toward distinct and narrow audiences, clinicians, researchers, and students with broad interests are hindered from gaining a clear grasp of the diverse (and sometimes polarized) perspectives.

In our view, scholarly debate and empirical scrutiny of divergent viewpoints is a healthy method by which our understanding of OCD can be enhanced. How do biologically oriented researchers reconcile seemingly parsimonious accounts of obsessions and compulsions that do not appeal to diseased neuroanatomic processes? How do cognitive-behaviorally oriented theorists deal with proposals for animal models of OCD based on shared response to serotonin medication? Unfortunately, owing to the relative insularity of the different scientific communities that contribute to research on OCD, such discussions rarely occur; at least not in published form. Therefore, our aim for this edited book is to subject differing viewpoints on a variety of key conceptual, etiological, and therapeutic issues in the field of OCD to mutual debate. It is our hope that by bringing under one cover this vast literature, the volume will be a unique resource for clinicians, researchers, and students, regardless of theoretical and professional allegiances.

We have chosen to focus on seven topics that represent sources of disagreement among OCD experts. These topics are organized into three sections. The first section, *Phenomenology*, covers the issues of OCD symptom subtypes, animal models, and explores the possibility of a spectrum of obsessive-compulsive disorders. In the second section, *Etiology*, neuropsychiatric and cognitive-behavioral models of OCD are each presented and critiqued. The third section, *Treatment*, includes chapters on the use of cognitive therapy versus exposure and response prevention, the importance of therapist involvement in exposure-based treatment, and whether combining medication and cognitive-behavioral treatment is preferable to monotherapy.

The book was composed as follows: First, noted authorities were asked to produce an empirically grounded position paper on their particular area of expertise. After receiving both chapters on a particular issue, the manuscripts were given to the author of the opposing viewpoint for a brief response. We purposely limited the scope of our editing in order to uphold the authors' intended points; and authors were not allowed to amend their original manuscripts.

We are very pleased with this book and what it represents. The world's experts on the nature and treatment of OCD debate and provide a clear and firm statement of their positions. Readers will become aware of the finer points of many arguments and enjoy a revealing look into each author's reaction to an opposing point of view. In some cases, the authors have discovered overlaps in ostensibly diverse positions; with some opening a critical eye toward their own point of view. In other cases, authors have used their rejoinder as an opportunity to reiterate differences and further refine their own arguments.

We are fortunate to be standing at the dawn of a new century when we can look back and forward with hope. In looking back, we can hope that the days are gone for good when individuals with OCD endured years of psychoanalytic treatment with little improvement and provided endless intellectual fodder for speculative theories that were ill-formed and unhinged from both behavior and physiology. Today, we can see that there are treatments that are useful and oftentimes highly effective. We can also see that there is energetic disagreement among experts in the OCD research communities, and it is the sort of disagreement that can lead to productive competition, fruitful debate, and more refined care of patients. In looking forward, we can hope that investigators with differing backgrounds and traditions of research will stay engaged with one another as they pursue their own lights so that at the close of the next century we will have an understanding of OCD that integrates the best methods of neurochemistry and neurophysiology with the best methods of behavioral science. To be able to explain in a causal way the development and the maintenance and extinction of the vexing and self defeating behaviors of OCD remains the goal, and that is a prize worthy of many a lifetime of work.

Jonathan S. Abramowitz, Rochester, Minnesota, USA
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CONTENTS

Part I

PHENOMENOLOGY

Chapter 1

SYMPTOM DIMENSIONS IN OCD: DEVELOPMENTAL AND EVOLUTIONARY PERSPECTIVES

James F. Leckman, David Mataix-Cols, and Maria Conceição do Rosario-Campos

The extraordinary intricacy of all the factors to be taken into account leaves only one way to presenting them open to us. We must first select one and then another point of view, and follow it up through the material as long as the application of it seems to yield results.

—Sigmund Freud, 1915

The idea of a disease-entity is not an objective to be reached, but our most fruitful point of orientation.

—Karl Jaspers, 1923

At the present time, in the absence of definitive etiological markers of vulnerability for obsessive-compulsive disorder (OCD), obsessive-compulsive (OC) symptom dimensions appear to offer a fruitful point of orientation. The complex clinical presentation of OCD can be summarized using a few consistent and temporally stable symptom dimensions. These can be understood as a spectrum of potentially overlapping vulnerabilities that are likely to be continuous with "normal" worries and extend beyond the traditional nosological boundaries of OCD. Although the dimensional structure of OC symptoms is still imperfect, this quantitative approach to phenotypic traits has the potential to advance our understanding of OCD and may aid in the identification of more robust endophenotypes. Preliminary data suggest that these dimensional phenotypes may be useful in studies of the natural history, genetics, neurobiology, and treatment outcome of OCD. A dimensional approach also appears to be congruent with evolutionary and developmental perspectives on OCD. This view point posits that each symptom dimension reflects the dysregulation of highly conserved complex and partially overlapping neural systems that serve to detect, appraise, and respond to potential threats. A dimensional approach is also not mutually exclusive of other methods to parse the larger spectrum of disorders related to OCD. Indeed, the combined use of categorical subtypes and dimensional assessments are likely to offer

the greatest promise. Thus far, age-of-onset of OC symptoms and the individual's "tic-related" status appear to be particularly useful categorical distinctions. Finally, existing assessment methods are inadequate and new dimensional scales are needed to take full advantage of a dimensional approach in clinical and population-based studies.

INTRODUCTION

Obsessive-compulsive disorder is a chronic and potentially disabling condition affecting from 1% to 3% of the general population. Patients with OCD describe the sudden intrusion into consciousness of unwanted thoughts or unpleasant images. Frequently these obsessions are accompanied by a profound sense of dread and the urge to complete specific compulsions. Compulsions are repetitive acts, typically performed a certain number of times or according to certain private rules, that the individual is driven to complete, even though these acts are perceived as excessive.

The *DSM-IV* (American Psychiatric Association [APA], 1994) and other standard diagnostic classifications such as ICD-10 (World Health Organization [WHO], 1992) regard OCD as a unitary nosological entity. While this parsimony has a certain formal appeal, it is misleading. The symptoms used to define OCD are heterogeneous and include various intrusive thoughts and preoccupations, rituals, and compulsions. Two individuals with OCD may have totally different and non-overlapping symptom patterns.

From as far back as the earliest descriptions of OCD, investigators have attempted to dissect the phenotype into homogeneous subtypes. For example, Falret made the distinction between "Folie du doute" (madness of doubt) and "Délire du toucher" (delusion of touch) in 1869 (Hantouche & Lancrenon, 1996). Most commonly investigators have distinguished "washers" from "checkers" (Horesh, Dolberg, Kirschenbaum-Aviner, & Kotler, 1997; Khanna & Mukherjee, 1992; Matsunaga et al., 2001; Rachman & Hodgson, 1980). With a few notable exceptions, these attempts had limited success in relating the identified subtypes to biological markers, genetic factors or treatment response in part because pure subtypes of patients are rare, and the recruitment of sufficient sample sizes of each subtype is difficult and highly impractical.

The following review considers an alternative approach to OC symptoms that aims to identify valid quantitative dimensions for use in genetic, neurobiological, and treatment outcome studies. The review then proceeds to examine the potential value of a dimensional approach from developmental and evolutionary perspectives. The chapter closes with a call for the development of state-of-the-art assessment methods.

CHALLENGES TO THE CONCEPT OF OCD AS A UNITARY DIAGNOSTIC ENTITY

In addition to the clinical diversity seen in OCD, genetic and treatment studies also support the view that OCD is a heterogeneous disorder. The influence of genetic factors has been suggested from the earliest descriptive accounts of OCD. Data from

twin and family aggregation studies provide limited support for the vertical transmission of genetic vulnerability factors within some families (Pauls & Alsobrook, 1999). However, inspection of the available pedigrees suggests that OCD is likely genetically complex and heterogeneous. Some cases are familial and related to tic disorders, some cases are familial and unrelated to tics, while in other cases there is no family history of either OCD or tics. Recent segregation analyses also support the view that OCD is genetically heterogeneous (Cavallini, Pasquale, Bellodi, & Smeraldi, 1999).

With regard to treatment, evidence from the past 20 years indicates that OCD is preferentially responsive to pharmacotherapy with potent serotonin reuptake inhibitors (SRIs) and to specific forms of cognitive-behavior therapy. Despite these advances, a substantial number of patients remain symptomatic or show no improvement with these treatments. Between 40% and 60% of patients are nonresponders to SRIs (Greist, Jefferson, Kobak, Katzelnick, & Serlin, 1995). Even among responders to SRIs, the magnitude of response is variable with few patients becoming asymptomatic. Similarly, while cognitive-behavioral treatment with exposure and response prevention frequently leads to significant and lasting improvement in OCD symptoms, at least 25% of patients fail to respond and many more patients refuse to participate (Foa & Kozak, 1996).

The factors associated with an unfavorable treatment response remain largely unknown. For example, pretreatment symptom severity appears not to be a useful predictor of response (Ackerman, Greenland, Bystritsky, Morgenstern, & Katz, 1994; Steketee, 1993). Preliminary evidence, however, indicates that patients with a comorbid personality disorder appear to be less responsive to SRIs as well as to exposure and response prevention (Cavedini, Erzegovesi, Ronchi, & Bellodi, 1997; Ravizza, Barzega, Bellino, Bogetto, & Maina, 1995). Likewise, comorbid tic disorders are also associated with a poor response to SRI monotherapy (McDougle et al., 1994).

While subtyping OCD cases based on the presence or absence of tics or some other patient characteristic, such as age of onset, may lead to increased biological homogeneity, other quantitative approaches may prove to be of greater value in identifying the relevant genetic vulnerability factors. Such quantitative approaches might also aid in the identification of treatment responders. Ideally, such a quantitative vulnerability factor would be readily measurable and could serve as an "endophenotype," one that is functionally intermediate between a specific vulnerability gene or pathway for treatment response and specific phenotypic features.

In the absence of such neuropsychological, neurophysiological, or neurochemical measures, another potentially valuable approach concerns the identification of component aspects of the clinical phenotype itself. A similar approach has proven useful in the study of dyslexia in which component features of this learning disability appear to be associated with specific genetic loci that segregate independently of one another (Grigorenko, Wood, Meyer, & Pauls, 2000). If OCD is like dyslexia, then it may be useful to consider the possibility that the symptoms of OCD can be decomposed into several dimensions that are themselves continuous within the population. If true, the use of these quantitative phenotypes may provide superior power and efficiency of parameter estimation within linkage analyses as well as potentially important predictors of treatment response.

A WORD ABOUT AVAILABLE RATING INSTRUMENTS

In this review, we have only included studies that used comprehensive and nonbiased instruments to ascertain OC symptoms, such as the Yale-Brown Obsessive-Compulsive Scale Symptom Checklist (Y-BOCS-SC; Goodman et al., 1989) and the Obsessive-Compulsive Inventory (OCI; Foa et al., 2002). The Y-BOCS-SC lists more than 50 examples of obsessions and compulsions, organized under 13 major categories plus two categories of miscellaneous obsessions and compulsions, covering the vast majority of OC symptoms (Goodman et al., 1989). Other frequently used instruments such as the Maudsley Obsessive-Compulsive Inventory (Rachman & Hodgson, 1977) and the Padua Inventory (Sanavio & Vidotto, 1988) were excluded because their items are heavily biased toward specific symptoms (eg, checking, cleaning) or that omit key symptoms (eg, hoarding, symmetry). More problematic is the use of composite severity ratings based on all of the patient's obsessions and compulsions (Fals-Stewart, 1992; Kim, Dysken, Pheley, & Hoover, 1992; McKay, Danyko, Neziroglu, & Yaryura-Tobias, 1995). We also take note of the limitations of the Y-BOCS-SC, particularly the fact that some of the symptom categories are too broadly conceived, and as such are heterogeneous and inherently ambiguous. For example, any given checking compulsion could be related to any one of a number of symptom dimensions, yet any checking-related compulsion can only be rated within a single symptom domain. Similar critiques can only be made for the wide range of mental rituals and avoidance-related behaviors.

QUANTITATIVE OCD PHENOTYPES: INITIAL STUDIES

The first study to factor-analyze the Y-BOCS-SC was that of Baer (1994). He factor-analyzed the 13 major categories of the Y-BOCS-SC in a sample of 107 patients and identified three factors, accounting for 48% of the variance; these were named "symmetry/hoarding," "contamination/cleaning," and "pure obsessions." Following Baer's seminal work, Leckman et al. (1997) evaluated the 13 a priori Y-BOCS-SC categories in two large groups of OCD patients totaling over 300 cases (Leckman, Walker, Goodman, Pauls, & Cohen, 1994; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995). A principal components factor analysis was performed with a Varimax rotation separately using the symptom information from each group of OCD patients. In an effort to identify valid "traits," they included any OCD symptoms that patients "ever" experienced over the course of their lifetimes, as opposed to limiting these analyses to current symptoms. Remarkably, both data sets yielded nearly identical results. Four factors were identified that in total accounted for more than 60% of the variance in each data set (Leckman et al., 1997). The first factor included obsessions about aggressive behavior toward self or others, sexual obsessions and obsessions related to moral rightness or religion, as well as related checking compulsions. This factor accounted for 30.1% of the variance. A second factor accounted for 13.8 % of the variance and included obsessions concerning a need for symmetry or exactness, repeating rituals, counting compulsions, and ordering/arranging compulsions. A third factor that accounted for 10.2% of the variance was composed of contamination obsessions and cleaning and washing compulsions. The fourth factor included

hoarding and collecting obsessions and compulsions and accounted for 8.5% of the variance.

Subsequently, Summerfeldt, Richter, Antony, and Swinson (1999) evaluated existing models of OCD symptom structure in a sample of 203 individuals. Using confirmatory factor analyses, they examined four models: a single-factor (ie, OCD as a single dimension), a two-factor (ie, obsessions and compulsions), the three-factor model of Baer (1994), and our four-factor model. Adequate fit was found solely for the four-factor model described in Leckman et al. (1997), specifying aggressive, sexual, and religious obsessions and checking compulsions; symmetry/ordering; contamination/cleaning; and hoarding. However, parameter estimates showed within-factor heterogeneity, as well as overlap between factors, most notably between factors 1 and 3.

At present, there have been at least 11 studies corresponding to 10 large OCD datasets and involving over 2000 patients (Table 1.1; Mataix-Cols, Fullana, Alonso, Menchon, & Vallejo, 2004). While the factorial studies available to date have been fairly consistent, the number of factors has ranged from 3 to 6. Some of the symptom dimensions have been consistently replicated across studies (eg, contamination/washing, symmetry/ordering, hoarding) but the aggressive/checking and sexual/religious dimensions need further study, as it is unclear whether they form a unique factor (Cavallini, Di Bella, Siliprandi, Malchiodi, & Bellodi, 2002; Leckman et al., 1997, 2003; Summerfeldt et al., 1999), or can be broken down into two separate dimensions (Baer, 1994; Foa et al., 2002; Hantouche & Lancrenon, 1996; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002a; Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999; Summerfeldt, Richter, Anthony, & Swinson, 1997; Tek & Ulug, 2001). Similarly, it is unclear how to regard somatic obsessions, as they loaded on the contamination/washing factor in two studies (Baer, 1994; Hantouche & Lancrenon, 1996), on the obsessions/checking factor in three other studies (Cavallini et al., 2001; Leckman et al., 1997, 2003), and just with sexual obsessions in two other studies (Feinstein, Fallon, Petkova, & Liebowitz, 2003; Mataix-Cols et al., 2002a).

TEMPORAL STABILITY OF OCD SYMPTOM DIMENSIONS

Preliminary data are available that support the temporal stability of the OC symptom dimensions, at least in adult patients. Rettew, Swedo, Leonard, Lenane, and Rapoport (1992) assessed the longitudinal course of OC symptoms in 76 children and adolescents with OCD followed over a period of 2–7 years, using the categories of the Y-BOCS-SC. They found that none of the patients maintained the same constellation of symptoms from baseline to follow-up. Nevertheless, these authors acknowledged that these changes could have occurred within (rather than between) symptom dimensions; and this hypothesis was not specifically addressed. In the Leckman et al. (1997) report, two sets of OCD patients were studied. Fourteen subjects participated in both studies. The mean (SD) test–retest interval was 51.2 (11.7) months with a range of 17–61 months. In each case, the family-genetic study data were collected prior to the data for the phenomenological studies. Pearson correlations were in the moderate to strong range for each symptom dimension, ranging from .51 to .75. These preliminary test–retest findings indicate that these symptom dimensions may be relatively stable individual characteristics over time. In a more recent study (Mataix-Cols et al., 2002b),

TABLE 1.1. The structure of obsessions and compulsions as reported in 11 available studies

		Contamination/ washing	Aggressive/ checking	Sexual/ religious	Hoarding	Symmetry/ ordering	% Explained variance
Study	Baer (1994)	X_2	X_3	X_3	X_1	X_1	48
Instrument	Y-BOCS-SC						
Analysis technique	PCA, current symptoms						
N	107 OCD						
Study	Hantouche and Lancrenon (1996)	X_3	X_2	X_2	X_1	X_1	
Instrument	Y-BOCS-SC						
Analysis technique	PCA, current symptoms						
N	615 OCD						
Study	Leckman et al. (1997)	X_3	X_1	X_1	X_4	X_2	62.6
Instrument	Y-BOCS-SC						
Analysis technique	PCA, lifetime symptoms						
N	292 OCD						
Study	Leckman et al. (1997)	X_3	X_1	X_1	X_4	X_2	
Instrument	Y-BOCS-SC						
Analysis technique	PCA, lifetime symptoms						
N	292 OCD						
Study	Summerfeldt et al. (1999)	X_3	X_1	X_1	X_4	X_2	
Instrument	Y-BOCS-SC						
Analysis technique	CFA, current symptoms						
N	203 OCD						
Study	Mataix-Cols et al. (1999)	X_3	X_4	X_5	X_2	X_1	65.6
Instrument	Y-BOCS-SC						
Analysis technique	PCA, current symptoms						
N	354 OCD						
Study	Tek and Ulug (2001)	X_1	X_2	X_4	X_2	$X_{1,2,3}$	65.6
Instrument	Y-BOCS-SC						
Analysis technique	PCA, current symptoms						
N	45 OCD						

Study	Cavallini et al. (2001)	X_1	X_3	X_3	X_2	$X_{4,5}$	58.8
Instrument	Y-BOCS-SC						
Analysis technique	PCA, lifetime symptoms						
N	180 OCD						
Study	Mataix-Cols et al. (2002a)	X_2	X_1	X_1	X_4	$X_{1,3}$	63.5
Instrument	Y-BOCS-SC						
Analysis technique	PCA, current symptoms						
N	153 OCD						
Study	Leckman et al. (2003)	X_3	X_1	X_1	X_4	$X_{2,3}$	
Instrument	Y-BOCS-SC						73.1
Analysis technique	PCA, lifetime symptoms						
N	236 TS + FDFM						
Study	Foa et al. (2002)	X_1	X_2	X_2	X_3	$X_{4,5,6}$	80.8
Instrument	OCI						
Analysis technique	PCA, CFA, current symptoms						
N	215 OCD						
Study	Feinstein et al. (2003)	X_2	X_2	X_4	X_3	X_1	54.2
Instrument	Y-BOCS-SC						
Analysis technique	PCA, current symptoms at item level, excluding miscellaneous						
N	160 OCD						

Note: TS = Tourette's Syndrome; FDFM = first degree family members; Y-BOCS-SC = Yale-Brown obsessive compulsive scale symptom checklist; OCI = obsessive compulsive inventory; PCA = principal components analysis; CFA = confirmatory factor analysis. Numeric subscripts refer to separate factors in descending order based on the amount of variance explained. Symptom categories that share the same numeric subscript where identified as a single dimension in the study. Categories with multiple subscripts were identified as containing more than one dimension.

a large sample of adult patients were repeatedly administered the Y-BOCS-SC over a period of 2 years. For the most part, patients maintained their symptoms across this interval and the most robust predictor of having a particular symptom was having had that symptom in the past. For those symptoms that changed across time, changes typically occurred within rather than between previously identified symptom dimensions, suggesting that the symptoms of adult OCD patients are more stable than it is often assumed. Longitudinal studies following patients from childhood to adulthood are needed to gain a more complete understanding of the natural history of OC symptoms.

HOW USEFUL ARE OC SYMPTOM DIMENSIONS IN SORTING OUT COMORBID DISORDERS AND OC SPECTRUM DISORDERS?

Obsessive-compulsive disorder shares a relatively high comorbidity with a number of other psychiatric conditions, including other anxiety disorders, panic disorder, specific phobias, affective disorders, and tic disorders. Separation anxiety, while an index of healthy attachment in typically developing infants and toddlers, is much more common in children and adolescents with OCD—at a point in development when significant separation anxiety is indicative of disorder rather than positive adjustment (Carter, Pauls, & Leckman, 1995). Some studies suggest that as much as 65% of individuals with OCD also meet diagnostic criteria for depression (Pauls, Leckman, & Cohen, 1994). Obsessive-compulsive symptomatology is also typically part of the clinical presentation of individuals with Tourette syndrome (TS) or other chronic tic disorders (Pauls & Leckman, 1986; Pauls, Raymond, Stevenson, & Leckman, 1991).

In recent years, there has been a growing popularity of the concept of a spectrum of disorders related to OCD (Hollander, 1993; Rasmussen, 1994). While there has been some debate about the breadth of membership and the criteria used for inclusion, certain disorders including chronic tic disorders, body dysmorphic disorder, eating disorders, and trichotillomania are routinely considered to be part of this spectrum (Jenike & Wilhelm, 1998).

How useful are OC symptom dimensions in sorting out comorbid disorders and OC spectrum disorders? Baer (1994) reported that patients with high scores on his symmetry/hoarding factor were more likely to have a comorbid diagnosis of chronic tics and OC personality disorder. Similarly, Leckman et al. (1997) found that patients with high scores on the obsessions/checking and symmetry/ordering factors were more likely to present with tics. Mataix-Cols et al. (1999) found that male but not female OCD patients with chronic tics scored higher than patients without tics on the symmetry/ordering dimension. There also appears to be a clear overlap with some eating disorders. For example, Halmi et al. (2003) reported that approximately 70% of patients with anorexia nervosa had lifetime OC symptoms, especially symmetry and somatic obsessions and ordering and hoarding compulsions.

Mataix-Cols, Baer, Rauch, and Jenike (2000) examined the presence of all *DSM-III-R* Axis II diagnoses and their relation to OC symptom dimensions in a sample of 75 OCD patients. They found that hoarding symptoms were strongly related to the presence and number of all personality disorders, especially from the anxious-fearful

cluster. Similarly, Frost, Steketee, Williams, and Warren (2000) found that hoarding was associated with higher levels of comorbidity (ie, anxiety, depression, and personality disorders), as well as work and social disability, compared to nonhoarding OCD and other anxiety disorders. In another study (Samuels et al., 2002), the presence of hoarding was associated with male gender, earlier age of onset, comorbid social phobia, personality disorders, and pathological grooming conditions (skin picking, nail biting, and trichotillomania). While Samuels et al. (2002) found that hoarding was associated with greater overall illness severity (total Y-BOCS scores) another study did not (Saxena et al., 2002).

Taken together, these studies suggest that a symptom-based dimensional approach may be useful in efforts to integrate previous classification attempts based on age of onset, gender, or presence of comorbid and OC spectrum conditions. A dimensional approach has the advantage of allowing each patient to have scores in one or more symptom dimension. It also permits studies that cut across traditional diagnostic boundaries. This approach may be particularly valuable for genetic studies where it increasingly appears that some vulnerability genes may be shared by more than a single disorder and subthreshold cases are likely to be found in family members.

INITIAL VALIDATION FROM FAMILY GENETIC STUDIES

Like many other psychiatric disorders, twin and family studies suggest that genetic factors play a role in the expression of OCD (Alsobrook & Pauls, 1998). Recent advances in molecular genetics have greatly increased the capacity to localize disease genes on the human genome. These methods are now being applied to complex disorders, including OCD. Although earlier studies have indicated that the vertical transmission of OCD in families is consistent with the effects of a single major autosomal gene (Cavallini et al., 1999; Nestadt et al., 2000; Nicolini, Kuthy, Hernandez, & Velazquez, 1991), it is more likely that there are a number of vulnerability genes involved. One of the major difficulties in the application of these approaches is the likely etiologic heterogeneity of OCD and related phenotypes. Heterogeneity reduces the power of gene-localization methods, such as linkage analysis (Alcais & Abel, 1999; Gu, Province, Todorov, & Rao, 1998; Zhang & Risch, 1996). Etiologic heterogeneity may be reflected in phenotypic variability, thus it would be highly desirable to dissect the syndrome, at the level of the phenotype, into valid quantitative heritable components.

Alsobrook, Leckman, Goodman, Rasmussen, and Pauls (1999) were the first to use OC symptom dimensions in a genetic study. They found that the relatives of OCD probands who had high scores on the obsessions/checking and symmetry/ordering factors were at greater risk for OCD than were relatives of probands who had low scores on those factors.

Subsequently, using data collected by the Tourette Syndrome Association International Consortium for Genetics (TSAICG) Affected Sibling Pair Study, Leckman et al. (2003) selected all available affected TS pairs and their parents for which these OC symptom dimensions (factor scores) could be generated using the four factor algorithm first presented by Leckman et al. (1997). Remarkably, over 50% of the siblings with TS were found to have comorbid OCD and greater than 30% of mothers and 10% of fathers also had a diagnosis of OCD. The factor scores for aggressive, sexual, and

religious obsessions and checking compulsions and symmetry and ordering obsessions and compulsions scores were significantly correlated in sibling pairs concordant for TS. In addition, the mother–child correlations, but not father–child correlations, were significant for these two factors. On the basis of the results of the complex segregation analyses, significant evidence for genetic transmission was obtained for all factors. More recently, a genome scan of the hoarding dimension was completed using the same TSAICG data set (Zhang, Leckman, Tsai, Kidd, & Rosario-Campos, 2002). The analyses were conducted for hoarding as both a dichotomous trait and a quantitative trait. Not all sibling pairs in the sample were concordant for hoarding. Standard linkage analyses were performed using GENEHUNTER and Haseman-Elston methods. In addition, novel analyses with a recursive-partitioning technique were employed. Significant allele sharing was observed for both the dichotomous and the quantitative hoarding phenotypes for markers at 4q34, 5q35.2, and 17q25. The 4q site is in proximity to D4S1625, which was identified by the TSAICG as a region linked to the TS phenotype. A recursive-partitioning analytic technique also examined multiple markers simultaneously. Results suggest joint effects of specific loci on 5q and 4q.

In summary, the use of quantitative traits that are familial may provide a powerful approach to detect the genetic susceptibility loci that contribute to OCD presentations. Thus far, this approach has provided especially promising leads with regard to the hoarding OC phenotype. Next steps include, first, the use of these symptom dimensions in large multigenerational families in order to refine the initial genetic linkage results for the hoarding phenotype. Obviously, if specific loci are identified, this will provide compelling evidence for the validity of this multidimensional approach to OCD. Second, genome scans also need to be conducted using the remaining OC symptom dimensions. Families segregating for TS or early onset OCD may be especially valuable in this enterprise. Given the high mother–child correlations in the Leckman et al. (2003) study, it may also be valuable to examine the linkage results for alleles that are identical by descent from the mother. Third, twin and cross-fostering studies are needed to further evaluate the heritability of these symptom dimensions within the general population. Finally, future genetic studies will also need to examine the relationship between these dimensions and other closely related phenotypes including various eating disorders (Halmi et al., 2003) and body dysmorphic disorder.

HINTS AT VALIDATION FROM IN VIVO NEUROIMAGING STUDIES

Functional neuroimaging studies have the potential to increase our understanding of the neural mechanisms underlying OCD. Taken as a whole, these studies strongly link OC symptoms with activation of the orbitofrontal cortex, with less consistent involvement of anterior cingulate gyrus, lateral frontal and temporal cortices, caudate nucleus, thalamus, amygdala, and insula (Saxena & Rauch, 2000). We would predict that if a dimensional approach is useful, then a significant portion of the individual variation seen in these studies might be accounted for by the unique mix of symptom dimensions seen in any given patient. Initial studies generally support this conclusion. Rauch et al. (1998) completed a study with seven adult OCD subjects who received ^{15}O water during a classic continuous performance task as part of a positron emission tomography study. They found that scores on the obsessions/checking

dimension correlated with increased blood flow to the striatum during the continuous performance task.

Subsequently, Phillips et al. (2000) studied seven OCD subjects with prominent contamination preoccupations and cleaning compulsions and seven OCD subjects with prominent checking compulsions. All patients viewed a range of normally disgusting and washer-relevant (rated as more disgusting by the OCD patients with washing compulsions) stimuli. They found that unlike the nonpatient control subjects and checkers, washers showed a pattern of visual cortical and insular activation, with some activation by washer-relevant pictures in frontal regions including the left inferior frontal gyrus and right fusiform gyrus not the striatum or thalamus.

Shapira et al. (2003) studied eight OCD subjects with prominent contamination preoccupations and cleaning compulsions who viewed a range of threat-inducing and disgust-inducing pictures from the International Affective Picture System during functional magnetic resonance imaging scans. They found that in these OCD patients the disgust-inducing condition was strongly associated with activation in the insula, the parahippocampal region, the inferior frontal gyrus (Broadman area [BA] 47), the caudate nucleus, and the primary sensory cortex.

More recently, Saxena et al. (2004) measured regional cerebral glucose metabolism using [^{18}F]-fluorodeoxyglucose-in 45 adult OCD subjects meeting *DSM-IV* criteria for OCD. They stratified the patients into two groups based on the prominence of their hoarding symptoms. They found that compared to controls, patients with prominent hoarding symptoms had significantly lower glucose metabolism in regions of the cingulate gyrus and higher metabolism in the right dorsolateral prefrontal cortex; and that OCD patients without prominent hoarding symptoms had significantly higher glucose metabolism in left orbitofrontal cortex, bilateral thalamus, left caudate, and left dorsolateral prefrontal cortex. Perhaps most interestingly, hoarding severity was negatively correlated with glucose metabolism in the cingulate gyrus, and positively correlated with metabolism in the right dorsolateral prefrontal cortex.

Finally, in a recent fMRI study, Mataix-Cols et al. (2003) found that when presented with stimuli that could induce OC symptoms, healthy volunteers experienced increases in subjective anxiety. Furthermore, anxiety associated with different symptom dimensions was associated with different patterns of activation in ventral, dorsal prefrontal, and limbic/paralimbic regions. Specifically, provocation of washing-related anxiety was predominantly associated with a pattern of activation within bilateral visual regions, bilateral dorsal and dorsomedial prefrontal regions (medial and superior frontal gyri, dorsolateral prefrontal cortex, precentral gyrus, and dorsal anterior cingulate gyrus), and bilateral ventral prefrontal and limbic regions (ventrolateral prefrontal cortex, orbitofrontal cortex, medial frontal gyrus [BA 10], ventral anterior cingulate, insula, and parahippocampal gyrus). Additional regions included bilateral cerebellum and right putamen bilateral dorsomedial prefrontal cortices, ventral prefrontal cortex/limbic regions, and visual regions. In contrast, checking-related pictures induced activations mainly in dorsal prefrontal and visual regions, with less activation in ventral prefrontal or limbic regions. These findings are similar to those of Phillips et al. (2000) and Shapira et al. (2003). Finally, the provocation of hoarding-related anxiety was associated with activation predominantly ventral prefrontal and limbic regions (orbitofrontal cortex, ventral anterior cingulate cortex, medial/superior frontal gyrus, anterior insula, ventrolateral prefrontal gyrus, parahippocampal gyrus, and amygdala) and visual regions. In contrast to the OCD patients with prominent

hoarding symptoms, there were few dorsal prefrontal activations (middle and superior frontal gyrus, dorsolateral prefrontal cortex, and precentral gyrus). Surprisingly, there were no significant differences in activation within any regions in response to normally aversive and hoarding-related pictures.

Taken together, these studies raise the question of whether the lack of perfect replicability of the findings in previous functional imaging studies of OCD could be accounted for by phenotypic variations among subjects. If these preliminary findings are confirmed, they would suggest that discrete neural systems may mediate the expression of different classes of OC symptoms and that these patterns of activation differ in degree from the activations seen among normal controls exposed to the same stimuli.

PREDICTION OF TREATMENT RESPONSE: PHARMACOTHERAPY

While controlled trials with SRIs have demonstrated a selective efficacy in OCD, up to 40–60% of patients do not have a satisfactory outcome (Hollander et al., 2002). Nonresponse to treatment in OCD is associated with serious social disability. These differences in treatment outcome emphasize the heterogeneity of OCD and the need for identifying predictors of treatment response. While definitive studies have not been undertaken, recent studies have suggested that a symptom-based dimensional approach may prove to be valuable for identifying significant predictors of treatment outcome. For instance, several studies have shown that patients with high scores on the hoarding dimension respond more poorly to SSRIs (Black et al., 1998; Erzegovesi et al., 2001; Mataix-Cols et al., 1999; Saxena et al., 2002; Winsberg, Cassic, & Koran, 1999). In another study, high scores on the sexual/religious obsessions factor (Mataix-Cols et al., 1999) were associated with poorer long-term outcome with SSRIs and behavior therapy (BT) in 66 adult outpatients who were followed up from 1 to 5 years (Alonso et al., 2001). Another study (Erzegovesi et al., 2001) reported that patients with somatic obsessions had poorer insight and responded less well to SSRIs. Other somatic treatments may also help patients with specific symptoms. For instance, one study found that patients with symmetry and unusual somatic obsessions may respond well to monoamine-oxidase inhibitors (Jenike, Baer, Minichiello, Rauch, & Buttolph, 1997). In another study, the presence of symmetry obsessions, ordering compulsions and hoarding rituals predicted better response in refractory cases treated with cingulotomy (Baer et al., 1995).

COMPLIANCE AND RESPONSE TO BEHAVIORAL INTERVENTIONS

The efficacy of BT for OCD has been demonstrated in numerous controlled and meta-analytic studies. However, a significant number of patients still remain unimproved or simply refuse or drop out from this treatment. Some studies have suggested that checking rituals may respond less well to BT (Basoglu, Lax, Kasvikis, & Marks, 1988; Rachman & Hodgson, 1980) but others found no differences in outcome between washers and checkers (Foa & Goldstein, 1978). Foa and Goldstein (1978), however,

reported that washers and checkers responded at different rates to behavioral treatments, with checkers being slower to respond. It is often assumed that patients with "pure" obsessions and mental rituals respond less well to classic behavioral interventions, although data supporting these assumptions are sparse. In a meta-analysis, patients with primary obsessive thoughts without rituals tended to improve less with BT than those who had overt, motor rituals (Christensen, Hadzai-Pavlovic, Andrews & Mattick, 1987). In the Alonso et al. (2001) study, the presence of sexual and/or religious obsessions predicted poorer long-term outcome but, because most patients had both SSRIs and BT, it was not clear from this study whether these symptoms predicted poorer outcome with SRIs, BT, or both.

Patients with hoarding symptoms have been described as having poor compliance with and response to BT (Ball, Baer, & Otto, 1996), but little empirical evidence is available from large patient samples. Using a dimensional approach, Mataix-Cols et al. (2002a) examined 153 OCD outpatients who took part in a randomized controlled trial of BT. Results showed that high scorers on the hoarding dimension were more likely to drop out prematurely from the trial and also tended to improve less than nonhoarding OCD patients. In addition, high scorers on the sexual/religious dimension responded less well to BT. Interestingly, patients with mental rituals did as well as other OCD patients in this study. Therefore, it seems that BT is mostly indicated for patients with contamination/washing, aggressive/checking, and symmetry/ordering symptoms. Perhaps, previous anecdotal reports of unsuccessful BT in patients with hoarding symptoms are due in part to the propensity of such patients to discontinue treatment prematurely.

A DEVELOPMENTAL PERSPECTIVE

Children engage in a significant amount of ritualistic, repetitive, and compulsive-like activity that is part of their normal behavioral repertoire. Clinically, this phenomenon reaches a peak at about 24 months of age (Gesell & Ilg, 1943). Quoting Gesell and Ilg:

> Going to bed is also complicated for the two year old . . . bedtime demands have often grown into an elaborated and rigid structure. There is a coming upstairs ritual, brushing the teeth ritual, getting into bed, pulling down the shades, kissing, and even a specially worded good-night ritual. . . . The ritualism so characteristic of 30-months may weigh heavily on the entire household. The child . . . is likely to know where everything belongs and to insist that everything remain in its place. . . . Chairs must be placed at specific angles and certain pictures must remain on certain tables.

Using a parent-report questionnaire, the Childhood Routines Inventory (CRI), to assess compulsive-like behavior in young children we collected data from 1492 parents with children between the ages of 8 and 72 months of age (Evans et al., 1997). The CRI was found to have a strong internal consistency and a two-factor structure. The first factor accounted for 33% of the variance and included items such as "lines up objects in straight lines or symmetrical patterns," "arranges objects or performs certain behaviors until they seem 'just right,'" and "prefers to have things done in a particular order." As such, its content closely resembles obsessions concerning a need for symmetry or exactness, repeating rituals, counting compulsions, and

ordering/arranging compulsions—the symmetry/ordering dimension of OCD. Evans et al. (1997) found the early emergence of specific behaviors that resemble the symptom dimensions observed in OCD patients. For example, parents reported that their children "arranged objects or performed certain behaviors until they seemed 'just right"' on average, beginning at age 22–25 months. They also reported that their children "lined up objects in straight lines or in symmetrical patterns," on average, beginning at age 24–25 months. Similarly, behaviors resembling those associated with the contamination/washing dimension identified with such items as, "seemed very concerned with dirt or cleanliness," were found to have their mean age of onset from 22 to 24 months. Finally, parents reported that their children on average began to "collect or store objects" (resembling the hoarding dimension) from 25 to 27 months of age. Although direct evidence linking the emergence of these behaviors to the later development of OCD is lacking, investigators have found that aspects of these ritualistic and compulsive-like behaviors are correlated with children's fears and phobias (Evans, Gray, & Leckman, 1999; Zohar & Felz, 2001). Further exploration of the factors that underlie the emergence and resolution of these behaviors in normally developing children may provide valuable insights into the neurobiological substrates and evolutionary origins of these behaviors.

EVOLUTIONARY PERSPECTIVES

The ultimate causes for many neuropsychiatric disorders including OCD are likely built into the genetic and neurobiological mechanisms that underlie highly conserved behavioral and cognitive repertoires (Leckman & Mayes, 1998; Marks & Nesse, 1994). In the case of OCD and its composite dimensions, such an evolutionary perspective seems particularly apt. Indeed, we hypothesize that each of these OC dimensions corresponds to unconscious neural evaluation of specific threats (Table 1.2), so that during the evolution of our species, it is likely that if our forbears had not been acutely attuned to potential external threats posed by other humans, by predators, by the external manifestations of microbial disease, or periods of privation due to drought, natural disasters, or internecine conflict, our species would not have survived.

Specifically, it is possible that during our evolutionary history, there were times of great privation such that hoarding was adaptive and likely to enhance the probability of survival and reproductive success. A similar argument can be made for each of the other dimensions. For example, compulsive checking that items in the home environment were "just right," or ensuring that food and key aspects of the home environment were free of contamination, would have served families well at some points in what Darwin called "the struggle for life."

The possible evolutionary origins of obsessions and compulsions related to fears about harm befalling a close family member are of particular interest as they may reveal something of the normal states of heightened preoccupation that are associated with formation of intimate interpersonal relationships. For example, for expectant parents, the immediate perinatal period involves an altered mental state characterized by excitement, and heightened sensitivity to environmental and emotive cues. The infant becomes an increasingly exclusive focus of thought and action towards the end of pregnancy and the early postpartum period. Cues from the infant before and after birth as well as the infant's proximity, physical appearance, and temperament

Table 1.2. Threat domains, conserved behaviors, and developmental epochs associated with heightened sensitivity and obsessive-compulsive-like behaviors

Threat domain	Focus of concern	Mental state	Behavioral response	Developmental epochs
Harm from aggressive behavior from conspecifics	Well-being of self and close family members	Intrusive images or thoughts that contain feared outcomes of separation or loss; among older children and adults—a heightened sense of responsibility	Physical proximity; checking to ensure the safety of close family members; avoidance of danger	Early childhood—formation of attachment to caregivers; early family life—pregnancy, delivery, and care of young children; threats to family members due to injury or other external threats
Physical security	Immediate home environment	Heightened attention to the placement of specific objects in the environment	Checking to ensure that things look "just right" and are in their expected places; arranging/ordering objects	Early childhood—initial period of exploration of the home environment by infants and toddlers; early family life—pregnancy, delivery, and early childhood; threats to family members due to injury or other external threats
Environmental cleanliness	Personal hygiene; hygiene of family members; cleanliness of the home	Preoccupation with intrusive images or thoughts that contain feared outcomes of being dirty or causing others to be ill; among older children and adults—a heightened sense of responsibility	Washing; checking to ensure cleanliness; avoidance of shared or disgusting items	Early childhood—initial period of selection of items of food and drink by toddlers; early family life—pregnancy, delivery, and care of young children; threats to family members due to injury or other external threats
Privation	Essential resources	Preoccupation with intrusive images or thoughts that contain feared outcomes of privation; a heightened sense of responsibility	Collecting items; checking to ensure the sufficient supplies are available	Latency—initial period of collecting; early family life—pregnancy, delivery, and care of young children

provide a major stimulus for these preoccupations and associated behaviors. Guided by this perspective, we recently completed a prospective longitudinal study of 80 expectant parents using a modified version of the Y-BOCS (Leckman et al., 1999). Consistent with our a priori hypothesis the content of the parents' preoccupations involved anxious intrusive thoughts and harm avoidant behaviors that closely resemble some obsessions seen in OCD patients with aggressive symptoms; namely, worries about aggressive behavior, unintentional or intentional, that would lead to the baby being harmed were commonplace. Consistently, such intrusive thoughts were relieved by the performance of compulsive checking behaviors that the parents may regard as excessive or unnecessary.

At 2 weeks after delivery, mothers of normal infants, on average, reported spending nearly 14 h per day focused exclusively on the infant. The mental content of preoccupations at this time included upsetting intrusive, recurrent thoughts about the possibility of something bad happening to their baby. Less commonly, intrusive thoughts of injuring the child may beset the new mother (or father) and can in turn lead to postpartum OCD or depression or both (Abramowitz, Schwartz, Moore, & Luenzmann, 2003).

Even before the child is born, parents preoccupy themselves with creating a safe, clean, and secure environment for the infant. Major cleaning and renovation projects are commonplace as the human form of nest building unfolds. After birth, unimpeded access and safety are among the parents' uppermost concerns. Safety issues include the cleanliness of the infant and the infant's immediate environment, taking extra care not to drop the infant, as well as protection from potential external threats. This sense of heightened responsibility leads parents to check on the baby frequently, even at times when they know the baby is fine (Leckman et al., 1999).

Viewed from an evolutionary perspective, it seems nearly self-evident that the behavioral repertoires associated with early parenting skills would be subject to intense selective pressure. For one's genes to self-replicate, sexual intimacy must occur and the progeny of such unions must survive. Pregnancy and the early years of an infant's life are fraught with mortal dangers. Indeed, it has only been during the past century that infant mortality rates have fallen from over 100 per 1000 live births in 1900 to about 10 per 1000 in 1984 (Corsini & Viazzo, 1997). Little wonder then that a specific state of heightened sensitivity on the part of new parents would be evolutionarily conserved.

Consistent with the emerging data from brain imaging studies, this evolutionary perspective suggests that each of the OC symptom dimensions is based on overlapping brain-based alarm systems that have the potential to become dysregulated due to genetic vulnerability, adverse environmental change during the course of development (maladaptive learning leading to brain changes), or brain injury. Viewed in this light, the diverse behaviors and mental states encountered in OCD are not in themselves pathological. It is only by the distress they cause, their persistence, and their tendency to occupy time to the exclusion of more normal activities that they become pathological.

LIMITATIONS

While much of the available data concerning a dimensional approach to OCD symptoms are promising, there are many questions yet unresolved. Principal among

these is how best to measure these putative dimensional traits in patients and populations. The patient-based methods have relied on a priori symptom categories derived from the Y-BOCS-SC, while the population-based studies have been based on parent report. Further, is it best to conceptualize these dimensions as measuring individual differences in the degree of obsessive worry and alarm (and related compulsive behaviors), or is there some converse set of "anti-obsessional" or "carelessness" traits that properly belong on these dimensions as well?

Other issues include the accuracy of the four-factor solution. Some investigators have proposed that the factor containing aggressive, sexual, and religious obsessions and related checking compulsions is divisible within two separate domains; aggressive obsessions and related checking compulsions versus obsessions with either sexual or religious content (see Table 1.1). Other problems relate to the method of analysis itself. Principal components analysis is limited in that there is no probability model, it is sensitive to variable scaling, and it depends on the decision rules to retain the factors. As Summerfeldt et al. (1999) have noted, most factorial studies of the Y-BOCS-SC used a priori defined symptom groupings rather than its individual symptoms. Indeed, when individual items have been examined, the results have not always been consistent with the prevailing factor structures. For example, Feinstein et al. (2003) recently reported that some of the symptoms typically found in the contamination and washing dimension loaded on the aggressive/checking dimension while others formed their own unique category. Another challenge concerns miscellaneous obsessions and compulsions. In many instances, these symptoms were not included in the analyses. A further limitation relates to the convergent and divergent validity of the Y-BOCS-SC; in one study, the Y-BOCS-SC factor scores correlated poorly with scores on the Padua Inventory, a self-administered measure of OC symptoms (Mataix-Cols et al., in press).

CONCLUSIONS

Despite the limitations discussed above, a strong case can be made to support the continued use of a dimensional approach to OC symptoms. It is consistent with an emerging theory of OCD, which posits that OC symptoms arise when evolutionarily conserved neural threat detection systems are damaged or become dysregulated. The conceptual framework of this evolutionarily based model provides a powerful framework for understanding disease pathogenesis and should permit the integration of new knowledge from a broad range of scientific disciplines from genetics and neurobiology to the development of safe and effective treatments, perhaps ones specifically tailored to specific dimensions. The quantitative nature of these dimensions should also prove to be another important asset as it will add statistical power and readily allow the inclusion of subthreshold cases across a broad range of studies including population-based studies (Maser & Patterson, 2002).

Aspects of this approach may permit a deeper empathic understanding of our patients. For example, if some forms of OCD bear some relationship to the conserved mental states with highly conserved behavioral repertoires typically encountered in expectant parents, it should be easier for clinicians to have a deeper emotional empathy for the anguish the patient is experiencing as they relate the patient's symptoms to emotional experiences in their own lives.

FUTURE DIRECTIONS

In addition to continuing to explore the heuristic value and utility of a dimensional approach, there are at least two other directions for future research efforts. First, we need to acknowledge that a dimensional approach is not mutually exclusive of other methods to parse the larger spectrum of OC-like disorders. The most promising subtypes have been identified based on clinical characteristics, such as age of onset or comorbid diagnoses, particularly tic disorders (Grados et al., 2001; Leckman et al., 1995; McDougle et al., 1993, 1994; Nestadt et al., 2000; Pauls et al., 1995; Rosario-Campos et al., 2001; Wewetzer et al., 2001). Future studies will need to explore the value of combining these methods. Indeed, as noted above, initial genetic linkage studies offer preliminary support for this approach (Zhang et al., 2002).

Second, it is clear that new clinical rating instruments need to be developed to confirm the dimensional structure of OC symptoms and measure the severity of symptoms within each dimension. Practically, by dividing symptoms by their respective dimensions, it is possible to inquire about symptom types that at present are inherently ambiguous. For example, checking compulsions can be assessed in several domains—checking related to unacceptable sexual, aggressive, and religious obsessional impulses or images versus checking to make sure that surfaces or objects are not contaminated. These new instruments should permit the development of better quantitative traits for genetic analyses (based on lifetime symptoms) as well as more discriminating data for use in clinical trials. For example, a patient with contamination worries and hoarding compulsions might show a consistent and marked benefit for the treatment of their contamination symptoms, but little or no benefit in the treatment of their hoarding.

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Chapter 2

DIMENSIONAL AND SUBTYPE MODELS OF OCD

Steven Taylor

Although obsessive-compulsive disorder (OCD) is recognized in *DSM-IV* as a unitary syndrome (American Psychiatric Association [APA], 2000), clinical investigators have increasingly come to regard it as a heterogeneous condition (eg, Pato, Pato, & Pauls, 2002). Some regard OCD as being composed of sets of dimensions, with each dimension corresponding to a distinct set of mechanisms. A dimension may be defined by an aggregate of causal factors that incrementally influence the risk for a particular set of obsessive-compulsive (OC) symptoms (eg, contamination obsessions and washing compulsions).

A different approach to understanding OCD holds that there are discrete subtypes (categories or taxa) of the disorder. Subtypes are defined on the basis of being, in some way, more homogenous than OCD in general. A subgroup can be defined, for example, by whether or not OCD is associated with tic disorders. Tic-related OCD is a more homogenous collection of symptoms than OCD in general. By identifying homogenous phenotypes, researchers hope to identify discrete sets of mechanisms.

The purpose of this chapter is to consider the merits of dimensional and subtype approaches to understanding OCD, with particular attention to the most widely used or innovative approaches. We will consider the relative advantages and disadvantages of the various approaches, with the goal of identifying the most promising ways of conceptualizing and investigating OCD.

The dimension versus subtype distinction has important implications for theory and research (Strube, 1989). A categorical (subtype) variable implies a different set of causes than a continuous variable. Subtypes presumably arise from a small set of causal factors (eg, the presence versus absence of an agent damaging the brain circuits implicated in OCD). In comparison, dimensional variables are probably the result of a multitude of factors. For example, numerous, additive genetic factors, with each making a small but important contribution to the risk of OCD. Dimensional approaches are consistent with current thinking about the role of genes in psychiatric disorders; investigators are increasingly interested in identifying numerous genes that each make only tiny (eg, 1–2%) contributions to phenotypic variance (Plomin, Defries, Craig, & McGuffin, 2003). Thus, the assumption about whether OCD is dimensional

focuses research efforts differently than does the assumption that OCD is composed of subtypes.

Conceptually, typologies lead us to expect that disorders have an all-or-nothing state, with no intermediaries. That is, either the person has an OCD subtype or does not. Typologies imply that treatments should have a similar effect on the disorder; once the critical mechanism is addressed the disorder should rapidly remit. Change may be difficult to initiate with a class variable, but once initiated should be more complete and dramatic (Strube, 1989). In comparison, dimensional approaches assume both a continuum of disorder severity (ranging from absent to very severe) and a continuum of treatment effectiveness (ranging from weak to very strong interventions).

DIMENSIONAL APPROACHES

Factor Analytic Studies

Dimensional approaches to OCD arose from the observation that OC symptoms vary in severity, ranging from very mild (eg, the so-called "normal" obsessions and compulsions: Rachman & de Silva, 1978) to very severe. Scales measuring OC symptoms were developed to capture this range of severity. Factor analyses of these scales suggest that OC symptoms can be decomposed into a small number of dimensions (eg, Baer, 1994; Goodman et al., 1989; Leckman et al., 1997; Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999; Summerfeldt, Richter, Antony, & Swinson, 1999; Taylor, 1995). Factor solutions have varied to some extent from study to study, depending on the nature of the sample, the scale used to assess OC symptoms, and the factor analytic techniques. Even so, a number of consistencies have emerged, suggesting that OC symptoms can be partitioned into what may eventually emerge as a set of reliable (replicable) dimensions. Currently, one of the best supported factor solutions is that reported by Leckman et al. (1997), which had been replicated in the author's original samples and by Summerfeldt et al. (1999). This solution, which is similar to many other factor analytic solutions, consisted of four dimensions:

- Obsessions (aggressive, sexual, religious, or somatic) and checking compulsions.
- Symmetry obsessions and ordering, counting, and repeating compulsions.
- Contamination obsessions and cleaning compulsions.
- Hoarding obsessions and collecting compulsions.

Factor analytic studies have typically not assessed cognitive compulsions in much detail, so the factor solutions may change to some extent when a broader range of OC symptoms is assessed.

The dimensions identified in factor analytic studies tend to be naturally correlated with one another, although the correlations are typically not large ($r < .50$). Even so, the correlations suggest that many of these factors probably load on a higher-order factor. The assumption underlying factor analysis is that each factor corresponds to a distinct set of mechanisms (Cattell, 1978). The finding that dimensions are often correlated suggests that OCD may arise from a combination of general factors (ie,

those influencing OCD in general, and possibly other disorders), and specific factors (corresponding to a given set of symptoms).

Evaluating Dimensional Models

Factor analytic studies do not *prove* that OCD is dimensional. Factor analysis creates dimensions, just like cluster analysis creates categories. Taxometric statistical procedures (Waller & Meehl, 1998) can be used to determine whether a variable is dimensional or categorical, however, these procedures have yet to be applied to OCD. Accordingly, the question of dimensions versus categories must be addressed by considering the relative strengths and limitation of these approaches.

Dimensional models, such as those identified by factor analysis of OC symptom scales, have the advantage of being consistent with the fact that OC symptoms vary in severity. Longitudinal studies suggest that OC symptoms tend to be stable in adults but not in children (Mataix-Cols et al., 2002; Rettew, Swedo, Leonard, Lenane, & Rapoport, 1992). In adults, changes in symptoms tend to occur within rather than between symptom dimensions; shifts from one dimension to another are rare (Mataix-Cols et al., 2002). In other words, if OCD symptoms change in adults, the changes tend to consist of movement up or down the symptom dimensions. Rettew et al. (1992) similarly suggested that in children, the observed changes had actually occurred within rather than between symptom categories, although their design did not allow them to test this. In summary, the available research is consistent with the idea that the dimensions of OC symptoms tend to be stable over time. Changes tend to be within dimensions, which is what one would expect if discrete sets of mechanisms were being modified over time (eg, with treatment).

The merits or usefulness of dimensional models can be further gauged by whether they have meaningful correlates, such as correlations with other symptoms, biometric variables associated with OCD, or treatment response. A number of such findings have emerged. For example, the extent to which OCD runs in families also varies across the symptom dimensions; aggression, sexual, and symmetry OC symptoms have a familial component, whereas hoarding and contamination symptoms do not (Alsobrook, Leckman, Goodman, Rasmussen, & Pauls, 1999). Scores on a dimension assessing counting and repeating compulsions, but not other OC dimensions, tend to be associated with an insertion/deletion polymorphism in the promoter region of the serotonin transporter gene (5-HTTLPR; Cavallini et al., 2002). Scores on the hoarding dimension are correlated with poor response to selective serotonin reuptake inhibitors and to behavior therapy (Abramowitz, Franklin, Schwartz, & Furr, 2003; Alonso et al., 2001; Black, Monahan, Gable, Blum, Clancy, & Baker, 1998; Mataix-Cols et al., 1999).

Comment

To summarize, dimensional models, in which OC symptoms are regarded as arising from a small number of dimensions, shows promise for understanding OCD. Future research, using taxometric methods (Waller & Meehl, 1998), is needed to investigate whether the dimensions are truly continua, or whether they are better conceptualized as categories. Additional research, using expanded assessments of OC symptoms, is also needed to firmly establish the best-fitting dimensional model.

SUBTYPING APPROACHES

General Approaches to Subtyping

Obsessive-compulsive disorder subtyping research, like *DSM-IV*, is couched in the idea that psychiatric disorders can be usefully partitioned into categories. The categorical approach works best "when all members of a diagnostic class are homogeneous, when there are clear boundaries between classes, and when the different classes are mutually exclusive" (APA, 2000, p. xxxi).

As with the *DSM-IV* approach to defining psychiatric disorders, OCD subtyping efforts have been based, to a greater or lesser extent, on the framework laid out in the classic paper by Robins and Guze (1970). These authors proposed that advances in understanding and treating psychiatric disorders are most likely to occur if we study homogeneous groups.

> "Homogeneous diagnostic grouping provides the soundest base for studies of etiology, pathogenesis, and treatment. The roles of heredity, family interactions, intelligence, education, and sociological factors are most simply, directly, and reliably studied when the group studied is as homogeneous as possible." (p. 984).

To identify and validate such groups, Robins and Guze outlined five phases, which interact with one another so that new findings in any one of the phases may lead to modifications in one or more of the other phases. The entire process is therefore one of continuing self-rectification and increasing refinement leading to more homogeneous diagnostic grouping. The five phases are as follows.

1. *Clinical description*. The clinical description of a proposed diagnostic syndrome (or subtype) may be based on some striking clinical feature, or on a combination of features that are thought to be associated with one another. The clinical description need not simply be based on signs and symptoms; it can include demographic features (eg, age, sex, and ethnicity), age of onset, precipitating factors, and any other descriptive features that can define the clinical picture most precisely.
2. *Laboratory studies*. These include chemical, physiological, radiological (eg, neuroimaging), and anatomical (biopsy and autopsy) findings. Psychological studies (eg, tests of cognitive processing) may also be included. When laboratory tests are consistent with the defined clinical picture, they permit a more refined classification.
3. *Exclusion of other disorders.* Exclusionary criteria (including criteria for discriminating subtypes) are developed on the basis of clinical descriptions and laboratory findings. The criteria should permit exclusion of borderline or doubtful cases so that the index group may be as homogeneous as possible.
4. *Follow-up studies.* These studies can be used to determine whether the diagnostic category or subtype is stable over time. Do patients with one putative OC subtype, for example, tend to switch to another subtype over time? Follow-up studies can also investigate whether members from a putative homogeneous group differ in their course of disorder or treatment response. A group may not be a homogenous disorder if it can be clearly divided into patients with good versus poor prognosis.

> "Marked differences in outcome, such as between complete recovery and chronic illness, suggest that the group is not homogeneous. . . . The same illness may have a variable prognosis, but until we know more about the fundamental nature of the common psychiatric illnesses marked differences in outcome should be regarded as a challenge to the validity of the original diagnosis." (Robins & Guze, 1970, p. 984).

5. *Family studies.* The validity of a proposed type or subtype of psychiatric disorder would be supported by showing that it runs in families, reflecting the effects of genetic or shared environmental factors.

OCD Subtype Models

Researchers interested in identifying OCD types have used some or sometimes many of these five phases. Some studies have focused primarily on clinical descriptions for identifying subtypes, while others have focused mainly on family studies or laboratory tests. Still others have attempted to examine all five phases to validate OC subtypes. As a result of these efforts, various subtyping schemes have been proposed, as reviewed in the following sections.

Subtyping by Clustering OC Symptoms

Cluster analyses of OC symptoms have yielded various cluster schemes, depending, in part, on range of symptoms assessed (eg, Abramowitz et al., 2003; Calamari et al., 1999; Khanna et al., 1990). Abramowitz et al. clustered the broadest sampling of OC symptoms, obtaining a five-cluster solution: Harming, contamination, hoarding, unacceptable thoughts, and symmetry. Poorest response to behavior therapy was found for among patients with hoarding symptoms, compared to other patients with other OC symptoms.

There are two major problems with such symptom-based subtyping schemes. First, subtypes (categories) defined by OC symptoms are unable to account for the fact that, phenotypically, OC symptoms vary along a continuum of severity. A subtype model leads one to expect that a person either falls into a subtype category or does not. The range of symptom severity, including so-called "normal" obsessions and compulsions is inconsistent with this notion. A further problem is that discrete, non-overlapping subtypes of OC symptoms are the exception rather than the rule. This problem was recently noted by Mataix-Cols et al. (2002).

> "The efforts based on categorical classification of patients with different OCD symptom subtypes (ie, washers, checkers, etc) have been relatively fruitless, in part because there are so few monosymptomatic patients; therefore, the recruitment of sufficient numbers of subjects with 'pure' OCD subtypes is impractical because such an approach excludes a majority of patients." (p. 263).

Calamari et al. (1999) acknowledged this problem in their cluster analysis: "The five subgroups were characterized by dominant symptom patterns and significant secondary concerns reflecting the symptom heterogeneity often seen in the clinical presentation of obsessional patients" (p. 113).

Autogenous Versus Reactive Obsessions

Lee and Kwon (2003) provided data and argument to distinguish between two types of obsessions—autogenous and reactive obsessions—which were said to be associated with distinct subtypes of OCD. People with autogenous obsessions are said to perceive the obsessions as ego-dystonic and irrational. The person attempts to expel or suppress the unwanted thoughts from consciousness, and frequently employs covert or superstitious compulsive behaviors (eg, counting, praying, undoing the thought with a more acceptable thought) to control the obsession. People with reactive obsessions, on the other hand, are said to believe the thoughts to be relatively rational and realistic, although they frequently or superficially describe their thoughts as being irrational and absurd to clinicians. People with reactive obsessions devote themselves to coping behaviors for preventing the unwanted possible consequences of the obsessions rather than from expelling the thoughts themselves. Thus, people with reactive obsessions resort to overt compulsive behaviors (eg, washing, checking, arranging, hoarding) for preventing the unwanted possible consequences of the obsessions. The compulsions are maintained by anxiety reduction and by the fact that compulsions block the opportunity for disconfirming the obsession.

Lee and Kwon's innovative subtyping scheme merits further investigation. One question worth investigating is whether the mechanisms underlying each type of obsession are categorical or dimensional. Given that obsessions vary along a range of continua (eg, intensity, duration, believability), a dimensional mechanism might be more appropriate. If so, then Lee and Kwon's formulation might provide a basis for understanding the various dimensions of OC symptoms, as identified in factor analytic studies.

Personality Traits

Researchers have attempted to subtype OCD according to personality traits, such as schizotypal personality features (Fals-Stewart & Lucente, 1993; Sobin et al., 2000). A problem with personality-based subtyping schemes is that they are not directly concerned with OCD; they are more appropriately viewed as subtype models of personality. Personality disorder traits co-occur with all kinds of Axis I disorders. Although personality pathology may be associated with poor treatment outcome for OCD (Fals-Stewart & Lucente, 1993), this tells us little about OCD per se; personality disorders predict poor outcome for OCD and other Axis I disorders (eg, panic disorder: Taylor, 2000).

Age at Onset, Tics, and Family History

Three features, early age at onset of OCD, history of tics, and family history of OCD or tics, have been used collectively or individually to define subtypes of OCD. There is suggestive evidence that OCD has a bimodal age of onset; most cases develop in adolescence or early adulthood, while a subgroup develop the disorder in childhood (Geller et al., 1998; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995). The latter are mostly males, while later-onset OCD is split evenly among genders, or contains more females (Geller et al., 2001a; Leonard et al., 1999).

Childhood-onset OCD is more likely to be comorbid with tic disorders, such as Tourette's disorder (Geller et al., 2001b; Leonard et al., 1992; Pauls et al., 1995). The

research generally suggests that aggressive, sexual, symmetry, and exactness obsessions are more common in OCD with comorbid tics (George, Trimble, & Ring, 1993; Holzer et al., 1994; Leckman, Grice, & Barr, 1995; Leonard et al., 1992, 1999; Miguel et al., 1997; Zohar et al., 1997). Tic-like compulsions (touching, blinking, rubbing, tapping, staring) are more common in OCD patients with comorbid tics (Holzer et al., 1994; Leckman et al., 1995; Miguel et al., 1997).

Childhood OCD, compared to OCD arising later in life, also differs in particular clinical features (eg, Albert, Maina, Ravizza, & Bogetto, 2002; Alsobrook et al., 1999; Geller et al., 2001a, 2001b; Hanna et al., 2002; Rosario-Campos et al., 2001). Although there are some inconsistencies in the literature, it appears that childhood OCD is more likely to be associated with poor insight and comorbid attention-deficit hyperactivity disorder (ADHD). Some differences in the expression of OC symptoms over the lifespan may reflect developmental influences rather than being an indication of subtypes. Insight, for example, may simply reflect the person's level of cognitive development.

There is also evidence that adults with early- versus late-onset OCD differ in patterns of regional cerebral blood flow in the frontal-subcortical regions implicated in OCD (Busatto et al., 2001). These results offer preliminary evidence that brain mechanisms in OCD may differ depending on the age at which the disorder first arises.

People with childhood-onset OCD, compared to those with later-onset OCD, are more likely to have first-degree relatives with OCD or tics (Nestadt et al., 2001, 2002; Pauls et al., 1995; Rosario-Campos et al., 2001). Recent evidence suggests that familial OCD has a lower threshold for precipitating events. That is, life events prior to the onset of OCD appear to be more common and more severe in non-familial OCD (Albert et al., 2002).

In summary, three features, early age at onset, comorbid tics, and family history of OCD or tics, tend to co-occur and may define a particular subtype of OCD. However, the co-occurrence of these features is far from perfect. A number of people with childhood-onset OCD, for example, do not have tics or tic-like compulsions, and do not have a family history of OCD (Pauls et al., 1995). Therefore, the three features do not, strictly speaking, define a clear-cut OC subtype. Bimodality of age at onset may suggest a subtype, although under some circumstances a continuous (dimensional) variable can give rise to a bimodal phenotype (Waller & Meehl, 1998). It is possible that age at onset, occurrence of tics, and familiarity are markers of psychobiological dimension that determines the risk for OCD. At the present time, however, early-onset, tic-related, familial OCD seems to be a good candidate for an OC subtype.

Future research will be facilitated if investigators can identify an empirically defined demarcation point for distinguishing "early-onset" from "late-onset" OCD. Previous research has been inconsistent in this regard. Some investigators define early-onset as less than 10 years old, and late-onset as greater than 17 years old (eg, Rosario-Campos et al., 2001). Others use difference criteria; for example, early-onset as less than 10 years old and late-onset as greater than 12 years old (Busatto et al., 2001). Taxometric methods may prove helpful in identifying the optimal cut-off.

Infectious Diseases

It has long been observed that OC symptoms can arise from brain-injuring agents, such as particular infectious diseases. von Economo (1931), for example, described patients who developed OC as a result of encephalitis (ie, a postencephalitic syndrome). More recently, attention has been directed to the possibility that some forms of OCD,

particularly early onset OCD, may be a result of certain diseases that most commonly strike during childhood.

Swedo et al. (1998) observed that Sydenham's chorea, a well-recognized manifestation of rheumatic fever, is commonly associated with OC symptoms. These investigators also observed that some cases of childhood OCD are rapidly acquired after the child develops a Group A β-hemolytic streptococcal infection (GABHS), which is associated with illnesses such as scarlet fever or streptococcal pharyngitis. Streptococcus-related OC symptoms are thought to be commonly associated with tics, separation anxiety, motoric hyperactivity, and neurological symptoms such as clumsiness and choreiform movements. Swedo et al. (1998) referred to this syndrome as pediatric autoimmune disorder associated with streptococcal infection (PANDAS). This syndrome is defined when all of the following are met (Swedo et al., 1998):

- Presence of OCD or a tic disorder.
- Symptom onset between age of 3 and puberty.
- Episodic course, with abrupt and substantial symptom exacerbations
- Symptom onset and exacerbations are associated temporally with GABHS infection.
- Presence of neurologic abnormalities during symptom exacerbations.

The major distinguishing feature of the PANDAS subgroup is the temporal association between neuropsychiatric symptom exacerbations and GABHS infection, that is, positive (or rising) antistreptococcal antibody titers or a positive throat culture during neuropsychiatric symptom relapses and evidence of GABHS negativity during periods of remission (Swedo, 2002). Note that PANDAS is conceptualized as a poststreptococcal disorder; exacerbations usually occur long after the acute symptoms of the streptococcal infection have gone, not at the initial point of infection.

Many children develop streptococcal infections, yet few develop OCD. Swedo (eg, 2002) proposed that susceptibility to PANDAS is probably due to a combination of genetic, developmental, and immunologic factors. Developmental vulnerabilities are suggested by the high rates of streptococcal infection among grade-school age children. For the PANDAS subgroup, the peak age at onset of OC symptoms is 6–7 years (Swedo et al., 1998). The role of genetic factors is suggested by a family study finding increased rates (compared to controls) of rheumatic fever among the parents and grandparents of PANDAS children (Swedo, 2002). Such children also have increased rates of OCD and tics among family members (Lougee, Perlmutter, Nicolson, Garvey, & Swedo, 2000). Swedo (2002) speculates that "the combination of increased familial rates of OCD/tic disorders and increased rates of rheumatic fever suggests that children in the PANDAS subgroup may have a dual genetic vulnerability—with inherited susceptibility to both OCD/tic disorders and post-streptococcal sequelae" (p. S25).

The proposed pathophysiology of streptococcus-induced OCD is similar to that of Sydenham's chorea. Susceptible people respond to the infection by producing antibodies in a normal fashion. However, these antibodies are thought to cross-react with neuronal tissue and compromise their function, leading to the observed clinical symptoms (ie, an abnormal immune response) (Swedo, 2001). Thus, streptococcal infection is thought to produce OC symptoms by a process of inflammation of the basal ganglia (an autoimmune process), which occludes blood supply to these regions and eventually causes tissue necrosis. This suggests that treatments that reduce the inflammation-inducing antibodies (eg, plasma exchange) would reduce OC

symptoms for children with acute-onset OC symptoms, but probably not for more chronic OCD.

A handful of experimental treatment studies have investigated the role of streptococcal infection in causing some types of OCD. Garvey et al. (1999) attempted to treat PANDAS with prophylactic oral penicillin. Treatment had no impact on OC symptoms or tics. However, it also had no impact on the prevalence of streptococcal infections, and so their study failed to adequately test the hypothesis of streptococcal-induced OC symptoms. Perlmutter et al. (1999) conducted a randomized, placebo-controlled study of two treatments: intravenous immunoglobulin and plasma exchange. Such interventions should reduce or eliminate streptococcal antibodies and thereby eliminate the autoimmune-related inflammation of the basal ganglia. Consistent with this, both therapies produced significant improvements in OC and related symptoms; at one-year follow-up, 14 out of 17 children (82%) were rated as "much" or "very much" improved. Further consistent with the notion of PANDAS, Nicolson et al. (2000) found that plasma exchange was ineffective in reducing OC symptoms in children who did not have evidence of streptococcal infection.

Giedd, Rapoport, Garvey, Perlmutter, & Swedo (2000) performed an MRI study of PANDAS subjects versus controls. The PANDAS group had significantly larger basal ganglia (ie, caudate, putamen, and globus pallidus, but not total brain volume), which is consistent with the presence of localized inflammation. A case study suggested that this was normalized with plasma exchange treatment, which presumably reduced inflammation in these regions (Giedd, Rapoport, Leonard, Richter, & Swedo, 1996).

Immunological findings (eg, assays of antistreptococcal antibodies and autoantibodies) in OCD have yielded mixed results, with some but not all studies supporting a connection between streptococcus and OCD (Murphy, Petitto, Voeller, & Goodman, 2001). Peterson et al. (2000), for example, produced results that were interpreted as evidence that prior reports of an association between antistreptococcal antibodies and either tics or OCD may have been confounded by the presence of ADHD. The authors found that antibody titers were correlated with ADHD but not with tics or OCD. However, streptococcal infections are thought to account for a minority (no more than 10%) of childhood OCD (Trifiletti & Packard, 1999). Therefore, findings such as Peterson et al.'s results are not surprising; only a small proportion of their OCD patients would be expected to have antistreptococcal antibodies.

PANDAS-like syndromes associated with GABHS have been identified in adults (Bodner, Morshed, & Peterson, 2001; Greenberg, Murphy, & Swedo, 1998), although PANDAS is considered a childhood-onset disorder because GABHS infections are more common in childhood (Swedo, 2002).

It is noteworthy that PANDAS resembles the early-onset, tic-related, familial subtype of OCD described in the previous section of this chapter. It is possible that they refer to the same or perhaps highly overlapping subtypes. The abrupt onset and offset of PANDAS is consistent with a categorical rather than dimensional model of OCD; one either has infection-related OCD or one does not. In fact, unlike typical OCD, which presents during the teen or early adult years and is characterized by a gradual onset over months, patients with PANDAS tend to be younger and experience an explosive onset of symptoms, that sometimes could be pinpointed to a particular day (Stephenson, 2002).

Although some clinical investigators have expressed doubts about the value of the concept of PANDAS (Kurlan, 1998), others see it as an important advance

in understanding OCD. March and Vitiello (2001), for example, concluded that "PANDAS will likely yield the first empirical demonstration of an etiopathogenically defined subtype of OCD and tic disorders." (p. 142).

Although PANDAS is a promising OC-related subtype, its importance should not be overestimated. It is thought that poststreptococcal autoimmunity might be responsible for up to 10% of cases of childhood OCD (Trifiletti & Packard, 1999). This leaves the other 90% unaccounted for. Other sorts of infectious diseases might account for another proportion of OCD, although many patients are seemingly in good physical health when the disorder arises.

Comment

There are two broad approaches to categorizing psychopathology. Following Robins and Guze (1970), some psychopathologists—called *splitters*—have sought to define smaller and smaller diagnostic categories. The concept of *neurosis*, for example, has been split into distinct disorders (eg, the *DSM-IV* anxiety disorders), and, in turn, these disorders have been split into smaller units (eg, the various sub-forms of specific phobia are listed in *DSM-IV*). Researchers proposing OCD subtypes have continued this tradition.

A contrasting approach is taken by *lumpers*, who argue for broad diagnostic categories. Tyrer (1985) is perhaps the best known advocate of this approach. Lumpers begin with the observation that disorders such as OCD are commonly comorbid with many other disorders, such as other anxiety disorders and mood disorders (APA, 2000). Comorbidity may be concurrent (disorders present at the same time) or lifetime (disorders may or may not co-occur at a given time). A common diathesis may account for much of the comorbidity among the disorders. Tyrer (1985) and others have argued that the frequent comorbidity among anxiety and mood disorders indicates the presence of a unitary, general neurotic syndrome.

> "Acceptance of the existence of a broad neurotic syndrome does not necessarily deny the existence of separate neurotic disorders. . . . However, such diagnoses can only be retained for those patients who have pure syndromes, maintain their diagnostic appearance, and who do not pass, chameleon-like, through different diagnostic hues depending on the nature of the stresses they encounter." (Tyrer, 1985, p. 687).

A challenge for proponents of OCD subtyping schemes is to demonstrate that splitting OCD into subtypes had advantages over other, broader classifications, such as "unsplit" OCD or the general neurotic syndrome. Researchers and clinicians would be more likely to adopt a given subtyping scheme if it can be shown to have clear advantages over other schemes. The work on PANDAS seems most promising in this regard. Preliminary work suggests that splitting OCD into PANDAS and non-PANDAS types may have important implications for treatment (eg, whether or not to use plasma exchange).

An important research direction is to compare the various subtyping schemes with one another to discern their relative merits. Some schemes may be compatible with one another. Research may eventually show, for example, that an infection-based scheme can be integrated with a scheme in which OCD is subtyped according to age at onset, presence of tics, and familiarity. Obsessive-compulsive disorder arising from various childhood diseases might largely correspond to early-onset, tic-related

OCD. If Swedo and colleagues are correct in assuming a genetic predisposition for developing infection-related OCD, then an early-onset, tic-related subtype would also tend to have a family history of OCD or tic disorders.

CONCLUSIONS AND FUTURE DIRECTIONS

The various dimensional and subtype approaches to understanding OC symptoms have largely developed in isolation of one another. The time is ripe for a new generation of studies to compare these models with one another. The models can be compared on the extent that they help us understand, predict, and treat OC symptoms. The available evidence, although limited in all sorts of ways, suggests that age at onset, tics, familiarity, and presence of particular infections (eg, streptococcal infections) are the most promising variables for subtyping OCD. The least promising approach, in my view, is to subtype OCD according to symptom clusters. This approach has two disadvantages compared to dimensional models. First, subtypes (categories) defined by OC symptoms are unable to account for the fact that, phenotypically, OC symptoms vary along a continuum of severity. A subtype model leads one to expect that a person either falls into a subtype category or does not. The range of symptom severity, including so-called "normal" obsessions and compulsions is inconsistent with this notion. A further problem is that discrete, non-overlapping subtypes of OC symptoms are the exception rather than the rule.

Dimensional models, particularly models consisting of correlated dimensions, are more consistent with the patterns of covariance of OC symptoms; people vary in their severity along each dimension, and since the dimensions are correlated, the person can have more than one type of OC symptom (eg, washing compulsions plus checking rituals).

It remains to be established whether taxometric methods support the dimensional and subtyping models. Some putative subtypes might turn out to be better regarded as dimensions. A subtyping scheme based on infectious diseases is most likely to be truly categorical; barring the possibility of subclinical syndromes, one either has an infection or does not have it. The rapid onset and offset of PANDAS-related symptoms is consistent with this conjecture.

The approaches considered in this chapter have consisted of either subtypes or dimensions. It is possible that other, more complex models may be needed to account for OC phenomena. Hybrid models combining dimensions and categories await investigation. Such models could posit that some forms of OCD are categorical (eg, a PANDAS subtype), while others are dimensional. With greater understanding of the brain circuits, genes, and environmental factors involved in OCD and related phenomena, classification will eventually move from phenotype-based classification (eg, based on symptoms, age at onset, etc) to one based on mechanisms.

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Reply to Taylor:

COMBINED DIMENSIONAL AND CATEGORICAL PERSPECTIVES AS AN INTEGRATIVE APPROACH TO OCD

James F. Leckman, David Mataix-Cols, and Maria Conceição do Rosario-Campos

Steven Taylor's scholarly chapter provides the reader with another critical review of the efforts to refine our understanding of the heterogeneity of OCD. Overall, these attempts can be divided into categorical and dimensional approaches. Dimensional studies regard OCD as being composed of sets of symptom dimensions, with each dimension corresponding to a distinctive set of evolutionarily conserved bio-behavioral mechanisms related to specific forms of threat detection and harm avoidance. On the other hand, categorical methods envision discrete subsets of patients whose phenotypes and etiological origins are more homogenous than OCD in general.

In our view, Taylor's analysis is sound and there is little to dispute. Indeed, our two chapters are largely complementary to one another. But given the opportunity to discuss Taylor's chapter, there are a few points to enlarge upon. First, although we would agree with Taylor that, "The categorical approach works best when all members of a diagnostic class are homogeneous, when there are clear boundaries between classes, and when the different classes are mutually exclusive," we would emphasize that "taxonicity" (the search for useful taxa or subtypes) does not preclude dimensionality. This is a point that is nicely made by Waller and Meehl (1998, p. 9), and we concur. We see heuristic value in subtyping patients according to specific taxa as this adds specificity along with the latent dimensions that in turn "underlie" the manifest dimensions and that contribute to their expression. More specifically, we consider that the combined use of categorical subtypes and dimensional assessments are likely to offer the greatest promise in the near term to explore the genetics, neurobiology, natural history, and treatment response of OCD. Thus far, an early age-of-onset of OC symptoms and the individual's "tic-related" status appear to be particularly useful categorical distinctions. However, since even within these subcategories there is a fair amount of symptomatic heterogeneity, the use of symptom dimensions seems to offer even greater specificity.

Second, we would argue it is highly unlikely that the exclusive use of a categorical system will reflect complexity, subtlety, or range of OCD presentations. Obsessive-compulsive symptoms are remarkably heterogeneous, to the extent that two patients with this diagnosis can display completely different symptom patterns. More importantly for OC symptoms, we see no absolute, qualitative break between "normal" and "abnormal." How best to establish the threshold criteria for "definite" OCD remains a debatable issue. The boundaries that divide normal and abnormal patterns of threat detection and harm avoidance are at times indefinite. This discussion is particularly important for genetics, as well as for the study of normal periods of heightened sensitivity to threat. In the case of genetic studies, family members presenting with OC symptoms below the threshold for a *DSM-IV* diagnosis should also be assessed and included in research studies (Leckman et al., 2003; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995). In contrast, there are periods in life, such as childbirth, when otherwise normal individuals experience marked OC behaviors and mental states, and when a diagnosis of OCD would be inappropriate (Leckman, Mayes, Feldman, Evans, King, & Cohen, 1999).

Other shortcomings of the categorical method mentioned by Taylor include the fact that OC symptoms vary along a continuum of severity, and that patients presenting with discrete, non-overlapping subtypes of OC symptoms are the exception rather than the rule. We agree. We also agree with Taylor's argument that dimensional models are more consistent with the patterns of covariance of OC symptoms, and allow researchers to incorporate subjects with different symptom severity and those presenting with more than one type of OC symptom. Besides, dimensions are not mutually exclusive as each patient can score on one or more symptom dimensions at any one time.

Next, although Taylor defines a dimension as "an aggregate of causal factors that incrementally influence the risk for a particular set of OC symptoms," we would argue that, the use of OC symptom dimensions do not necessarily imply or preclude causality. Rather, these dimensions should be viewed simply as useful quantitative constructs that may or may not be independent of the etiological factors associated with them.

Amongst the shortcomings presented by Taylor to the dimensional perspective, he mentions that "factor analytic studies have typically not assessed cognitive compulsions in much detail". We agree that the lack of such items, such as mental rituals and avoidance behaviors, is a major shortcoming of the OC symptom severity scales currently in use. Other problems with generating factor scores from symptom categories include the fact that some symptoms might be considered inherently ambiguous in categorical studies. For example, checking compulsions could be related to sexual and religious obsessions, aggressive images, or to contamination worries; but in the current assessment schemes there is no way to differentiate this, or to measure the severity of individual dimensions. Taking these methodological shortcomings of the factor-analytic method into consideration, it is important to point out that despite these clear limitations, it is remarkable that the dimensional structure of OC symptoms has been fairly consistent in the 12-factor-analytic studies published so far (Mataix-Cols, Rosario-Campos, & Leckman, submitted). We also view these shortcomings as an impetus to develop truly dimensional symptom severity scales (Rosário-Campos et al., in preparation).

Although Taylor acknowledges that dimensional models show promise for understanding OCD, he emphasizes the need for "taxometric methods to investigate whether the dimensions are truly continua, or whether they are better conceptualized as categories." We agree that factor analytic studies do not prove that OCD is dimensional. However, there are no studies reporting on the taxometrics of OCD, although one study suggests that anxiety disorders are most likely to be dimensional (Hasslam, 2003). We are also not convinced that the taxometric statistical procedures proposed by Meehl (1995) can really be used to answer the question whether OCD is dimensional. In our opinion, additional studies are needed to address this issue properly. They should include a longitudinal follow-up of OCD patients, the development of assessment instruments capable of measuring OC symptoms dimensionally, and research on the onset and course of these behaviors in normal populations across the life span.

In fact, we believe that until we identify definitive etiological markers of vulnerability for OCD, a dimensional approach to OC symptoms should be regarded simply as a useful tool for research studies. As presented in our chapter, and also by Taylor, there is a growing body of evidence from different fields suggesting that these OC symptom dimensions are fairly consistent (Leckman et al., 1997; Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999; Summerfeldt, Richter, Antony, & Swinson, 1999), temporally stable (Mataix-Cols et al., 2002b), and have meaningful correlates with biometric variables (Alsobrook, Leckman, Goodman, Rasmussen, & Pauls, 1999; Cavallini, Di Bella, Siliprandi, Malchiodi, & Bellodi, 2002; Leckman et al., 2003; Mataix-Cols et al., 2003, in press; Zhang et al., 2002) and treatment response (Alonso et al., 2001; Black et al., 1998; Mataix-Cols et al., 1999; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002a; Saxena et al., 2002).

Another interesting point is the fact that some of the categorical sub-typing attempts described by Taylor, that is, the ones based on an early age of onset, presence of tics, and previous streptococcal infections, all have one characteristic in common: a higher probability of having symptoms in the symmetry/ordering/"just-right" dimension. In our opinion, a dimensional perspective would be the most efficient way to look more carefully into these phenotypes, considering that it cuts across the diagnostic boundaries. That is, the more symmetry/ordering/"just-right" symptoms one has, the more likely he is to have tics, early onset, a positive family history for OCD and/or streptococcal infections, independent of diagnosis.

Regarding the discussion of the PANDAS subtype, Taylor states that the abrupt onset or exacerbation of symptoms associated with streptococcal infections in PANDAS patients should be considered as consistent with a categorical model of OCD. Nevertheless, it is important to keep in mind that even if this immunological hypothesis for etiology of OCD were proven true, PANDAS cases would account for less than 10% of OCD patients. Second, when taking into account a more longitudinal follow-up of these patients, we frequently observe a fluctuating course, with marked oscillations in symptom severity.

CONCLUSIONS

The knowledge on OCD has progressed over the past two decades. Phenomenological, neurobiological, genetic, and treatment response studies have provided

evidence for the heterogeneity of this disorder. This heterogeneity reduces the power and obscures the findings from gene-localization methods, neuropsychological tests, neuroimaging techniques, clinical observations, and treatment response trials.

To better understand this clinical and genetic heterogeneity, categorical and dimensional approaches have developed relatively independent of one another. The categorical versus dimensional distinction runs deeply through psychiatric thinking. Categorical and dimensional views of psychopathology each have their advantages and disadvantages. However, to a large extent the belief that we must choose between them is simply a misconception. We see that these approaches are complimentary and believe that the combined use of both categorical and dimensional approaches are likely to offer the greatest promise for a better understanding of the complex picture of OCD.

In summary, much research remains to be done. Considering all the evidence presented both in Taylor's and in our chapter, we conclude that the combined use of symptom dimensions as well as the judicious use of subtypes is most likely to capture the heterogeneity of OCD and help to advance our understanding of the OCD complexity. Future studies should use a dimensional perspective as a way of integrating various classificatory attempts, looking at common and distinct genetic and environmental mechanisms involved in the expression of the various OCD phenotypes.

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Reply to Leckman et al.:

PUTTING THE SYMPTOM DIMENSION MODEL TO THE TEST

Steven Taylor

There is a good deal of agreement in the conclusions drawn in my earlier chapter in this volume and those of Dr Leckman and colleagues in their erudite and creative analyses of the phenotypic (symptom) dimensional approach to OCD. Their developmental/evolutionary perspective on the dimensional approach holds great promise for enriching our understanding of OCD. In addition, I agree with the following of their conclusions: (*a*) obsessive-compulsive symptom dimensions offer fruitful means of understanding the mechanisms of OCD; (*b*) each symptom dimension may represent the damage or dysfunction of complex neural systems; and (*c*) hybrid models proposing a mix of dimensions and categories may be needed to fully understand OCD, where early-onset tic-related OCD are especially likely to represent to categorical disorders.

Leckman and colleagues have drawn data from a variety of sources to support the dimensional model, including factor-analytic studies of symptom covariation, genetic linkage studies, functional neuroimaging research, and studies of the relationship between symptom dimensions and treatment outcome. Leckman et al. rightly observe that many of the findings offer only suggestive evidence for the dimensional model. I agree, the findings are encouraging but far from compelling. Rather than uncritically accepting the dimensional approach as the best way to proceed, we need to be mindful of its current limitations, and we need to subject the dimensional approach to strong empirical tests. That is, tests that place the model at risk of falsification.

The limitations of the empirical basis of the dimensional approach are as follows. Although factor analytic studies of OC symptoms tend to support the four-factor solution described by Leckman and colleagues, there is a fair amount of inconsistency among studies. Similarly, genetic linkage studies are notoriously inconsistent (difficult to replicate), as shown in studies of other disorders, such as major depression and bipolar affective disorder. Neuroimaging studies are based on small samples, thereby making it difficult to disentangle the effects of individual differences from the effects of symptom dimensions in the patterns of brain activation. Individual differences are routinely neglected in neuroimaging studies. Instead, researchers standardize or average their data, thereby neglecting the fact that a given brain function (eg, the brain processes involved in the analysis of a given type of threat) may vary to some

degree from person to person in terms of the neuranatomic structures associated with this function. In other words, the same brain function (or neural responses to a given stimulus) can be supported in different brain regions, varying from person to person. Finally, there have been a number of inconsistencies among the studies linking symptom dimensions to treatment outcome. Leckman and colleagues cite only a portion of these studies; there are other studies that do not support Leckman et al.'s assertions about the prognostic significance of particular OC symptom dimensions (eg, McLean et al., 2001; Taylor et al., 2003).

A further limitation of the dimension approach concerns an obvious but often over-looked question: Why would we expect a classification of OC symptoms to form a basis for identifying the underlying mechanisms. One can draw an analogy with pain. There are all kinds of different forms of pain, differing in location, duration, sensory quality (eg, burning, stinging, gnawing), and intensity. If we were to factor analyze a checklist measuring all kinds of pain, then we would no doubt arrive at a multidimensional model. Would we expect this to reveal the underlying mechanisms of pain? Perhaps to some extent: we might obtain, for instance, a "migraine headache" factor and perhaps an "arthritic conditions" factor. But such a factor analysis would fail to reveal the common mechanisms involved in most (all?) types of pain: the central mechanisms (eg, the gate control mechanism) and the peripheral mechanisms (eg, the slow and fast fibers involves in pain transmission) (eg, Melzack & Wall, 1965). Indeed, if we relied on a simple dimensional (factor-analytic) model of pain, then we tend to focus on the mechanisms responsible for each factor, thereby overlooking the more important peripheral and central mechanisms that are implicated in most types of pain.

The dimensional approach to OCD, as described by Leckman and colleagues, runs the same risk of side tracking us from the important common factors that may be involved in most sorts of OC symptoms. Leckman and colleagues claim that OCD is not a unitary disorder because the symptoms are heterogeneous; one person may have checking compulsions, another may have washing rituals, and a third may have ordering compulsions. The dimensional model suggests that these different symptom presentations are the result of dysregulations in different neural systems. That may be, but this ignores the possibility that all forms of OCD share important common mechanisms. Although the *contents* of OC symptoms are many and varied, they all share the same *form*; they can be reliably classified into a small set of functionally similar types (obsessions, compulsions, fears, and avoidance). Moreover, the dimensions of OC symptoms tend to be correlated with one another. Leckman and colleagues seem to underestimate the importance of this point. The fact that the dimensions are intercorrelated is consistent with the idea that there may be some overarching, unitary mechanism that contributes to OCD in general, regardless of symptom type.

Thus, although the dimensional approach has merits, we should not overlook its limitations. Each dimension may correspond to a distinct set of mechanisms, although further research is needed to firmly establish this conclusion. It is likely that OCD consists of a hierarchy of dimensions; for example, the four factors identified by Leckman and colleagues, which in turn load on one (or more) higher-order factors. A similar hierarchical arrangement has been identified in studies of fears and other forms of anxiety symptoms (eg, Taylor, 1998; Zinbarg & Barlow, 1996). In a series of elegant behavioral-genetic studies (twin), Kendler and colleagues (eg, Kendler, Neale, Kessler, Heath, & Eaves, 1992, Kendler et al., 1995) have identified a hierarchy

of environmental and genetic factors that seem to contribute to a range of anxiety disorders. That is, there appear to be disorder-specific genetic factors (eg, genes specific to agoraphobia), as well as nonspecific factors, which contribute to a number of disorders. Similarly, there appear to be specific and nonspecific environmental factors. Obsessive-compulsive disorder was not investigated in any of these studies, although the findings raise the possibility that the same arrangement may well apply to OC symptoms.

There are a number of ways that the phenotypic dimensional model of OCD could be subjected to strong empirical tests. Leckman and colleagues believe that the most important issue is to develop better measures of the putative dimensions. This issue is important, but I believe it is more important to first test the assumptions underlying the dimensional model. We have many good measures of OC symptoms (eg, the Yale-Brown Obsessive-Compulsive Scale in its various versions, along with a new generation of good self-report measures such as the revised Obsessive-Compulsive Inventory). These instruments are amply sufficient for testing two major assumptions underlying the phenotypic dimensional model. The first is the assumption of dimensionality; taxometric methods can be used to determine whether the OC symptom dimensions are truly dimensional or whether they are categories (for a good illustration of this method, see Ruscio, Ruscio, & Keane, 2002).

The second is the assumption that each phenotypic dimension corresponds to a distinct causal mechanism. Behavioral-genetic studies can provide a strong test of this assumption. That is, multivariate twin methods can be used to compute the genetic correlations (and variances and covariances) among pairs of OC symptoms, and the resulting symptom correlation matrix (or variance–covariance matrix) can be factor analyzed to identify the genetic dimensions underlying OC symptoms. Environmental dimensions can be similarly computed. One can then see whether the genetic and environmental dimensions correspond to the phenotypic dimensions. A good example of this approach can be found in a recent twin study by Livesley, Jang, & Vernon (1998), which was concerned with the dimensions of personality disorders. Those investigators identified four major phenotypic dimensions of personality pathology (eg, emotional dysregulation, compulsivity, antisocial traits). Further analysis revealed that each phenotypic dimension corresponded to a distinct, underlying genetic dimension. In other words, each phenotypic dimension of personality pathology appeared to be the expression of an underlying genetic factor.

There are several other ways that the phenotypic dimensional model could be tested (the fact that the dimensional approach is amenable to multiple risks of refutation is a strength of this model). Given that Leckman and colleagues propose that the OC symptom dimensions reflect evolved mechanisms (that become damaged or dysregulated in OCD), then this suggests that (*a*) the dimensions should be culturally invariant; (*b*) historical analyses should reveal evidence that the dimensions have existed for millennia (this might be tested by a multidisciplinary collaboration of historians with OCD researchers); and (*c*) primates, our near relatives (evolutionarily speaking), should display evidence of similar dimensions (as indexed by observable behavior, such as ordering, arranging, hoarding, and cleaning/grooming activities).

In summary, I am largely in agreement with the conclusions of Leckman and colleagues, and I believe that the dimensional model, combined with Leckman et al.'s developmental/evolutionary conjectures, should enrich the way that we understand the causes of OCD. However, we also need to be mindful of the limitations of the

dimensional approach, and consider how this model might constrain our thinking about OCD (eg, it could side track us from searching for factors that contribute to most or all forms of OCD). In my view, the most fruitful avenue for further research, at least in the near future, is to test the basic assumptions of the dimensional model.

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Chapter 3

ANIMAL MODELS OF OBSESSIVE-COMPULSIVE BEHAVIOR: A NEUROBIOLOGICAL AND ETHOLOGICAL PERSPECTIVE

Nicholas H. Dodman and Louis Shuster

Animal models of obsessive-compulsive disorder (OCD) either may be induced in a laboratory setting or may arise spontaneously in veterinary patients. Both models have their usefulness and may replicate some or a constellation of signs associated with the human disorder. Classical laboratory models of OCD are induced by behavioral or pharmacological means. They include rat pup isolation distress calls, conjunctive behaviors, including polydypsia and increased gnawing, spontaneous alternation, and displacement grooming (Dodman & Olivier, 1996). Spontaneously occurring repetitive behavior problems in domestic animals were originally described as stereotypies before a link with human OCD was suggested. The first inkling of this connection surfaced when Goldberger and Rapoport (1991) described the successful treatment with clomipramine of acral lick dermatitis (ALD) in dogs. Their findings were subsequently confirmed in a more comprehensive publication the following year (Rapoport, Ryland, & Kriete, 1992), which represents a landmark in understanding the parallels that exist between canine ALD and a classical form of human OCD, specifically, hand washing. Both conditions involve extreme, apparently irrational assiduousness regarding personal hygiene and involve excessive repetition of self-cleansing behavior. Both conditions may lead to minor degrees of self-injury; both may affect normal behavioral agendas and social relationships; and both respond similarly to serotonin-enhancing pharmacological strategies. The veterinary community became excited about this diagnosis, which offered a new therapeutic approach to a formerly inexplicable and refractory condition. Before too long, it occurred to some veterinarians to view other repetitive disorders in dogs and other species in the same light. Though other repetitive behavior disorders, such as canine compulsive tail chasing, feline psychogenic alopecia (FPA), avian feather picking, and equine cribbing, appeared compulsive, there remained the difficulty of uniting them within the same biological framework. What did they have in common besides their repetitive nature?

Although compulsive paw licking characteristic of canine ALD was analogous to the compulsive hand washing of some human OCD sufferers, avian feather picking seemed a far cry from it, as did equine cribbing and feline FPA. What became apparent was that each species had an assortment of repetitive behavioral conditions that could fall under an umbrella diagnosis of *compulsive disorder*; that each of these behaviors was phenotypically distinct; and that each was derived from a normal species-typical behavior. For example, horses that graze in the wild for 60–70% of their waking time, sauntering around from place to place between times, tend to develop compulsive disorders derived from normal feeding or locomotor behavior. Cows and pigs, two species known for their oral penchant, tend to develop tonguing or chewing/biting compulsions, respectively. Cats, known for their fastidious grooming rituals, sometimes develop compulsive hair pulling, leading to "psychogenic alopecia," or repetitive lip-licking, causing "lip granuloma." Dogs, naturally inclined toward self-grooming and predation, develop compulsions involving excessive grooming or object/tail chasing compulsions.

What helped in the understanding of the wide variation in phenotype within and between species was the realization that human compulsive disorders involve a wider spectrum of behaviors than was formerly thought (Hollander et al., 1996). Though controversial when first suggested, the existence of obsessive-compulsive spectrum of disorders gradually became more widely accepted, embracing human behaviors that involve repetitive thoughts and actions distinct from more classical forms of OCD. In addition, it appears that OC spectrum conditions, including classical OCD, reflect our own species' primordial behaviors and concerns. The primordial agendas were vital for our species' survival in the past, so the neural frameworks underlying them may have become hard-wired in the interests of our very survival (Duchaine, Cosmides, & Tooby, 2001). Early hominids, as hunters, would have required to pay close attention to personal safety, constantly being on the lookout for obvious physical threats and trying avoid contamination. Exaggerated concerns over personal safety and hygiene now emerge as the classical symptoms of OCD today. As a gatherer species, early humans also had a profound investment in collecting things to them, in the interests of their survival. Such deep-seated drives and desires may sometimes express themselves in excess as compulsive hoarding, compulsive shopping, and kleptomania. Compulsive gambling may have derived from primordial calculated risk-taking—another behavior that presumably had survival value ("Nothing ventured, nothing gained"). Extrapolating from pyromania, lighting fires probably provided survival benefit to those most well endowed with this predilection.

This is not to say that OCDs are normal, for that is clearly not the case, only that OCDs appear to derive from survival-necessary, species-typical behaviors. These behaviors are probably encoded in primitive areas of the brain, such as the limbic system, basal ganglia, and hypothalamus, for access at appropriate times. That these centers might be accessed and regulated by cortical centers that exercise some executive control function is not surprising, since it would be biologically appropriate to access encoded cognitive and behavioral sequences only when necessary. What seems to occur in the various OCDs is that the retrieval process for these behaviors is somehow disrupted, so that the encoded behaviors are inappropriately, and sometimes almost continuously, activated (Heinz, 1999). The concept of stored behavioral units is not new, it was established a half-century ago by European ethologists, but has become somewhat neglected recently. Individual sequences of behavior were known

as fixed-action patterns. In the language of the early pioneers, OCD might represent over-activation or derepression of neural processes encoded as fixed-action patterns.

But what might cause such over-activation or derepression? Looking at and learning from veterinary models, it appears that anxiety, typically resulting from conflict or thwarted biological objectives, is at the root of the matter. Canine and equine compulsions, for example, are far more common in animals that are closely confined and unable to engage in species-typical behaviors. The same is true regarding zoo animals, where compulsive disorders are rife. If spontaneously occurring veterinary models of human compulsive disorders are accepted as valid models, physicians would be well advised to pay more attention to the circumstances under which the compulsive disorders are generated and propagated in their patients with a view to addressing the matters arising.

There seems to be a genetic susceptibility toward developing and expressing compulsive disorders in veterinary patients, as there is in humans (Pato, Schindler, & Pato, 2001). Certain dog breeds, for example, are prone to developing ALD, whereas others are more susceptible to compulsive object/tail chasing (Dodman, Moon-Fanelli, Mertens, Pflueger, & Stein, 1997). Certain cat breeds are more likely to develop feline compulsive disorders, and certain breeds of bird are more likely to develop compulsive feather picking. In humans, OCD is more prevalent in certain families (Nestadt, Samuels, Riddle, Bienvenu, 2000). The same seems to be true in dogs (Dodman et al., 1997). Some lines of bull terriers, for example, are more likely to show compulsive tail chasing than others. In addition, some lines of Siamese cats are more prone to developing the oral compulsion known as wool-sucking/pica, or the grooming compulsion known as FPA (Dodman et al., 1997). In addition, certain lines of horse may be more prone to developing the oral/ingestive compulsive behavior known as cribbing (Bachmann & Stauffacher, 2002).

While the developmental, phenotypic, and genetic similarities between human and animal OCDs are intriguing, the similar response to anti-obsessional medication adds more credibility to the validity of animal models. The almost parallel response to these medications implies remarkably similar neurochemical underpinnings between the two sets of conditions and suggests that similar brain regions may be involved.

The similarities between human OCD and domestic animal analogs may be summarized as follows.

1. Both involve the performance of a natural species-typical behavior gone awry.
2. Both involve elements of anxiety or stress.
3. Both have genetic roots.
4. Both respond to similar pharmacological treatments, along a similar time course, and in like manner.

MECHANISMS OF OCD

Studies of the functional and structural neuropathology of OCD in humans have consistently pointed to abnormal activation of an orbital frontal-basal ganglia-thalamic loop (Cohen, Hollander, & Stein, 1997). While no functional imaging studies have been performed in animal models of OCD, physiological/pharmacological studies in experimental animals have indicated that the basal ganglia play an

important role in the propagation of the type of natural behaviors that are typically expressed as compulsions, including grooming and feeding behavior (Meyer-Luehmann, Thompson, Berridge, Aldridge, 2002; Perier, Tremblay, Feger, & Hirsch, 2002; Perez, Colasante, Tucci, Hernandez, & Rada, 2000; Nishino, Hattori, Muramoto, & Ono, 1991; Ono, Nishijo, & Nishino, 2000). In addition, frontal-basal ganglia systems are involved in processing reward information that facilitate continued performance of these behaviors (Schultz, Tremblay, & Hollerman, 1998). Brain regions other than the basal ganglia that may be involved in propagating obsessive-compulsive behavior include the dorsal raphe nucleus, limbic system, thalamus, and possibly the hypothalamus (Grove, Coplan, Margolin, Hollander, 1996). Neurochemically, serotonergic, dopaminergic, glutamatergic, and opioidergic systems have been implicated (Carlsson, 2000; Rosenberg et al. 2000a, 2000b; Stein, Dodman, & Moon-Fanelli, 1999; Zohar, Chopra, Sasson, Amiaz, & Amital, 2000). It is possible that situations involving chronic anxiety or conflict release a cascade of neurotransmitters in susceptible animals promoting the various forms of OCD. Which neurotransmitters are involved and where they are released, may determine which behavior is expressed. For example, in compulsive grooming, glutamate and dopamine release in the basal ganglia may be facilitatory (Berridge & Aldridge, 2000a, 2000b; Duva et al., 2002; Nordstrom & Burton, 2002). Where eating or drinking compulsions are involved, opioid release in the hypothalamaus may be key (Glass, Billington, & Levine, 1999; Johnson, 1995; Yeomans & Gray, 2002). Where compulsive predatory behavior is involved, dopamine release in the lateral hypothalamus may be instrumental (Gregg & Siegel, 2001). In addition, serotonin would be expected to have a modulatory influence on all of these pathways (Jacobs, Wilkinson, Fornal, 1990). It may be that any or all of these neurotransmitters, and even acetylcholine, may be involved to a greater or lesser extent in the various forms of OCD (Carlsson, 2001).

Attempts to explain the neurophysiologic and neuropathologic roots of OCD by a unitary theory have not met with success, because OCD, like many other neuropathologies, is a complex disorder. The most prevalent theory over the last 15 years has been the hypothesis of serotonin depletion (Barr, Goodman, Price, McDougle, & Charney, 1992). However, serotonin replacement or augmentation strategies have met with only limited success (Cartwright & Hollander, 1998; Jenike et al., 1990). It appears that serotonin's effect is merely palliative, attenuating symptoms of OCD, rather than causing complete remission.

An overview explanation for the pathogenesis of OCD is that genetic susceptibility, in the face of anxiety or stress, promotes the release of opioid precursors and ACTH, activating a cascade of neurophysiological events that prime dopaminergic, noradrenergic, and/or glutamatergic excitatory pathways (Van Wimersma Greidanus, et al., 1985). The compulsion ultimately arising will be governed by the brain region and neurotransmitter system activated. Therefore, for example, excessive grooming might be spawned following ACTH activation of dopaminergic pathways, whereas compulsive consummatory behaviors, like cribbing, might be activated primarily through opioid pathways. Any or all OCDs may involve glutamate and norepinephrine in facilitatory roles, with serotonin exerting an inhibitory or palliative effect. On the basis of such reasoning, the drug treatments that we have applied in the treatment of animal OCDs have met with the success that was predicted. In addition, this paradigm explains the results of other researchers' work entailing a plethora of pharmacological agents. For example, grooming disorders in animals would be expected to respond,

at least to some extent, to treatment with dopamine antagonists, and this has been found to be true (Jenkins, 2001; Willemse, Mudde, Josephy, & Spruijt, 1994). Dopamine antagonists have also been found useful in an animal compulsive disorder involving repetitive movements and motor tics (Dodman et al., 1994). Drug treatments that alleviate anxiety may be partially effective in treating some compulsive disorders in animals (Conceicao & Frussa-Filho, 1993; Sawyer, Moon-Fanelli, & Dodman, 1999), and serotonergic strategies often have a positive therapeutic effect (Moon-Fanelli & Dodman, 1998; Nurnberg, Keith, & Paxton, 1997; Stein, Dodman, & Moon-Fanelli, 1996; Wynchank & Berk, 1998). Equine cribbing responds to treatment with opioid antagonists, as well as an *N*-methyl-D-aspartate (NMDA) receptor blocker (Dodman, Shuster, Court, & Dixon, 1987; Rendon, Shuster, & Dodman, 2001). NMDA antagonists, that block glutamate neurotransmission, have been found effective in other models of OCD in animals, including compulsive grooming in dogs (Dodman et al., in press).

CLASSICAL MODELS

The Lesch—Nyhan syndrome in human patients includes compulsive self-mutilation, aggression, mental retardation and hyperuricemia (Lesch & Nyhan, 1964). The primary biochemical disorder is a deficiency of the salvage enzyme hypoxanthine-guanine-phosphoribosyl transferase (HGPRT; Seegmiller, Rosenbloom, & Kelley, 1967). There is also severe dopaminergic dysfunction, expressed as a deficit in dopamine, dopamine transporters, homovanillic acid, and dopa decarboxylase in all dopaminergic pathways. These changes appear to derive from a decreased number of both dopaminergic nerve terminals and cell bodies (Ernst et al., 1996; Nyhan & Wong, 1996).

It has been suggested that these dopaminergic deficits lead to supersensitivity of postsynaptic dopamine receptors, which causes obsessive self-injurious behavior (SIB). However, SIB is not seen in the dopamine deficiency that causes Parkinsonism, whether spontaneous or induced by antipsychotic dopamine antagonists. Self-injurious behavior can be induced in adult rats and monkeys by unilateral injection of 6-hydroxydopamine into the striatum. This produces supersensitivity to dopaminergic agonists such as apomorphine and L-dopa and pronounced SIB after the administration of these agents (Casas-Bruge et al., 1985; Ungerstedt, 1971). Breese et al. (1984a) administered 6-OH dopamine intracisternally to newborn rats (5 days old) and tested them as adults (60–70 days) with L-dopa, which produced intense SIB. Adult rats treated with 6-OH dopamine had as much depletion of striatal dopamine (95%) as the newborns, but no SIB. They did display paw treading and head nodding (Breese et al., 1984b).

Self-injurious behavior has also been produced in adult rats and mice by the injecting caffeine. The problem with SIB induced by stimulation of dopamine or methylxanthine receptors is that the resultant behavior resembles exaggerated stereotypic gnawing rather than the exaggerated grooming behavior of OCD. The animals show increased motor activity and gnaw on whatever is available—cage contents, other animals, and themselves.

There is also an ethical problem in allowing animals to injure themselves to the point that they produce bloody open wounds. Breese et al. (1984a) handled this

problem by immediately anesthetizing with pentobarbital any rat that bit through its skin. However, this intervention is not always possible, for example, when caffeine is administered orally over a period of 10–15 days (Nyhan, 1973).

GENETIC MODELS

Because the Lesch–Nyhan Syndrome results from a genetic anomaly, it is only natural that several researchers have attempted to construct a model by deleting the gene for HGPRT in mice. The unexpected finding is that HGPRT knockouts are outwardly completely normal and do not display SIB. One explanation for the lack of SIB in knockouts is that adenine phosphoribosyl transferase is able to compensate for the lack of HGPRT (Kuehn, Bradley, Robertson, & Evans, 1987). However, HGPRT knockout mice display more locomotor activity and stereotypy after amphetamine than controls. Even the highest dose of amphetamine produced little SIB (Jinnah, Gage, & Friedmann, 1991). Significant strain differences in amphetamine responses were observed between knockouts on a C57BL/6J background and those on a 129/J background. Additional strain and age differences were reported by Jinnah et al. (1999). Double knockouts involving both HGPRT and adenine phosphoribosyl transferase have been prepared, but they too do not display any SIB (Engle et al., 1996).

CORTICAL-LIMBIC NEUROPOTENTIATED COMPULSIVE MICE

Campbell, McGrath, and Burton (1999) have made a transgenic mouse (D_1CT) in which neurons in the cortex and amygdala that express D_1 receptors have been endowed with a neuropotentiating cholera toxin transgene. These mice are believed to resemble human Tourette and OCD patients. They display increased gnawing, nonaggressive biting of other mice during grooming, repeated leaping, and episodes of perseverant normal behaviors (Campbell et al., 1999). These authors have explained the abnormal behaviors of D_1CT mice as complex compulsions mediated by chronic excessive stimulation of motor pathways in the striatum by glutamatergic neurons in the cortex and amygdala. Cutting excitatory corticostriatal inputs suppresses compulsive behavior in Tourette patients with severe OCD (Kurlan, Kersun, Ballantine, & Caine, 1990). The conclusion of an extensive review by Carlsson (2000) that OCD is a hyperglutamatergic condition has interesting implications for therapy (Carlsson, 2000; Rosenberg et al., 2000a, 2000b).

HOXB8 MUTANTS

Because OCD has been defined in terms of exaggerated grooming, one would expect to see such grooming in an appropriate animal model. This goal seems to have been achieved by Greer and Capecchi with a HOXb8 knockout (Greer & Capecchi, 2002). Mutant mice display exaggerated self-licking, biting, and grooming of control cage mates. There is still no clear indication of the exact role of HOXb8, which is normally expressed in the orbitofrontal cortex, anterior cingulate cortex, and caudate

nucleus (the "OCD circuit"). There is a need to test various drugs that may increase or decrease the grooming behavior of the knockout mice. This model may suffer from some of the drawbacks encountered with other knockouts, for example, deficits during embryonic development. Most knockouts are derived from strain 129 mice. This strain has multiple abnormalities such as loss of the corpus callosum, defective NMDA receptors, and has anomalous drug responses such as failure to develop tolerance to morphine (Kolesnikov, Jain, Wilson, & Pasternak, 1998; Lariviere, Chessler, & Mogil, 2001). These problems are surmountable by constructing conditional knockouts in which the candidate gene is electively inactivated in adult animals, and by backcrossing the mutants to another strain, such as C57 Black. In all cases, the genetic background of controls should be as close as possible to that of the mutant mouse.

DRUG-INDUCED GROOMING

A number of substances, including peptide hormones and neurotransmitters such as bombesin, vasopressin, substance P, β-endorphin, and ACTH, when injected directly into the brain or spinal cord of rats and mice can induce vigorous grooming bouts (Dunn & Berridge, 1987; Gispen & Isaacson, 1981; Rees, Dunn, & Iuvone, 1976). The meaning of these responses is unclear, although dopamine plays an important role (Drago, Contarino, & Busa, 1999). However, the use of intracisternal, intracerebral, or intrathecal injections in a standard assay is undesirable.

One aspect of grooming that we have concentrated on is compulsive scratching and licking. This behavior in dogs responds to treatment with narcotic antagonists and SSRIs (Dodman et al., 1988; Rapoport et al., 1992; Wynchank & Berk, 1998). A brief period of vigorous scratching is readily produced in mice by an intradermal injection of the mast-cell degranulating compound 48–80 (Inagaki et al., 2002; Kuraishi, Nagasawa, Hayashi, & Sato, 1995). Kuraishi et al. (1995) have demonstrated that this response is not due to the release of histamine. Mouse mast cells are rich in serotonin rather than histamine, and Yamaguchi, Nagasawa, Satoh, and Kuraishi (1999) have shown that serotonin induces scratching by stimulating $5HT_{2A}$ receptors. In unpublished work, we have found that narcotic agonists and antagonists, as well as NMDA antagonists, can block the pruritus produced by injecting serotonin into the skin of mice. There are strain differences. We have observed good responses with Swiss-Webster, CD-1, and C57BL/6 mice, but 129/J mice show much less scratching. Takano et al. have described a mutant strain, Nc/Nga, that scratches spontaneously, and they use it as a model of spontaneous atopy (Takano, Anai, & Kurachi, 2003). Because scratching mice respond to medications used for OCD in the same way as spontaneously scratching dogs and cribbing horses, this model seems useful for testing drugs for their ability to decrease OCD. The scratching assay has recently been automated, so that quantitative data are readily accumulated (Inagaki et al., 2002; Nojima & Carstens, 2003).

A simpler, less intrusive way to induce grooming is to mist an animal with a gentle water spray (Berridge, Fentress, & Parr, 1987). This method was also used by Greer and Capecchi (2002), who found that HOXb8 mutants spent twice as much time grooming as controls after a water spray. We have used it to back up measurements of scratching induced by serotonin. One advantage of this assay is that it adds additional components of grooming: licking, face-washing, "wet dog shakes," to the scratching

response. However, we have found that of the various grooming behaviors evoked by a water spray, only fur-licking and face-washing occur with sufficient frequency to allow for meaningful analysis. Another advantage is that there is no confounding effect from the absorption of intradermal serotonin and its delivery to the central nervous system, although this possibility can be tested by comparing with mice that have been made to scratch by an intradermal injection of compound 48–80.

SPONTANEOUSLY OCCURRING MODELS

A number of spontaneously occurring animal models of OCD have been suggested (Dodman et al., 1997), but the most well-documented of these is canine lick granuloma, a.k.a. ALD (Rapoport et al., 1992; Stein, Shoulberg, Helton, & Hollander, 1992). Dogs and cats may groom themselves excessively, and may also show nursing/ingestive compulsions and predatory-type compulsive behavior. Some pet animals hoard objects or seemingly have concerns about symmetry. Horses display appetitive/ingestive compulsions, including cribbing and tonguing, and locomotor compulsions, like stall walking and weaving. Pigs chew chains and bite bars, and zoo animals rock, weave or pace, masturbate, engage in rectal probing, or hair pulling, depending on the circumstances and their natural predilection.

Some of these models have face validity. For example, ALD in dogs appears analogous to compulsive hand washing by people; FPA in cats and feather picking by birds are phenotypically similar to human trichotillomania (Moon-Fanelli, Dodman, & O'Sullivan, 1999); hoarding by Munchkin cats closely resembles human hoarding compulsions (Dodman & Oliver, 1996).

Predictive validity has been confirmed for some of these putative models, for example, certain animal compulsions have been attenuated following treatment with traditional human anti-obsessional medications, like clomipramine and fluoxetine (Hewson, Luescher, Parent, Conlon, & Ball, 1998; Moon-Fanelli & Dodman, 1998; Overall & Dunham, 2002; Rapoport et al., 1992; Seksel & Lindeman, 1998, 2002; Sawyer et al., 1999; Wynchank & Berk, 1998). Porcine bar biting and chain chewing responds to fluvoxamine along a precisely similar time course and to a similar extent as humans with OCD, and is regarded by one key researcher as the model of choice for the study of OCD (Olivier, 1996, Personal communication, 1996). Compulsive pacing in a polar bear was effectively treated with fluoxetine (Poulsen, Honeyman, Valentine, & Teskey, 1996). In other conditions, like feline FPA and avian feather picking, the effectiveness of dopamine blocking or opioid blocking drugs parallels the efficacy of similar treatments for trichotillomania (Christenson, Crow, & MacKenzie, 1994; Moon-Fanelli et al., 1999; Stein & Hollander, 1992; Sawyer et al., 1999). In the following sections, we review specific spontaneously occurring animal models of OCD.

Dogs

The best known, most widely publicized, model of OCD is that of canine ALD. The behavior manifested by dogs with ALD, involves repetitive licking of the distal extremities of the limbs, most often the forelimbs. ALD occurs most commonly in certain breeds of dogs, mostly large breeds, and seems to affect those of anxious disposition. Sometimes a situation of acute or chronic conflict is associated with the

onset of the behavior and it may originally start as a displacement behavior that serves as a stress-reducing strategy. ALD has face value as a model of OCD and has predictive validity as an OCD model in the sense that its response to selective serotonin reuptake inhibitors was predicted and subsequently confirmed (Rapoport et al., 1992; Wynchank & Berk, 1998). In addition, ALD responds to treatment with opioid antagonists (Dodman et al., 1988), which, as it turns out, may be working on either opioid or NMDA receptors.

Compulsive tail chasing by dogs has emerged as another possible model of OCD (Dodman et al., 1997; Moon-Fanelli & Dodman, 1998; Seksel & Lindeman, 2001). Tail chasing is a canine compulsive disorder in which affected individuals spin in tight circles while focusing on their tail. Sometimes, affected dogs bite their tail causing injury or even auto amputation. By our definition, the dog must engage in tail chasing for more than an hour a day to be considered compulsive, though there are many subthreshold cases that are clearly far from normal. Tail chasing is thought to be derived from predatory behavior because of its phenomenology and because it occurs in dogs with high prey drive. The breeds most commonly affected are Bull Terriers and Bull Terrier crosses, and German Shepherds and German Shepherd crosses. Conflict frequently precedes the onset of tail chasing, which occurs most often in peripubertal dogs, between 5 and 8 months of age. The condition responds to treatment with serotonin reuptake inhibitors (Moon-Fanelli & Dodman, 1998) and has also been reported to respond to opioid receptor blockade (Brown, Crowell-Davis, Malcolm, & Edwards, 1987). Though the phenotype of compulsive tail chasing is not as compelling as ALD, many other aspects of the condition are analogous:

1. The behavior is compulsive, repetitive, and serves no useful purpose, except, perhaps, providing an outlet, by way of displacement, from some irresolvable dilemma (like being shut in a crate for hours each day).
2. Performance of the behavior is time consuming (>1 h per day) and it may cause significant social and/or physical impairment.
3. Compulsive tail chasing usually arises at a time in a dog's life equivalent to the human teenage/early adult period.
4. There seems to be a familial pattern for the behavior.
5. Anxiety or conflict often bring about expression of the behavior.
6. Physically preventing the dog from performing the behavior will cause it to become more anxious.
7. There is a positive response to treatment with serotonin reuptake inhibitors.

Of course, it is impossible to say whether affected dogs are actually obsessing (though they appear to be), and it can be assumed that they have no insight regarding the excessive nature of their behavior. However, children sometimes exhibit compulsive behaviors without preceeding obsessions, and their insight is often poor.

Compulsive playing with objects is another canine model of OCD thought to be derived from innate predatory instincts. Like tail chasing, it occurs mainly in breeds with high prey drive and in individuals that are of high-strung anxious temperament. The compulsive behaviors typically occurs for the first time around puberty and is precipitated by stress. Like tail chasing, it appears to respond to treatment with serotonin reuptake inhibitors, though this has not yet been tested empirically.

Flank sucking is another compelling canine model of OCD. It involves primarily Doberman Pinchers and presents as mouthing or sucking directed toward the dog's

flank. Some Dobermans also engage in blanket sucking, which is probably related behavior. We believe flank sucking derives from nursing behavior and thus the nearest human equivalent might be thumb sucking. The condition arises in young dogs but may persist into adulthood. It tends to affect high-strung, nervous dogs, and may be precipitated or exacerbated by stress. Severe flank sucking, causing alopecia and skin abrasion, was successfully treated in one dog brought to Tufts University Animal Behavior Clinic, following a short course of treatment with the opioid antagonist naltrexone.

A new canine model of OCD is provided by dogs that have developed a cycle of repetitive self-scratching, biting, and chewing. While these dogs may have ongoing evidence of atopy or allergy, there does seem to be a central component involved in propagation of the itch/scratch cycle, and the behavior can be significantly attenuated by treatment with an NMDA antagonist (Dodman et al., 2004).

Other canine compulsive behaviors that have received little or no attention in terms of their etiology or treatment include, light or shadow chasing (mainly dogs with high prey drive eg, terriers, herding/hunting breeds), compulsive digging, ingestion of inedible objects, stone chewing, nail biting, and pacing/circling.

Cats

Perhaps the best feline model of OCD is FPA. Like trichotillomania, FPA is an aberration of normal grooming behavior. Genetic factors may underlie the expression of FPA because oriental breeds of cat are overrepresented in the demographics of the condition (Moon-Fanelli et al., 1999; Sawyer et al., 1999). As with trichotillomania, females are more commonly affected than males and stress seems to be a precipitant (Folks & Warnock, 2001; Moon-Fanelli et al., 1999; Oranje, Peereboom-Wynia, & DeRaeymaecker, 1986). In addition, both FPA and trichotillomania respond to treatment with serotonin reuptake inhibitors (Sawyer et al., 1999; O'Sullivan, Christenson, & Stein, 1999).

Another interesting feline compulsion is a behavior known as wool sucking. The behavior takes the form of compulsive nursing on woolen substrates and often involves their ingestion. It can progress to the eating of inanimate objects, at which time it appears as a substrate-specific of pica. Plastic shower curtains and shoe laces are frequently the materials ingested, although sometimes affected cats are drawn to ingest newspaper, silver foil, or to chew at wires. In our clinic, the behavior has been shown to respond to serotonin reuptake inhibitors, though results of these clinical trials have not been formerly documented, as yet. Wool sucking would appear to be a reasonable model for the study of various oral and appetitive cravings in humans.

Other feline compulsive behaviors, including lip-licking, nail biting, hoarding, and water playing, have been less well studied (Dodman et al., 1997), but some, like hoarding, appear to be worthy of closer attention.

Horses

Equine stall vices, seemingly pointless repetitive behaviors, formerly referred to as stereotypies, are now thought to represent equine compulsive behaviors. They occur in domesticated, stabled horses only and are thought to represent some kind of coping strategy expressed in response to:

1. Lack of control over their environment.
2. Management factors, especially diet/mode of feeding.
3. Environmental restrictions, especially regarding space.
4. Inadequate opportunities to socialize with other horses.
5. Inactivity, boredom, frustration, and inability to engage in species-typical behaviors.

The prototypical equine compulsion is cribbing. Cribbing is a condition in which affected horses grasp the edge of their stall with their incisor teeth, lean back, tense their large neck muscles and make a swallowing motion. Affected horses are seemingly more active in the stall than their noncribbing counterparts, rarely resting, and appear hypervigilant. There is evidence that the tendency for cribbing is genetic (Bachmann & Stauffacher, 2002). From its form, cribbing appears to be derived from consummatory behavior. In support of this contention, many cribbing horses also "wind suck," which entails a swallowing sequence accompanied by a gulping sound. Aerophagia in humans is a disease that mainly affects people with developmental disabilities living in institutions (Van der Kolk, Bender, & Goris, 1999). Aerophagia also occurs in dogs that are fed infrequently and/or have been recently kenneled or transported (Elmwood, 1998). Stress, confinement, and the inability to control the environment is a common thread linking the expression of aerophagia across the species (Michel & Blanc, 1993). Whether gulping air is a compulsive disorder or simply a displacement behavior is unclear, but dogs exhibiting this behavior have been found to respond positively to treatment with serotonin uptake inhibitor medications (Overall, 1994).

Cribbing has been shown to respond positively to treatment with opioid antagonists (Dodman et al., 1987). While classic human OCD does not respond well to treatment with opioid antagonists, alcoholism, which may involve a compulsive drinking component, does respond to such treatment (Romach et al., 2002). It is noteworthy that cribbing and alcoholism (at least, the compulsive aspects of the latter), both ingestive behaviors, probably rely heavily on opioid mechanisms for their propagation (Gosnell, Morley, Levine, 1986). Recently, cribbing has been shown to respond to treatment with dextromethorphan (Rendon et al., 2001). Because dextromethorphan does not bind to opioid receptors, its effects must be mediated by some other action, probably blockade of glutamate at NMDA receptors. Opioid antagonists, like naltrexone, also block NMDA receptors. It is unclear, at this time, whether naltrexone's opioid receptor blocking properties are instrumental in its effect on cribbing. What is clear is that NMDA receptor blockade is an effective therapy, suggesting that this latter strategy may be worth trying in humans with OCD spectrum conditions, especially those linked to ingestive behaviors, such as compulsive drinking, smoking, nail biting, and trichotillomania.

Weaving is another well-known equine compulsive disorder that is actually a form of abbreviated fence walking, that is, it is a locomotor compulsion derived from walking behavior. Affected horses shift their weight from one forefoot to another, their rear feet tread a typical gait pattern, and their heads sway from side to side, as they effectively "walk in place." Weaving may arise as a displacement behavior in anxious horses awaiting feeding, but can progress to the extent that it occurs for hours each day without any obvious triggers. At this stage, the behavior is regarded as having assumed compulsive proportions. Weaving as been shown to respond well

to treatment with an SSRI, paroxetine (Nurnberg et al., 1997). Weaving may be a good model for the study of certain ritualistic behaviors and compulsive movement disorders exhibited by some OCD patients.

Stall walking, or box walking, is another locomotor stereotypy that occurs in horses. Affected horses pace mindlessly for hours each day, often displacing their bedding from the center to the periphery of the stall through their constant motion. This behavior can be induced by the injection of morphine or apomorphine and can be blocked by opioid antagonists (Shuster et al., 1984). Some horses engage in a peculiar flank-biting behavior associated with squealing, spinning, and hemiballismus. This behavior, which responds to treatment with opioid antagonists, and perhaps dopamine antagonists, is thought to represent an equine model of Tourette's syndrome (Dodman et al., 1993). Many affected horses also exhibit more classical equine compulsive disorders, such as cribbing or stall walking. The behavior appears to be familial and is precipitated by environmental conflict. Other equine compulsions include: lip flapping, tonguing, digging, head nodding, and wood chewing. Little work has been done with respect to any of these other conditions, though being repetitive, excessive, and seemingly pointless, they do fit well under the umbrella diagnosis of "compulsive behavior."

Pigs

Perhaps the best porcine model of OCD is that of pigs that constantly chew chains or bars at the periphery of their stall (Dodman & Olivier, 1996). This behavior was found to occur in susceptible pigs only and to be responsive to a selective serotonin reuptake inhibitor, fluvoxamine, to the same extent and over the same time course as human OCD sufferers respond. Again, this behavior might be valuable for the study of various oral and appetitive compulsions in humans.

Birds

Avian feather picking resembles trichotillomania in people (Moon-Fanelli et al., 1999). People with trichotillomania often select just the right hair (often new growth), pluck it out, inspect it carefully, chew on the hair bulb, sometimes ingest the hair (trichophagia), and then discard the hair before beginning again. Birds do much the same. They look for just the right feather, often new growth, pluck it out with their beak, inspect it, shred the shaft of the feather, discard it, and then repeat the process again and again, until the area that the bird can reach with its beak is completely denuded. Phenomenologically, avian feather picking is similar to trichotillomania in every detail. Feather picking, like OCD, seems to have genetic roots, being more common in highly-strung psittacine species, like African grey parrots. Furthermore, feather picking responds to the same medications as trichotillomania, including serotonin reuptake inhibitors, opioid antagonists, and dopamine antagonists (Dodman et al., 1997; Jenkins, 2001; Moon-Fanelli et al., 1999; O'Sullivan et al., 1999).

DISCUSSION

From an ethological perspective, it makes sense that animals other than the human animal would also suffer from OCD. However, perhaps the word "suffer" is not

the correct term to employ where animals are concerned, because animals seem to lack insight into their behavior, probably not regarding it as excessive and thus not experiencing ego-dystonia. Nevertheless, animals do display compulsive disorders, whether these behaviors cause them grief or not. The behaviors likely arise out of anxiety and conflict and represent some kind of coping mechanism. Another question that arises is, "do animals obsess?" While obsessions are impossible to validate in animals, those of us who spend hours observing these nonverbal species agree that some of them certainly appear to obsess. A tail chasing Bull Terrier, for example, may spend long minutes or even hours with its gaze transfixed on its tail, without moving a muscle. A light chasing dog may stare at a reflection for several minutes, and a dog with concerns about order or symmetry may be obliged to position one or more pieces of kibble in a precise way before he eats. If these animals are not thinking, it is hard to imagine what they are doing. Of course, as with humans, obsessions do not necessarily precede compulsions, and some animals monotonously engage in their compulsion seemingly without a thought in their mind. Chain chewing/bar biting pigs or cribbing horses provide examples of such apparently mindless behavior. It is not hard to see why these behaviors were formerly referred to as stereotypies, but the term stereotypy does not adequately explain all of the other facets of the behaviors that are now well substantiated. For example, from the above account, it may be seen that a large number of animal compulsive behaviors seem to have some familial or genetic underpinnings; that they seem to arise in anxious/type "A" individuals; that they arise under conditions of stress and conflict; and that they respond to treatments identical to those employed for a therapy of OCD in people.

From an ethological perspective, it makes sense that compulsive disorders would be expressed as a spectrum of behaviors representing behaviors typical for the species. This is one way in which human compulsive behaviors encompassed by the OC spectrum can be explained. Thus, mysterious repetitive behaviors of previously unknown etiology that have long challenged veterinarians and defied treatment can now be explained by the paradigm proffered by the ethological model of animal OCDs. One by one, these veterinary conditions are being found to have features in common with human OCD, from their genetics and etiology to their expression and treatment, and the predictive power of this paradigm is impressive.

One concern that pundits have had in the past, when closely comparing the pathophysiology of human with animal compulsive disorders, is that animals do not have a prefrontal cortex and, as such, may not be capable of the same ruminations and doubt as humans. But this concern is invalid, as many species we have discussed, dogs and cats in particular, have been shown to have a prefrontal cortex; if such as structure is indeed necessary for the performance of OCD. Psittacine birds, the biggest worriers and doubters of the avian species, may not possess this supposedly vital structure, as their brains are conformed in a different way from mammalian species.

As with human OCD, animal compulsions are often co-morbid with various anxiety-related conditions (Seksel & Lindeman, 2001, Sawyer et al., 1999). Also, as in human OCD, more than one compulsion may exist in one animal, and suppression of one OCD may lead to the emergence of another. In one case, a Bull Terrier that chased its tail in the center of its owner's kitchen for hours on end was subjected to aversion therapy when a dog trainer advised the owner to use an electric shock collar to arrest the behavior. The dog resorted to pacing around the periphery of the room

instead of the tail chasing. The new behavior was more acceptable to the dog's owner who was thus satisfied with the therapeutic result. ALD has also been suppressed by electric shock in a series of dogs (Eckstein & Hart, 1996). The authors did not state whether other compulsive disorders arose in lieu of ALD.

When considering spontaneous occurring animal models of OCD as models of the human condition, it is best to compare like with like. Thus, if one were planning to study compulsive hand washing, ALD would be a good model. If trichotillomania was the psychiatric condition of interest, then FPA or avian feather picking would be more appropriate to study. If compulsive eating was the subject under scrutiny, then equine cribbing or porcine chain chewing might be most relevant.

Presently, the flow of information has been from the field of psychiatry toward the field of veterinary medicine, but with suitable models the direction of flow of information could be reversed. If, for example, it had been discovered that ALD responded to treatment with clomipramine and fluoxetine prior to the therapeutic efficacy of these drugs being known in human OCD sufferers, we would certainly have advised that these drugs be tested as treatments for OCD. Such a measure would have lead to the discovery of the benefits that serotonin reuptake inhibitors are now known to provide in the treatment of OCD. Perhaps this reverse flow of information is about to occur, as it has recently been demonstrated, in mouse models of OCD, and in spontaneously occurring compulsive disorders in dogs and horses, that NMDA receptor blockade is effective in treating specific animal compulsions. The involvement of glutamate in the propagation of at least some OCDs is logical from a basic biological perspective. Whether NMDA blockage ever becomes a primary treatment for OCD, or simply an adjunctive therapy for OCD patients that fail to respond to treatment with serotonin reuptake inhibitors, this new approach clearly warrants further study.

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Chapter 4

BEHAVIORAL AND FUNCTIONAL ANIMAL MODELS OF OCD

Arthur C. Houts

A number of different animal models of obsessive-compulsive behavior have been proposed over the past half century, and recent veterinary models are reviewed in Chapter 3 of this volume. The current chapter focuses on a particular set of experiments conducted with mongrel dogs almost half a century ago. Those experiments, led by Richard L. Solomon and his colleagues and students, were among the most important studies ever conducted on behavioral theory of avoidance learning. Unfortunately, much of that work has been forgotten, and its relevance to understanding obsessive-compulsive disorder (OCD) has not been fully appreciated. At the time these investigations were conducted and published, neither their authors nor clinicians of the period saw the full relevance of their work to OCD as it has come to be conceptualized over the ensuing years. Solomon and his colleagues did indeed see their work and results as possibly having some relevance to human obsessions and compulsions, and a few clinicians also made connections to the human malady. Today, we have the benefit of hindsight wherein we can look again at this classic series of studies and view them from our current perspective on how to think about obsessions and compulsions and their interaction within an avoidance learning formulation.

This chapter contains four sections. First, to set the stage for understanding the relevance of the Solomon work, it is important to review formulations of OCD in an historical context. Second, the Solomon work is summarized with particular emphasis on how it relates to some current conceptions of obsessive-compulsive behavior and behavior therapy for OCD. Third, some limitations of the Solomon work for understanding obsessive-compulsive behavior are noted, and the relative utility of this work as compared with other animal models is examined. Finally and based on the Solomon work, some indications for future research in animal analogues of obsessive-compulsive behavior are presented.

LEARNING THEORY FORMULATION OF OBSESSIVE-COMPULSIVE BEHAVIOR

Current behavioral conceptualizations of OCD presume that the complex of behavior we refer to as OCD contains two components, the obsessive or anxiety provoking component and the compulsive or anxiety reducing component. Variations of this formulation have been incorporated into the various editions of the *DSM* since 1980 (American Psychiatric Association, 1980, 1987, 1994, 2001) even though the *DSM* formulations allow for a much broader formulation as well. This two-part formulation based on the anxiety reduction function of the compulsive behavior was not always widely accepted or taken for granted. To appreciate the significance of the Solomon work on avoidance learning, it is worth noting briefly the history of how this current formulation came about within clinical circles.

History of OCD Conceptualization in Behavior Therapy

Early behavior therapists emerged from multiple streams of investigators who came to behavior therapy from different vantage points (Krasner, 1971; Krasner & Houts, 1984). What ended up as a recognizable band of followers under the banner of behavior therapy actually began as a number of small groups and even individual clinicians who were drawn to a behavioral approach to clinical problems as distinct from the then dominant psychoanalytic view of the 1950s in the United States and the United Kingdom. Some of the earliest efforts to treat obsessions and compulsions, mostly obsessions, came from investigators in South Africa, many of whom eventually made their way to England and the Maudsley hospital training program headed by Hans Eysenck.

As a psychiatrist with a keen interest in learning theory and experimental psychology, Joe Wolpe had to turn to his psychology colleagues to find kindred spirits in terms of thinking about clinical problems from a behavioral point of view. Among those he consulted was James G. Taylor (1897–1973) who was a Senior Lecturer in the Psychology Department of the University of Capetown from 1924 to 1962. Trained at Aberdeen University in the United Kingdom, Taylor subsequently visited the United States and eventually retired to the United Kingdom where he was for a time affiliated with the Maudsley hospital group that developed behavior therapy in the United Kingdom. Taylor subsequently published his study of perception as a behavioral process (Taylor, 1962). In the 1950s, Taylor "experimented" with various behavioral procedures for the treatment of anxiety problems. Those case studies were rarely published, so a full description of his procedures and their outcomes has not been publicly presented. In an interview with Leonard Krasner in 1969 in London, Taylor described several cases of multiple phobia and obsessions with compulsions (Krasner, L., personal communication [audiotaped interview], 1969). He utilized what we would today call graduated in vivo exposure with response prevention. For example, in a case of anxiety attacks during driving, he accompanied the patient on drives designed to evoke the anxiety reactions. He also exposed compulsive hand washers to more and more anxiety provoking circumstances and blocked the washing behavior. Only hints of this work survive in published form. In his *Psychotherapy by Reciprocal Inhibition*, Wolpe cited a conversation with Taylor that described Taylor's treatment of obsessions, where Taylor used thought stopping to reduce the occurrence of obsessive ruminations and

unwanted thoughts (Wolpe, 1958). Taylor also published a case study in the first volume of *Behaviour Research and Therapy*, and in this publication he described the connection between obsession and compulsion as a behavioral chain that could be broken by interrupting the repetitive behavior (Taylor, 1963). Although this case was one involving trichotillomania and was not strictly speaking a case of OCD in the way that most behavioral clinicians would currently define OCD, the idea of blocking the repeated behavior of hair pulling because it was intimately connected to some stimulation (a perceived itch) could be regarded as a precursor to modern treatment of OCD by exposure and response prevention. Taylor reported that when the patient interrupted her act of lifting her arm toward her head, she was successful in blocking the remainder of the response that would have resulted in feeling the eyebrow area and pulling the hair. Curiously enough and consistent with Taylor's own theory regarding the behavioral basis of perceived experience, the patient's phenomenal experience of having an itch in the forehead also dissipated with the cessation of the chain of motor behaviors.

Just where and when our current two-part conceptualization of OCD arose historically is difficult to pinpoint. Like most such innovations it would seem that the concepts were "in the air" at the Maudsley training program and among those affiliated with the British founders of behavior therapy. Foa has noted that Victor Meyer was among the first to report procedures that we would now recognize as exposure and response prevention (Foa, 1996). In the presence of anxiety provoking cues, Meyer blocked hand washing in several patients and reported successful outcomes (Meyer, 1966). Meyer subsequently reported further success with multiple cases treated in a similar manner on the inpatient unit of Middlesex hospital where he conducted behavior therapy with his psychiatrist colleague, Ted Chesser (Meyer, Levy, & Schnurer, 1974). In his 1970 book with Chesser, *Behavior Therapy in Clinical Psychiatry*, Meyer was fairly explicit about his reliance on the work by Solomon and colleagues to inform his conceptualization and treatment of OCD (Meyer & Chesser, 1970), and this deserves considerable attention in the context of the present chapter on behavioral and functional animal models.

In reviewing what they regarded as the relevant animal experimentation that informed their clinical work at the Middlesex behavior therapy unit, Meyer and Chesser made it clear that they considered "traumatic avoidance" learning, a phrase introduced by Solomon, a prime candidate for the correct animal analogue. Following due consideration of possible limits of any animal models for human experience of compulsions, they wrote as follows:

> A variety of methods have been employed to induce stereotypy or fixated responses in animals. Maier (1949, 1956) found that a majority of rats develop fixated responses, either positional or symbolic, when presented with the combination of an insoluble discrimination problem and noxious stimulation. The fixated response may persist even when the problem is made soluble, no noxious stimulus is presented, and the correct response has been learned. Special methods were required to eliminate the fixated responses. The persistence and resistance to extinction of these is reminiscent of traumatic avoidance learning. The use of noxious stimulation in these experiments may therefore be a crucial factor in the persistence of the responses. In other words, any motor component of an active avoidance response emitted frequently because of the stimulus or drive conditions of the organism might appear to be a repetitive or fixated response and the parameters influencing it will be the same as those influencing avoidance conditioning (Meyer & Chesser, 1970, p. 63).

In the material following this quotation, Meyer and Chesser went on to cite Solomon's work. They also offered an explicit formulation of compulsive hand washing as a ritualistic behavior of active avoidance maintained by anxiety reduction.

The aforementioned anecdotes certainly suggest that the core clinical concept of obsessive-compulsive behavior as a form of active avoidance learning owes its conceptual lineage to the work of Solomon and his colleagues. Repetitive behavior having no obvious reinforcing consequences persists and does not extinguish. What Meyer and others had the wisdom to see is that this provided a possible model for repetitive behavior in humans when such behavior persisted even in the face of some rather obvious negative consequences. Solomon's models of traumatic avoidance provided the insight that such behavior is in fact reinforced, but the reinforcement is of a rather unique kind, namely the reduction of or perhaps prevention of a previously learned emotional reaction that is aversive. Doing the seemingly futile repeated behavior manages and copes with, and perhaps prevents, an emotional state that is extremely negative. This has been the core insight of contemporary behavior therapy for OCD in humans, and this formulation of such human problems has been the basis for effective behavioral treatment for OCD.

Current Treatment Procedures in Behavior Therapy

From Taylor's and Meyer's initial efforts to implement exposure and response prevention, behavior therapy approaches have expanded considerably to include other ancillary strategies. These more recent formulations of anxiety and the role of anxiety in obsessive-compulsive behavior built upon the work of such pioneers as Taylor and Meyer, but they have also probably contributed to the loss of contact with the Solomon work of over 50 years ago.

In the mid-1950s and for the next two decades, much of clinical behavior therapy was quite directly related to animal experimentation. The link between the animal laboratory and the human clinical arena was much stronger than it is today so it was not uncommon for clinical investigators to draw directly from animal experiments to formulate their interventions. That climate changed gradually with the availability of more sophisticated technology to study human physiological responding and with the opening of the Pandora's box of cognitive functioning and information processing, the cognitive revolution (Baars, 1986). The more recent blending of cognitive psychology with basic neurophysiology and cognitive neuroscience has probably made the learning experiments of 50 years ago even more remote.

Foa and Kozak (1986), who proposed one of the more widely accepted formulations of OCD, noted that two conditions were necessary to reduce anxiety: (*a*) anxiety must be aroused and experienced and (*b*) new information about the basis or reality of the fear must be provided and "emotionally processed." The notion of emotional processing reflects the influence of cognitive and physiological studies of anxiety as represented, for example, by Lang and his colleagues (Lang, 1994). When translated into therapy procedures, this has amounted to a good deal more attention to belief change as part of the successful use of exposure and response prevention. The learning-based behavior therapy of Taylor and Meyer has become *cognitive and behavior therapy*. The basic idea of exposing patients to conditions that can arouse anxiety and instigate ritual performance has remained a centerpiece of cognitive and behavioral

therapy for OCD, but consistent with more emphasis on human processes of change, greater emphasis than before has been given to changes in what people verbalize about their fears and the connection of those beliefs to the ritualized avoidance behavior. In some respects, exposure and response prevention has become an occasion to conduct talking therapy in addition to an occasion to mechanically provide conditions for extinction of learned negative emotional reactions.

Another aspect of contemporary cognitive and behavior therapy that has perhaps led to a loss of interest in the early Solomon work has been the search for origins or risk factors in human OCD. Among the more important recent developments in cognitive and behavioral therapies for OCD, the concept of thought—action fusion has been widely endorsed as a mechanism to explain how some individuals develop OCD whereas others do not (eg, Rachman & Hodgson, 1980). This began with the observation that the obsessive fears of OCD patients were not, as previously believed, all that unusual. Several investigations have now corroborated the fact that most people do in fact experience intrusive thoughts with content similar to that reported by OCD patients (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984). That phenomenon alone does not distinguish OCD patients from ordinary people. What does appear to make OCD diagnosed patients different is how they react to these otherwise normal thoughts. The concept of thought—action fusion supposes that when patients as compared to ordinary people experience the thought "I might be contaminated by touching the toilet seat" patients fail to dismiss the thought and fail to take limited corrective action. What makes the patients different is that they equate the thought with its actual content. Having the thought of being contaminated becomes the equivalent of actually being contaminated therefore requiring immediate action to reduce the perceived danger. Again, the shift of emphasis is from the emotional reaction to an explanation of how humans acquire the emotional reaction due to some type of cognitive distortion.

Recent developments in behavior therapy exemplified by the new term, cognitive and behavior therapy, reflect broader trends in the field of psychology where there has been a greater turn inward to such matters as cognitive and neurological processes. The latter may be eminently amenable to animal investigation, but it is quite difficult to see how one could examine the beliefs of animals without language. Having set that stage of background in behavior therapy and more current concepts of treatment for OCD, it is still instructive to revisit the Solomon work because that work has remained for over 50 years the basis for the most effective treatment for OCD, exposure and response prevention.

THE SOLOMON WORK ON TRAUMATIC AVOIDANCE

Solomon and Wynne (1953) described their procedures as "traumatic" avoidance because of the intensity of the animals' responses in the early stages of their experimental procedure, which is more fully described below. Their description of the dogs' reactions was vivid.

> The dog will scramble rapidly and vigorously around the compartment, slamming into walls, perhaps, or leaping up against them; he will simultaneously emit a high pitched

> screech, will salivate profusely, will urinate and defecate in a manner which could be called "projectile elimination," and will roll his eyes rapidly and jerkily; in addition, his pupils will dilate, portions of his hair will stand on end, small muscle groups all over his body will tremble, and his breathing will consist of short, irregular gasps (Solomon & Wynne, 1953, p. 1).

Inundated as we are 50-years later with talk of trauma and Post Traumatic Stress Disorder (Baldwin, Williams, & Houts, in press), it is important to note that Solomon and his colleagues did not in fact relate their work to then existing concepts of war neurosis and so called gross stress reaction of *DSM-I* (1952). Instead, what interested Solomon et al. was the dynamic of avoidance learning and unlearning under conditions where the initial response was indeed extreme as judged by the experimenters.

In casting this work as an animal model of OCD, it is important to note that the focus is on function and dynamics of acquisition and especially of extinction. No claim is made that the Solomon work is relevant for questions of etiology of OCD. In fact, it is unlikely that individuals with OCD behaviors acquired those behaviors under conditions of trauma and discreet moments where massive fear reactions marked the beginning of the compulsive behaviors. In what follows, the Solomon work is described in order to highlight its relevance for conceptualizing OCD in humans from a functional point of view, and this presentation is divided into four parts: (1) apparatus and experimental preparation, (2) results of acquisition trials, (3) extinction and resistance to extinction, and (4) additional findings and OCD-like behaviors.

Apparatus and Experimental Preparation

The Solomon work on traumatic avoidance was carried out using mongrel dogs, which were placed in a specially built two-chamber shuttle box that was 40 in. high. Each side of the box was about 4 ft long and 2 ft wide, and the two sides were separated by a hurdle barrier, and gate that could be raised and lowered to permit or prevent passage to the opposite side. The apparatus was constructed from 2 in. $\times$ 4 in. lumber and reinforced with sheet aluminum to withstand the force of the dogs' attempts to escape. The ceiling of each compartment was covered with heavy wire mesh and layers of cheesecloth that permitted overhanging lights to shine into each compartment to illuminate one compartment at a time. The experimenters could see into the compartments from above, and the cheesecloth served as a kind of crude one way vision screen. The floor of the apparatus consisted of steel grid bars that could be electrified to deliver shock to the animal's feet. Circuits were isolated to prevent serious injury to the animal, yet shock was delivered at an intensity that was painful and just below an intensity that would produce involuntary muscle movements.

Animals were pre-tested to insure that they were not already jumpers when the light in their starting compartment went off, the gate was raised, and the light in the adjacent compartment went on. Animals were then subjected to conditioning trials where the same sequence of events occurred along with the floor on the starting side compartment becoming electrified as the light went off. The shock remained on until the dog jumped or climbed over the hurdle into the lit compartment, at which point the gate closed and the training trial ended. If after 2 min of shock, the animal still failed to escape the shock, the trial was ended and then restarted after 1 min. On those

training trials where the animal escaped successfully after being shocked, another training trial commenced after 3 min by reversing the order of events and ended when the dog traversed to the other compartment.

For all of the training trials there was a 10 s delay between the onset of the conditioned stimulus (CS: light out, gate up, light on) and the onset of the unconditioned stimulus (US: shock). During this training, failure to escape the shock was called a *shock trial* as was a successful escape because the animal was still shocked even though it did escape within 2 min of the shock onset. Any trial in which the animal got to the other compartment in less than 10 s before the shock came on was called an *avoidance trial*.

Results of Acquisition Trials

In their initial study involving 30 dogs, Solomon and Wynne defined acquisition of the avoidance response as 10 successful avoidances of shock in 10 consecutive trials. They noted that their typical dog received four shocks in the first four trials but quickly began making avoidance responses by the fifth trial. From trials 6–11, the typical animal was shocked two out of six times and after 12 trials, most of the animals avoided shock with 100% accuracy. In addition to learning the avoidance response rather quickly, the animals also demonstrated faster and faster responding to the CS with each successive training trial.

The experimenters also noted that there were considerable individual differences from animal to animal. Indicating clearly that they had human anxiety conditions in view in conducting these studies, Solomon and Wynne and their students were careful to note their observations about the animals' differences in temperament. They recorded and weighted behaviors they regarded as signs of emotionality and intensity of fear reactions (eg, drooling, breaking wind, attacking the apparatus). These signs of emotional reaction were more frequent and more intense in the trials immediately preceding the first successful avoidance trials. They also noted that animals that were "more anxious" tended to learn faster and received fewer shocks before mastery of the avoidance response.

Although they were not especially focused on inter trial behavior, being thorough experimentalists, Solomon and Wynne did conduct their studies according to a protocol that required some recording of inter trial animal behavior, and this is most interesting from the standpoint of OCD. They noted the following description of behavior during the 3 min inter trial interval.

> There was a strong tendency for stereotyping to develop. For example, immediately after jumping a dog might position himself in a specific part of the apparatus, facing his body and head in a fixed direction, and he might maintain this position until the next presentation of the CS. In opposite compartments of the apparatus, such stereotyped behavior would often be symmetrical or mirror-image. When such a degree of stereotyping was observed, it was usual to find that many previously exhibited emotional signs were no longer evident (Solomon & Wynne, 1953, p. 15).

The experimenters did not draw what might in the current context be seen as an obvious connection between the stereotyped behavior and the absence of signs of anxiety, namely that the stereotyped behavior may have served the function of reducing anxiety.

These experimenters did however make the connection between the avoidance behavior of jumping and the concept of anxiety reduction.

> The reinforcement of learned avoidance instrumental responses comes about through drive reduction. Early in the learning process when the animal is escaping from shock, the instrumental act removes the US, as well as the CS. Drive reduction then consists of reduction in the intensity of both pain and emotional upset. Later, when the animal is avoiding the shock, drive reduction consists of reducing the intensity of the emotional upset by removing the CS (Solomon & Wynne, 1953, p. 15).

As it turns out, this more central conclusion about what maintained the avoidance behavior of jumping has become the more important conceptualization for OCD. The observations these investigators made about stereotyped behavior as ancillary findings in their experiments bore some surface resemblance to the rituals observed in obsessive-compulsive behavior of humans, but the more important lesson of these experiments lay in the explanation for why avoidance behavior failed to extinguish even hundreds of trials later when the animals were exposed to the CS with no subsequent shock. The resistance to extinction was a surprise to the investigators and led them to seek direct methods for extinguishing the jumping behavior.

Extinction and Resistance to Extinction

In their separate report on efforts to extinguish the jumping behavior of their dogs that had undergone the traumatic avoidance learning procedures, Solomon and his colleagues were rather more explicit in linking their work to some learning formulations of the "psychoneuroses" of the 1950s (Solomon, Kamin, & Wynne, 1953). They specifically noted that there might be an analogy between the persistence of their dogs' jumping behaviors and the persistence of certain neurotic behaviors in humans. Despite the fact that the neurotic behaviors seemed to be maladaptive or interfered with normal functioning, they persisted. At the end of their report on extinction experiments, the authors noted that the persistence of their dogs' jumping behaviors provided a picture "surprisingly akin to the clinical picture in compulsive neurosis" (p. 299).

To check their hunch that the avoidance behaviors they had trained were indeed highly unlikely to merely extinguish with repeated trials of the CS absent the shock, they observed a number of animals for extended periods of time. The pattern was clear and repeated. After as few as 11 trials where the CS had been paired with shock, the animals persisted in jumping when the CS was presented without shock, and they observed animals jump unabated for over 600 trials in some instances. They also observed that over time the latency to jump following presentation of the CS continued to decrease and remained stable at about 1.3 s. The animals jumped faster and faster until they reached an asymptote of 1.3 s whereupon they remained fast jumpers after hundreds of trials. They showed absolutely no signs of slowing down or stopping the avoidance response once it had been established.

The investigators also made several other distinctive observations about these attempts to achieve extinction through means that were considered ordinary or natural course extinction trials. They noted that during these extended ordinary extinction trials, the animals developed idiosyncratic styles of jumping, and they even referred to this phenomenon as the dogs developing their own individualized "rituals" (p. 293). They also noted that over time with the extended extinction procedures, the animals showed less and less of the emotional behavior observed during acquisition. In other words, the avoidance response became routinized and fairly automatic

without any sign of intense upset such as defecation, urination, or trembling. What started out as highly visible emotional reactions dissipated over time, and stable avoidance responding seemed to happen smoothly and routinely without any visible signs of emotional upset.

Having established that ordinary extinction procedures reliably failed to produce extinction of the jumping response, the Solomon group then tried two additional extinction procedures both singly and coupled together. They first attempted to punish the jumping behavior by turning on shock in the formerly safe compartment. In this procedure, the CS was presented and the animal jumped into the opposite compartment whereupon shock ensued immediately. Customary reasoning would lead to the hypothesis that such a punishment procedure ought to stop the jumping behavior. In fact, the opposite occurred. This jumping into shock procedure produced increased jumping behavior. After 100 trials of jumping into shock, 10 of 13 animals continued to jump into the shock. Moreover, they jumped faster into the shock than they had jumped into the "safe" compartment during ordinary extinction. The investigators speculated that this "punishment" procedure may have in fact functioned as a reconditioning procedure where the animals had in effect been subjected to further pairings of the CS with shock, thereby increasing their avoidance behavior rather than decreasing it.

In a second type of extinction procedure, the Solomon group used a glass barrier placed just on the other side of the gate between the two compartments. Tape was placed on the glass to make sure that the animals could discriminate when the barrier was present. The animals could see into the opposite compartment but they could not execute jumping because the avoidance response was prevented by the presence of the glass barrier. Over 10 days of this procedure, they concluded that some animals did stop the jumping behavior, but most did not. The glass barrier procedure did produce increased emotional responding in the presence of the CS. When the animals could not escape into the other compartment, they whined and panted more than on those ordinary extinction trials where the jumping response was available and not blocked.

What did in fact extinguish the jumping response was a combination of the glass barrier procedure with the shock punishment procedure. In this procedure, the animals had three trials where jumping resulted in jumping into shock, followed by four trials where the glass barrier was present. Of 16 animals subjected to this combined procedure, all but two stopped jumping within 7 days. Some of the animals had previously been exposed singly to either the shock punishment procedure or the glass barrier procedure, and when they were exposed to the combined procedure, those who had first been exposed to the glass barrier extinguished faster than those whose previous experience had been with the shock punishment only method. Prior exposure to the glass barrier condition seemed to afford some advantage to those animals as if it prepared them to respond to the shock punishment procedure when it was combined with the glass barrier procedure.

At the end of this report on various extinction procedures, Solomon and his colleagues provided the first formulation of what they later termed the anxiety conservation hypothesis (Solomon & Wynne, 1954). They noted that it would not be possible to claim that the jumping behavior of the dogs was motivated by the experience of anxiety when the CS was presented. The animals responded too quickly to be able to say that they first experienced anxiety and then jumped to reduce that anxiety. Such a

sequence may indeed have occurred during the acquisition of the jumping behavior, but once that behavior had been established, the fact that the animals jumped in less than 1.5 s meant there was not enough lapsed time for them to actually reexperience the anxiety that had been conditioned to the CS. In this way, the authors argued that the anxiety response that was indeed what motivated jumping had been conserved and in being so conserved, it was immunized from extinction.

> . . . Though, during ordinary extinction, with very short latencies of the instrumental act we see no emotional reactions, when the animals are later held in the presence of the CS by the glass barrier they demonstrate that the CS has *maintained its capacity to elicit anxiety*.
>
> But if the emotional response has not been extinguished, what has happened to it? The effect of early reinforcements has shortened the latency of jumping to the point where, since the dog's jump removes the CS so quickly, the conditioned emotional reaction may not be elicited at all! This would save the emotional reaction from extinction, since it is no longer exercised (Solomon et al., 1953, p. 299).

They also hypothesized that this conservation of anxiety contributed to the maintenance of avoidance responding by means of a type of cyclical reconditioning. In this formulation, the animal might on some occasions delay the jumping response long enough to experience some of the conserved anxiety; but if the animal then jumped, the net effect would be to strengthen the association between jumping and anxiety reduction. In this way, partial or intermittent delay of the jumping response led to a strengthening of the jumping response and not to its extinction. This idea of cyclical reconditioning was used to explain the extreme resistance to extinction observed in these animals. Left to a natural course, the animals would occasionally delay their jumping and reexperience the anxiety whereupon they then jumped. In effect, this was a reconditioning trial that intermittently reinforced jumping by anxiety reduction, and this intermittent reinforcement also contributed to stable and prolonged jumping behavior.

In addition to these core experiments and their theoretical formulation of anxiety conservation, Solomon and his colleagues also performed a series of experiments that have been less often cited but that are equally instructive for relating their work to OCD. This work focused on various physiological manipulations of their animals and parts of this work are described in what follows.

Additional Findings and OCD-Like Behaviors

In an effort to isolate the physiological substrates of their traumatic avoidance learning experiments, Wynne and Solomon (1955) collaborated with others to repeat aspects of their work on animals that had received surgery to alter their sympathetic nervous systems in such as way as to blunt or reduce the reactivity of their "emotional response systems." The surgeries were rather complicated and possibly crude by today's standards, but the logic of the studies was to see what would happen during both acquisition and extinction when the experiments were repeated on dogs that had damaged sympathetic nervous systems (Wynne & Solomon, 1955). They also employed limited drug procedures to provide additional blocking effects to the dogs' nervous systems. They reported results for 13 animals.

In terms of acquisition of the avoidance response, all 13 animals eventually acquired reliable jumping behavior in the presence of the CS, but they took longer to do so and often failed to show the characteristic reactions of normal dogs. In particular, the surgically altered animals showed many fewer signs of emotional upset during the acquisition phase of learning. They required many more trials to learn the avoidance response but appeared to go through the ordeal with much more calm than did their normal counterparts. They also showed very few of the stereotyped or ritualized patterns of jumping that had been observed in the normal dogs. All in all, and quite understandably in view of the state of their altered nervous systems, these surgically altered dogs showed much less of the "neurotic" behavior so typically seen in the normal animals that were previously studied and described in the above sections.

In contrast to their normal counterparts that withstood hundreds of extinction trials without ever stopping their jumping behavior, 8 of the 13 surgically altered animals showed spontaneous extinction of avoidance behaviors. Interestingly, two of the animals were trained in the customary traumatic avoidance learning and received their surgery after the training rather than before as did the others. For these two animals it was much more difficult to extinguish their jumping behavior than for those animals that had received surgery prior to acquisition of the jumping behavior. Wynne and Solomon clearly recognized that they could not make much from only two cases, but the results did suggest that acquisition of avoidance responses with an intact nervous system may have involved more than just sympathetic responding, and they speculated that central nervous system factors as well as hormonal systems might be involved in the preservation of such learned responses within a fully intact organism.

UTILITY AND LIMITATIONS OF THE SOLOMON WORK AS A MODEL FOR OCD

In resurrecting the Solomon et al. experiments of over 50 years ago, it is important to note both their enduring relevance to OCD as well as their limitations given our current understanding of OCD in humans. To their credit, Solomon and his colleagues never claimed to have produced an animal model of OCD. In fact, most of their references to OCD were aside comments that were typically marked with conservative modifiers such as "speculative" and "probable." On their own terms, these investigators seemed to see the stereotypies of some of their animals as analogous to ritualistic behaviors. Occasionally, they also drew inferences of a less superficial level when they alluded to the possibility that the function, not just the topography, of their animals' behaviors corresponded to what they suspected might be the function of compulsive rituals. In retrospect, that insight proved to be the one that became so important for the development of behavior therapies and behavioral models of OCD. It took the work of several clinicians to solidify the basic concept that compulsive behavior is a type of active avoidance behavior that is maintained by anxiety reduction. The relevance of the Solomon et al. dog experiments for OCD is that it specifies a functional model for how obsessions and compulsions are related to one another, and it specifies some of parameters to consider if the goal is to extinguish avoidance behavior that is maintained by anxiety reduction.

The limitations of the Solomon traumatic avoidance model for OCD are in fact numerous when viewed in the context of current research on human OCD. These studies on traumatic avoidance do not address etiology of OCD or the pathophysiology of the behavior.

The Solomon studies are likely not a model for how OCD develops in humans. The idea that a specific conditioning experience can lead to the onset of obsessive and anxiety arousing thoughts that are then neutralized by compulsive behavior seems highly unlikely. Only rarely are OCD patients able to report some primal incident that marked the onset of their behavior. Instead, the behavioral history is more typically one of increasing multiple obsessions and ritualistic compulsions. In this regard, the Solomon experiments are most relevant for suggesting models for extinguishing OCD behaviors and are less relevant for explaining how these behaviors get started. Some of their ancillary findings and informal observations are certainly consistent with some more recent work on human OCD. For example, the more "neurotic" dogs learned faster and were more difficult to extinguish. This is consistent with observations that individuals with OCD may be more generally nervous by temperament and may also be genetically influenced through a broad risk factor, rather than any very specific risk for OCD. On the basis of their limited study of animals with impaired central nervous system function and the fact that these animals were more difficult to condition, the Solomon work may also be consistent with such a generalized risk factor of anxiety. Animals that were more difficult to condition in terms of producing an exaggerated fear response were also the ones who took longer to acquire the avoidance behavior.

Although these investigators concerned themselves with the underlying physiological mechanisms for acquisition and extinction of conditioned emotional responses, their level of understanding of these matters was certainly crude by our current standards. They noted that temperament of certain animals seemed to play some role in how quickly they acquired the avoidance response and also in how long it took to extinguish the responses, yet they did not have the benefit of evidence suggested by human genetic studies or the capability of animal models that permit direct genetic manipulation.

ANIMAL MODELS THEN AND NOW: DIRECTIONS FOR FUTURE RESEARCH

When compared to more recent proposals for animal models of OCD, the Solomon experiments and their use in developing behavior therapies provide an interesting contrast. There is a fundamental difference of conceptualization about what constitutes an animal model of a human disorder from then to now. In the case of the Solomon experiments and animal experiments generally from that era, the aim was to provide an animal model of functional mechanisms based on learning principles. Today, animal models of OCD are not based so much on function as on visible similarity of outward behavior. Repetitive behavior, not repetitive behavior under certain conditions and brought about by certain mechanisms, has become the standard for animal models of OCD.

Current candidate models of OCD behavior in animals are consistent with a broadened definition of human OCD to include OCD spectrum disorders. This leads

to inclusion of any animal repetitive behavior that is apparently like some human repetitive behavior. Function is replaced by topography. Unlike the Solomon model, in the newer models of animal analogues of OCD, the conditions for performance of the behavior are shifted from the foreground to the background. Behaviors such as horse cribbing (chewing), pig gnawing, bird feather picking, cat hair pulling, and dog forelimb licking are said to be analogous to human eating compulsions, trichotillomania, and hand washing. There is some mention of the fact that these behaviors may occur in a context of conflict or distress, but the role of distress is minimized. In addition, the search for mechanisms has turned to genetics and brain physiology as opposed to conditioning and learning. All of that is, of course, to be welcomed as a broadening of the scope of animal models to include such obviously important levels of analysis. What is unfortunate is that those developments have occurred in large measure without acknowledgment of functional models such as the one forwarded by Solomon and his colleagues.

What could prove very useful in the future is to return to the Solomon type of preparation for studying the extinction of avoidance behavior and to also bring to bear on that type of study current hypotheses regarding genetic and physiological mediators of acquisition and extinction. This might produce better animal analogues from which to test the influence of various medications as protective mechanisms in the acquisition phase and as mediators and moderators of extinction during the equivalent of exposure and response prevention. That might bring us closer to a real animal laboratory for the study of OCD mechanisms.

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Reply to Houts:

A DYSFUNCTIONAL ANIMAL MODEL OF OCD

Nicholas H. Dodman

There is a notion in clinical psychiatry that obsessive-compulsive spectrum disorders simply arise spontaneously in anxious persons. A trichotillomania patient at Massachusetts General Hospital's Charlestown OCD clinic was considered to have developed her problem out of the blue with no specific antecedent stressors. On questioning, it emerged that her early life had been anything but stress free. It had been punctuated by such events as the death of a parent at an early age, continuous exposure to the remaining parent's alcoholism, and a precarious marriage, which precipitated the hair pulling. The hair-pulling abated only once, during a Caribbean cruise while she was away from her husband: The hair-pulling resumed 4 miles from Boston harbor on the return trip. Another trichotillomania patient at the same clinic started beard-pulling while immersed in an intensive postgraduate course at Harvard. The beard-pulling abated only once when the man extracted himself from the high stress situation during a hitch-hiking tour across Canada.

What seems to be missing from most studies of OCD is in-depth consideration of its etiology; not just genetics and susceptible personality type, but the often subtle pressures to which such individuals must be exposed for the ultimate expression of OCD. Sometimes the psychological pressures may be unclear, intentionally obscured, remote, or are low grade and chronic. In addition, potentially inciting circumstances are myriad, difficult to identify and categorize. This may be why it is difficult to associate environmental stressors with the development of OCD. It is the etiological aspect of OCDs that fascinates Houts in his revisitation of Richard L. Solomon's experimental research studies in dogs. Houts seems to view current veterinary models as superficial, examining the problem of OCD at face value and after its inception. His hope, by drawing peoples' attention to Solomon's research, is to focus their attention toward OCD's etiology and permanency and thus to open clinicians' minds to new therapeutic considerations. While the avoidance learning that Solomon's work details does possibly shed some light on the origins of OCD, it was not set up with this purpose in mind, and furthermore, its ends, however relevant, do not justify its means.

Houts first reviews OCD concepts in its historical context. He starts by discussing the origins of graduated in vivo exposure therapy with response prevention, and

thought-stopping techniques of interrupting the OCD behavioral chain. The idea, which at least in part, emanates from Solomon's work, is that OCD is a two-part process; ideation and enactment, obsession and compulsion, and that physical or cognitive interruption of the connection between these two processes can have therapeutic consequences. If one draws on Solomon's work with dogs to explain the origins of OCD, as Houts attempts to do, compulsive behavior propelled by an underlying thought process would seem to develop in connection with traumatic avoidance learning as a displacement or escape behavior. This idea has some merit, not that one needs to resort to Solomon's methods to illustrate it.

Building on Solomon's work, Meyer and Chesser (1970) postulated that the combination of an insoluble discrimination problem and noxious stimulation supposedly underlies the development of "fixated" stereotypic responses in animal models. Furthermore, the fixated responses persist even when the original dilemma is resolved. This may also be the case with human OCD and may explain why clinicians generally fail to make a connection between emotionally traumatic experiences and the expression of OCD. This is not to say that resolving any underlying dilemma is therapeutically immaterial, for it may well be that a successful therapeutic outcome cannot be achieved without addressing such background motivation. Houts is right to draw our attention to such historic and, perhaps, still relevant views. The irregular response of human OCD patients to SSRIs (Cartwright & Hollander, 1998; Hollander et al., 2002) might be explained in terms of successful or unsuccessful resolution of motivating causes.

According to Houts, Meyer and others' discuss the difficulty of extinguishing repetitive behaviors, even in the face of negative consequences. They report other studies in which, if special contingencies were arranged, it was possible to suppress repetitive responses learned and reinforced by traumatic avoidance and serving to reduce anxiety. In dogs with acral lick dermatitis, compulsive paw licking has been suppressed by electric shock aversive therapy (Eckstein & Hart, 1996). What is surprising regarding the latter study is that no new traumatic avoidance behavior was reported to develop in its place. We have seen compulsive tail chasing in a dog transmute into continuous room-circling as a result of punishment. Fortunately, the latter compulsion was more acceptable to the owner who regarded the treatment as successful.

Houts bemoans the fact that Solomon's traumatic learning experiments in dogs have been eclipsed by modern theories of OCD as an anxiety-driven behavior and by the cognitive revolution. He notes a change in philosophy from the circumstance-driven stress reduction concept to one more intimately connected to the human emotional experience as documented by verbal reporting. This change has led to a more cognitive-behavioral approach to therapy and a trend away from induced animal models. Animals are, after all, nonverbal, probably do not have insight into the dysfunctional nature of their repetitive behaviors, cannot express their anxiety, and are probably incapable of ego-dystonia.

Solomon's work is described in detail in the second section of Houts' chapter. As meaningful as these experiments may have been they were nonetheless hideous and inhumane. No conclusion justifies using experimental animals in such a way and such experimentation would not be approved by IACUCS today. Nevertheless, the experiments were performed and, though they should never be repeated, we can still learn from them, as Houts directs us to do. After describing the mechanical set up of what was a shuttle box for dogs, Houts goes into detail about the results

of the experimentation. We read about how all the dogs eventually learn to avoid shock by hopping across the partition dividing the two sides of the shuttle box. More interestingly, Houts draws our attention to Solomon's observations on the different temperaments and different responding of individual dogs. More anxious emotionally labile dogs, it seemed, learned faster how to avoid being shocked. Solomon also noted that some dogs developed stereotypies between trials, and the ones that did subsequently displayed less anxiety.

Another interesting point was the permanency of the avoidance learning seemingly paralleling the permanence of human OCD. Solomon's attempt to extinguish the behavior is where the experiment really became ugly. Punishing the dogs for a formerly correct avoidance strategy, rather than slowing the dogs down, actually increased the speed of their responding to the original cue. In attempting to extinguish the jumping response of the dogs, Solomon serially exposed the dogs to aversive therapy and then physical restriction. This combined method of extinction was relatively effective, though this finding itself was not particularly relevant to the clinical OCD therapy. However, the anxiety that the dogs showed when their learned response was physically prevented did parallel what would be anticipated in human OCD. In addition, their responding to avoid a negative experience helped dissipate their anxiety, though the anxiety was obviously conserved and sometimes surfaced during apparently intentional delayed responding, ensuring cyclical reconditioning.

Finally and also relevantly, Solomon demonstrated that the sympathetic nervous system was involved in (*a*) in the acquisition of avoidance learning, (*b*) the performance of stereotypies, and (*c*) the extinction of previously learned avoidance behaviors. Extrapolating from these results suggests that beta blockers may be useful adjuncts to other OCD therapies: Beta-blockers have, in fact, proven useful to augment OCDs only partially responsive to SSRIs (Dannon et al., 2000).

As Houts considers the utility and limitations of Solomon's work, with regard to its relevance to OCD, he seems to struggle to make the final connection because of what he sees as clear cut differences. For example, he points out that Solomon's dogs were exposed to acute physical and emotional dilemmas, whereas only rarely is such a history connected with the onset of OCD in human patients. He is also concerned about the sudden onset of the neurotic behaviors in the dogs as compared to the more gradual onset of obsessions and rituals in human OCD patients. He goes on to suggest that Solomon's work might be more relevant in terms of extinguishing OCD behaviors, as opposed to explaining how they arise. His opinion of the relevance of Solomon's work may be more pessimistic than necessary. When animals are confined and have their natural biological agendas thwarted, they do develop repetitive behaviors. Dogs exposed to chronic, irresolvable dilemmas of this type acquire ritualistic stereotypic behavioral responses, like self-licking and tail chasing, along a similar time course to humans with OCD. Caged birds pull out their feathers, horses bite the edge of their stalls (display cribbing), and big cats and bears in zoos pace mindlessly (Dodman et al., 1998). These behaviors arise as a result of chronic conflict as opposed to the acute dilemmas presented to Solomon's dogs.

In discussing animal models then and now, Houts expresses the opinion that current animal models of OCD are not so much based on function as phenomenology. Admittedly, it was visible similarities between the outward behavior of canine acral lick dermatitis and compulsive hand washing in humans that spawned a resurgence of interest in animal models of OCD but the phenomenology is not always comparable.

The features that are shared include genetic susceptibility, anxious personality type, initiation by conflict, and an assortment of repetitive species-typical behavior. As was the case with Solomon's mongrels, dogs susceptible to acral lick dermatitis do tend to be of more anxious, nervous disposition and seem to acquire their repetitive behavioral habit as a result of circumstances that, for a dog, could be construed as involving an irresolvable dilemma or conflict. While the onset of such behavior can be sudden and associated with an obvious precipitating event, in most cases it is not; the behavior being acquired more slowly over time, as occurs in human OCD. Houts may have been more excited about the application of Solomon's model if he had been aware that all spontaneous arising animal OCD's are created in anxious individuals subjected to acute or chronic psychological stress.

With Solomon's work and Houts' observation in mind, the clinical histories of the hair-puller and beard-puller narrated earlier assume a greater relevance. While psychiatrists may dismiss such life traumas in anxious individuals as "typical baggage" of life, when these pressures come to bear in susceptible individuals, they may well be responsible, in part, for the ultimate development of obsessions and compulsions. The idea that OCD develops as a displacement behavior to alleviate stress seems particularly apropos, whether one is considering Solomon's work, current veterinary models of OCD or, indeed, the human condition.

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Reply to Dodman and Shuster:

ANIMAL MODELS AND TWO TRADITIONS IN OCD RESEARCH

Arthur C. Houts

Literature on animal models of OCD reflects the two very different traditions in OCD research with humans. In commenting on the Dodman and Shuster chapter in this volume, it is important to place that chapter within a larger view of the state of the OCD research literature and the two predominant yet fairly distinct communities of scientists who make regular contributions to that literature. Having some appreciation for the state of affairs within a perspective on the history and philosophy of science may help to illuminate why investigators from two very different traditions can work along side one another and yet have great difficulty coming to terms with each other's evidence base and various claims based on their respective evidence bases.

In a now famous (or perhaps infamous depending on whom you ask) extended essay for a series of books entitled *Foundations of the Unity of Science*, Thomas Kuhn produced what became his most cited work, *The Structure of Scientific Revolutions* (Kuhn, 1962). The second edition of that work became one of the most widely cited works in psychology and the social sciences (Kuhn, 1970), and it has been frequently misused to argue that there is after all no such thing as unity in scientific disciplines but rather many self-interested and self-promoting groups that vie with one another to become the keepers of the "accepted" wisdom of a domain of knowledge. What Kuhn may have meant and what the implications of his work are for any vision of the unity of science or specific disciplines of science can now fill many library shelves, and the debates about normal science, paradigms, revolutionary science, and social influences on physics will continue well into the next century. For present purposes what matters most are Kuhn's discussions about incommensurability. Incidentally, it is worth noting that much of the inspiration for Kuhn's insights may have come from an obscure 1935 study of the history of the discovery of the syphilitic spirochete by Ludwig Fleck available at the time only in German and subsequently translated into English (Fleck, 1979). Fleck had noted the phenomenon of incommensurability in the various medical groups that were pursuing the causes of general paresis.

The concept of incommensurability was introduced by Kuhn to explain what he saw as the rather striking discontinuities in the history of science. He referred to these

periods of change as paradigm shifts or revolutionary periods of scientific change, and one has to be quite cautious about the meaning of his vexing terms. With that caveat in mind, however, it is useful to focus on the phenomenon of incommensurability because that very phenomenon is seen in the OCD animal and human literatures. In what follows, I will quote extensively from Kuhn (1970) to get across the phenomenon of incommensurability in his own words and hope that the reader can ignore some of the buzz words (paradigm, revolutionary science, etc.). I will then comment on the animal OCD literature to show how the phenomenon of incommensurability plays out there.

THE PHENOMENON OF INCOMMENSURABILITY

Kuhn (1970) introduced the term incommensurability to describe periods of scientific development where communities of investigators appeared to talk past one another:

> . . . proponents of competing paradigms will often disagree about the list of problems that any candidate for paradigm must resolve. Their standards or their definitions of science are not the same. . . .
>
> Within the new paradigm, old terms, concepts, and experiments fall into new relationships one with the other. The inevitable result is what we must call, though the term is not quite right, a misunderstanding between the two competing schools.
>
> In a sense that I am unable to explicate further, the proponents of competing paradigms practice their trades in different worlds. Both are looking at the world, and what they look at has not changed. But in some areas they see different things, and they see them in different relations one to the other. That is why a law that cannot even be demonstrated to one group of scientists may occasionally seem intuitively obvious to another (Kuhn, 1970, pp. 148–150).

Although, it is likely an exaggeration, the most striking aspect of incommensurability is the claim that scientists working from within different traditions seem to inhabit different worlds. The problems are not the same across different traditions, the priorities and methods are not the same, and there are even principles and "objects" in one tradition that cannot be found in the other. The similarities between this state of affairs and the current state of OCD research are striking.

INCOMMENSURABILITY IN OCD RESEARCH

As the current volume amply illustrates there are two predominant traditions within the OCD literature at the present time. I am deliberately avoiding the term paradigm because it is ambiguous and confusing due to the fact that most of the mental health field is far too underdeveloped to warrant the concept of paradigm analogous to what might be applicable for chemistry and physics. One tradition (and the one I have been affiliated with) is based on animal learning and interpretive extrapolations of animal learning to human OCD. For convenience, we can call this the learning and behavior therapy (LBT) tradition. Another tradition is based on a broader biological and physiological tradition that views human OCD as part

of a spectrum of disorders that may be linked via genetic mechanisms and common neurochemical and neurophysiological pathways that have been disrupted. Again for convenience we can call this the spectrum and physiological therapy (SPT) tradition.

Incommensurability is evident at the most basic level of defining the problem of OCD. In the LBT tradition, OCD is first and foremost an anxiety disorder where repetitive behavior is done to reduce anxiety (eg, Solomon's dogs). If there is no anxiety "motivating" the repetitive behavior, then there is no relevance to OCD. In contrast for the SPT tradition, repetitive behavior is the key feature of OCD, and the possibility is entertained that some behaviors are relevant to OCD even if it has not be shown that the behaviors are maintained by anxiety reduction (eg, canine acral lick, bird feather picking).

Incommensurability in the two traditions is also illustrated by the course of development from animal model to human therapy. For LBT, the trajectory of development was from animal model to human application, whereas for SPT, the effectiveness of medications was illustrated first with humans and afterward with animals. Therapists trying to develop behavior therapies for human patients looked back at the Solomon animal experiments for principles of therapy to apply to human OCD. In contrast for SPT, mental health professionals having observed improvements with clomipramine in humans with OCD went in search of animal models that might respond to clomipramine, and their search spawned a whole array of veterinary investigations using various drugs to alter repetitive behavior patterns believed to be linked to the OCD spectrum.

The ways that the two traditions used evidence from treatment interventions are quite different. The LBT tradition argues from evidence for the effectiveness of exposure and response prevention therapies that their formulation of OCD as behavior maintained by the negative reinforcement of anxiety reduction is the correct formulation even if it is not a satisfactory account of etiology. The SPT tradition argues from evidence for the effectiveness of SSRIs and other compounds that OCD is best regarded as part of a spectrum of disorders with dysregulation in various receptor types of the central nervous system. The LBT animal models have not been subjected to contemporary medication treatments, and the new SPT proposed animal models have not been subjected to the old extinction procedures of LBT models.

To the extent that both traditions acknowledge a role for anxiety in OCD behaviors, how they assign that role is quite different. In the LBT tradition, anxiety is the primary etiological component even if this tradition does not have a very good account of how anxiety in the form of obsessions gets started in the first place. In the SPT tradition, there is a general acknowledgement that anxiety plays a role in most of the behaviors that comprise the animal spectrum of OCD, but the role of anxiety is more along the lines of temperament and predisposing factors rather than as a specific etiological trigger for compulsive behavior. In the LBT tradition, stereotypy may arise from any number of causes such as amphetamine induced rat stereotypy to schedule induced polydipsia. In contrast, in the SPT tradition, the characterization of licking, tail chasing, and feather picking as stereotypes is rejected because these are observed in nervous animals under conditions of stress, which context makes them relevant to OCD and not merely interesting but otherwise irrelevant stereotypies (see Dodman and Shuster, Chapter 3) as in the LBT tradition.

RESOLUTION OF INCOMMENSURABILITY

One of the features of Kuhn's analysis that rationalist philosophers of science found most irritating was Kuhn's account of how incommensurability gets resolved. Kuhn was very explicit in stating that the resolution of incommensurability was not a matter of logic: "The competition between paradigms is not the sort of battle that can be resolved by proofs" (Kuhn, 1970, p. 148). Even casting the situation as a "battle" made it sound like a military conflict where the side with the most power overcomes the side with the least power. Elsewhere Kuhn likened the change from one set of ideas to another as a type of conversion experience with echoes of religious experience and sudden change of perception as in gestalt shifts between figure and ground. He even averred that scientific change occurred and incommensurability got resolved because older scientists died and new ones took their place. In this regard, he quoted from Max Planck's autobiography: "a new scientific truth does not triumph by convincing its opponents and making them see the light, but rather because its opponents eventually die, and a new generation grows up that is familiar with it" (cited in Kuhn, 1970, p. 151). What does all of this portend for the future of incommensurability in the animal and human OCD research literature?

I suppose we could have the members of the two traditions, many of whom are authors of chapters in this volume, meet some weekend in the backwoods of Michigan or Minnesota for a serious contest of paintball and see who gets to continue publishing and who must cease and desist according to who wins the game. Alternatively, we could wait for natural selection to run its course and see who survives in the game of research support and publishing and perishing, but this could take quite a while to play out as both sides are evidently well supported and quite productive in terms of papers and books. Religious type conversion seems out of the question for most barring some very remarkable genetic and/or physiological findings or results from some experiment that most would consider unethical to perform on humans.

What is more reasonable and also more likely to happen is that both traditions will carry on as they currently do in relative isolation from one another. They can continue to come together from time to time as in this volume and to take stock of their respective current state of affairs. The dialogue across traditions, to the extent this is possible, may foster some new challenges and collaborations. It would indeed be fascinating to see if the Solomon type of experiments could be repeated and to observe the effects of various SSRIs and other compounds on extinction of learned avoidance responses as well as their effects on preventing or modulating ancillary ritualistic behaviors during acquisition of learned avoidance. Likewise, it would be interesting to see how dogs blocked from paw licking would respond after prolonged blocking analogous to response prevention.

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Chapter 5

THE CASE FOR THE OCD SPECTRUM

Eric Hollander, Jennifer P. Friedberg, Stacey Wasserman, Chin-Chin Yeh, and Rupa Iyengar

Obsessive-compulsive disorder (OCD) is an anxiety disorder with an often chronic course that is estimated to have a lifetime prevalence rate of 1.9–3% in the United States (American Psychiatric Association, 2000). However, a substantially greater percentage of the population has symptoms that overlap with OCD and may be included within the so-called obsessive-compulsive spectrum disorders (OCSDs). OCD and OCSDs are characterized by obsessions, defined as recurrent and intrusive thoughts, impulses, or images that cause marked distress, and/or compulsions, which are repetitive behaviors performed in response to an obsession (American Psychiatric Association, 2000; Hollander & Wong, 1995a). As is shown in Figure 5.1, OCSDs may be subdivided into three basic clusters: (1) neurological disorders with repetitive behaviors, (2) impulse control disorders, and (3) body image, body sensation, and body weight concern disorders.

Shared features of OCDs and OCSDs include similarities in age of onset, comorbidity, clinical course, family history, underlying neurobiology, and treatment response. As is shown in Figure 5.2, OCSDs may also fall along a continuum of compulsivity and impulsivity. Compulsivity can be conceptualized as being driven by the need to decrease discomfort through repetitive behavior or rituals, while impulsivity may be viewed as being driven by the need to maximize pleasure or stimulation (American Psychiatric Association, 2000). While the drives behind them differ, disorders of compulsivity and impulsivity have in common the inability to delay or inhibit repetitive behaviors (Hollander & Benzaquen, 1997).

In this chapter, we first describe the various disorders proposed as part of the OC spectrum. Next, we present empirical evidence that forms the basis for the spectrum approach.

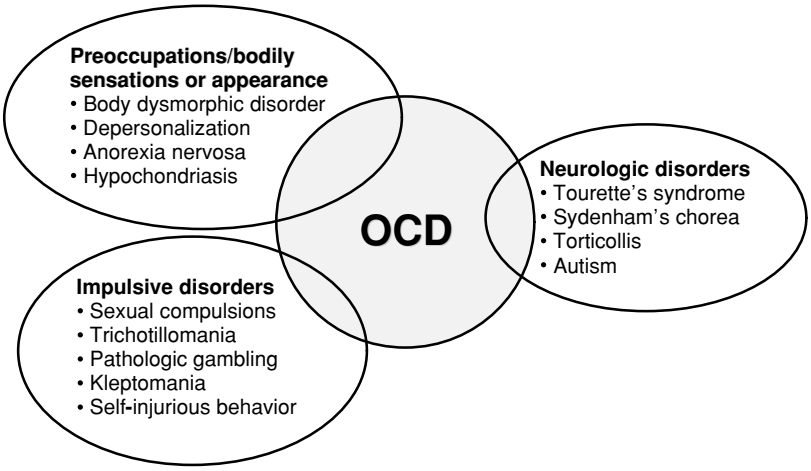

FIGURE 5.1. Obsessive-compulsive spectrum disorders.

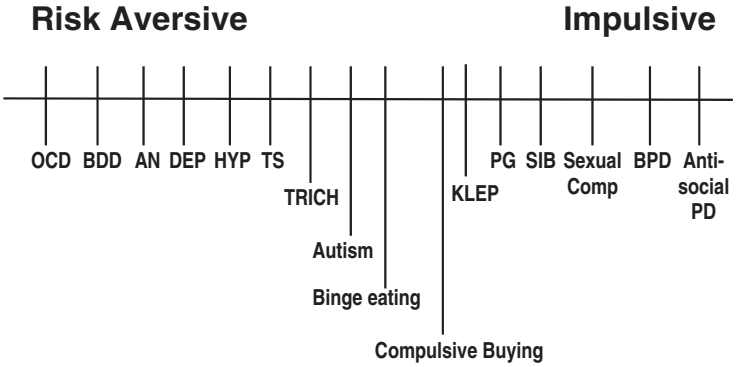

FIGURE 5.2. A dimensional approach to compulsivity and impulsivity. OCD = obsessive-compulsive disorder; BDD = body dysmorphic disorder; AN = anorexia nervosa; DEP = depersonalization; HYP = hypochondriasis; TS = Tourette's disorder; TRICH = trichotillomania; KLEP = kleptomania; PG = pathological gambling; SIB = self-injurious behavior; BPD = borderline personality disorder.

CLUSTERS OF THE OBSESSIVE-COMPULSIVE SPECTRUM

Neurological Disorders with Repetitive Behaviors

This group of OCSDs includes autism, Tourette's syndrome (TS), and Sydenham's chorea. Functional disturbances in the basal ganglia, which may result in the development of repetitive and stereotyped behavior, are often implicated in these disorders (Dale, 2003; Peterson et al., 2003; Sears et al., 1999). OCD patients have a higher incidence of tics and other neurological soft signs than do non-OCD patients, thus suggesting a link between OCD and neurological dysfunction (Aronowitz et al., 1994; Hollander et al., 1990). This cluster generally involves mostly motorically driven repetitive behaviors and few obsessions. However, the nature of the obsessions and compulsions in neurological disorders differs from those in OCD.

Autism is characterized by stereotypic complex hand and body movements, craving for sameness and rigid routines, motorically driven rituals and routines, and social and language deficits. In addition, narrow restricted interests or obsessive pursuits tend to be related to how things, rather than people, work (Baron-Cohen & Wheelwright, 1999). One study suggests that autistic patients tend to experience fewer aggressive, contamination, sexual, religious, symmetry, and somatic obsessions than OCD patients but greater compulsive behaviors such as repetitive ordering, hoarding, telling/asking, touching, and self-injurious behavior (McDougle et al., 1995). Behaviors related to checking, cleaning, and counting were less common among autistic patients than among OCD patients.

Patients who suffer from TS have motor and vocal tics characterized by sudden repetitive movements, gestures, or utterances (Leckman, Peterson, Pauls, & Cohen, 1997). Obsessions and compulsions also typically occur in TS, but the content of these thoughts and behaviors may differ from OCD obsessions and compulsions. Individuals with TS report having more obsessions and compulsions related to mental play, echophenomena, touching, and self-injurious behavior, whereas individuals with OCD tend to have more aggressive and contamination obsessions and washing compulsions (Cath et al., 2001). It has been suggested that sensory phenomena such as bodily sensations, mental urges, and physiological tension are more common among TS patients than among OCD patients, and that repetitive behaviors in TS are performed in response to these phenomena (Miguel et al., 2000).

Sydenham's chorea, a neurological disorder that occurs in association with rheumatic fever, is characterized by involuntary and uncoordinated movements, muscular weakness, concentration difficulties, emotional lability, and slurred speech (Faustino et al., 2003). In addition, approximately 75% of individuals with Sydenham's chorea experience violent, aggressive, or contamination-related obsessions and checking, washing, and ordering compulsions (Snider & Swedo, 2003). This association between Sydenham's chorea and obsessive-compulsive behavior has led to speculation that a subgroup of childhood-onset OCD may occur as a result of streptococcal infections (Swedo, Leonard, & Kiessling, 1994). The term pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection (PANDAS)

has been used to describe children who develop obsessive-compulsive and/or tic symptoms after a streptococcal infection but do not meet criteria for Sydenham's chorea (Swedo et al., 1998). There are five clinical characteristics of PANDAS: (*a*) presence of OCD and/or tic disorder, (*b*) pediatric onset (from age 3 to puberty), (*c*) acute onset and dramatic symptom exacerbation, (*d*) neurological abnormalities such as choreiform movements or motor hyperactivity, and (*e*) a temporal association with group A beta-hemolytic streptococcal infections (Heubi & Shott, 2003). Individuals in the PANDAS subgroup tend to be male and to have an unusually young age of onset, and obsessive-compulsive symptoms including contamination fears, somatic concerns, and rituals involving washing, checking, counting, and repeating that tend to be similar to the symptoms of childhood-onset OCD (Swedo et al., 1998).

Impulse Control Disorders

Another hypothesized category of OCSDs is impulse control disorders, which are characterized by the inability to resist impulses that have negative consequences. These impulses often increase tension or arousal and provide pleasure, gratification, or release during the act (McElroy, Harrison, Keck, & Hudson, 1995). Impulse control disorders include pathological gambling, intermittent explosive disorder, trichotillomania, kleptomania, sexual compulsions, compulsive buying, and self-injurious behavior. Males and females tend to have different impulse control disorders. Pathological gambling and sexual compulsions are more common among men, while kleptomania, trichotillomania, and compulsive buying are more common among women (Black, Kehrberg, Flumerfelt, & Schlosser, 1997; Christenson et al., 1994a; Christenson, MacKenzie, & Mitchell, 1994b; Lesieur, 1988; McElroy, Hudson, Pope, & Keck, 1991). Though these disorders are impulsive in nature, they also possess a compulsive component in that often the behaviors become driven as the individual experiences guilt and disgust resulting from his or her actions, and the goal of the behaviors is to reduce anxiety (Hollander & Wong, 1995b).

Pathological gambling is an impulse control disorder characterized by recurrent gambling thoughts and behaviors that significantly disrupt the patient's social and occupational functioning (Sood, Pallanti, & Hollander, 2003). In order to be diagnosed with pathological gambling, an individual must indicate a loss of control over gambling behavior, demonstrate a progressive increase in frequency, amount of money gambled, time spent thinking about gambling and obtaining money to gamble, and be unable to resist the impulse to gamble even when it has negative ramifications on his or her life. Although pathological gambling is conceptualized as an impulse control disorder, there is evidence of some degree of obsessive thoughts about gambling and compulsive behaviors in pathological gamblers. In addition to gambling behaviors, pathological gamblers may exhibit obsessions, compulsions (particularly hoarding behavior), and avoidance behavior (Frost, Meagher, & Riskind, 2001). Pathological gamblers also appear to exhibit higher levels of obsessionality than do healthy controls (Blaszczynski, 1999).

Sexual obsessions, compulsions, and paraphilias may also be included under the category of impulse control disorders. Paraphilias are defined as repetitive sexual fantasies and acts that involve arousal from inappropriate objects or persons (American Psychiatric Association, 2000). Paraphilias and OCD have similar ages of onset, and

paraphilias are similar to obsessions in that they are intrusive but differ in that they are not usually ego-dystonic (Bradford, 2001). Compulsive masturbation and other sexual behaviors of paraphilic patients are also similar to the compulsive rituals seen in OCD patients. In contrast, nonparaphilic sexual compulsions involve sexual behaviors that are culturally acceptable, but which have a frequency and intensity that may interfere with sexual intimacy. Both paraphilic and nonparaphilic sexual compulsions are associated with sexual gratification and are likely to be enacted. True sexual obsessions, on the other hand, are intrusive and vivid sexual images that are anxiety provoking and repugnant to the individual, and are not usually enacted (Hollander & Wong, 1995a, 1995b).

Kleptomania is an impulse control disorder characterized by recurrent, impulsive, pathological stealing which results in significant functional impairment. As in OCD, resistance to stealing is met with anxiety and tension (Dannon, 2003).

Compulsive buying is defined as being uncontrollable, markedly distressing, time-consuming, and/or resulting in problems in social and occupational functioning (McElroy, Keck, Pope, Smith, & Strakowski, 1994). Compulsive buying is preceded by a buildup in tension, and the behavior frequently results in guilt and financial problems (Christenson et al., 1994a, 1994b; Lejoyeux, Ades, Tassain, & Solomon, 1996). It has been speculated that compulsive buyers exhibit behaviors that are consistent with both OCD and impulse control disorders.

Trichotillomania is characterized by recurrent pulling out of one's hair with increased tension before pulling or when attempting to resist the urge to pull (O'Sullivan, Mansueto, Lerner, & Miguel, 2000). This pulling behavior may result in noticeable hair loss. Trichotillomania and OCD patients report similar degrees of lack of control of, and resistance to, their hair-pulling/compulsive behaviors (Tukel, Keser, Karali, Olgun, & Calikusu, 2001). Trichotillomania may be classified as an impulse control disorder but also can be thought of as a tic disorder since its treatment response and phenomenology is more similar to that of TS than it is of the other impulse control disorders (van Ameringen, Mancini, Oakman, & Farvolden, 1999).

Body Image, Body Sensitization, and Body Weight Concern Disorders

The final category of proposed OCSDs is characterized by a preoccupation with the body. Disorders in this category include two somatoform disorders: hypochondriasis and body dysmorphic disorder (BDD); two eating disorders: nervosa and binge eating disorder; and depersonalization disorder.

In hypochondriasis, the patient is often obsessed with having a physical illness despite lack of medical evidence (Fallon, Qureshi, Laje, & Klein, 2000). Hypochondriasis is similar to OCD in that hypochondriacal fears about illness are repetitive, intrusive, and cause marked distress (Fallon, Javitch, Hollander, & Liebowitz, 1991). Hypochondriacal compulsions are repetitive and performed in order to reduce distress, and often take the form of checking one's body, checking with others, or visits to medical professionals for reassurance (Fallon et al., 2000).

Body dysmorphic disorder is characterized by preoccupation with an imagined flaw in appearance or excessive distress about a slight imperfection. The perceived defect may be specific to a body part, especially on the face, or it may be anxiety over a general ugliness (Allen & Hollander, 2000). Body dysmorphic disorder can

lead to impairment in functioning as well as to psychiatric hospitalization, suicidal ideation, and suicide attempts (Phillips, McElroy, Keck, Hudson, & Pope, 1994). Like OCD patients, individuals with BDD have obsessive thoughts about their appearance and engage in compulsive checking behaviors such as mirror checking, attempts to conceal the defect with clothing or makeup, and repetitive cosmetic surgery. Both BDD and OCD have numerous other similarities, including age of onset and chronic course (Phillips, 1996). However, BDD patients typically have less insight than do OCD patients (Simeon, Hollander, Stein, Cohen, & Aronowitz, 1995).

Anorexia nervosa is characterized by disturbance in body image, obsessive fears of being fat, and compulsive, driven attempts to reduce weight via restricted eating, abuse of laxatives and/or diet pills, and excessive exercise (Hsu, Kaye, & Weltzin, 1993). People with anorexia nervosa also appear to exhibit obsessions and compulsions that are not related to eating or weight, including symmetry and contamination obsessions and ordering and hoarding compulsions (Halmi et al., 2003; Matsunaga et al., 1999).

Individuals who suffer from bulimia nervosa also perceive themselves as being overweight and are preoccupied with body image. Bulimic patients often exhibit episodes of binging followed by periods of purging, which includes compulsive behaviors such as self-induced vomiting and excessive exercise (Favaro & Santonastaso, 1998; Penas-Lledo, Vaz Leal, & Waller, 2002). It has been argued that, while behaviors such as excessive exercise are compulsive, behaviors like laxative abuse are impulsive because they are self-injurious and involve a self-punishing impulse (Favaro & Santonastaso, 1998).

In contrast, binge eating disorder involves the compulsive eating of excess amounts of food without any compensatory behaviors to prevent weight gain (Spitzer et al., 1993). Binge eating disorder is also characterized by depressive symptoms and body image distress, and may affect either obese or nonobese individuals (Devlin, Goldfein, & Dobrow, 2003).

Depersonalization disorder, which is characterized by recurrent experiences of unreality that cause marked distress and/or functional impairment without any loss of reality testing, has also been proposed to be an OCSD (Simeon et al., 1997; Simeon, Stein, & Hollander, 1995).

THE CASE FOR THE OBSESSIVE-COMPULSIVE SPECTRUM

Comorbidity

One of the best indications that the aforementioned disorders may belong within a spectrum of related disorders is the frequency of comorbidity among these disorders. In other words, patients with OCD often exhibit symptoms of other OCSDs and vice versa.

Neurological OCSDs have been found to be highly comorbid with OCD. A longitudinal study of 101 children with TS found that 50% of the patients already had comorbid OCD, and furthermore 8% of children without a comorbid psychiatric diagnosis at the time of their initial evaluation developed OCD during the observational period of the study (Park, Como, Cui, & Kurlan, 1993). Although the comorbidity

rates of OCD among autistic individuals have been previously reported to be very low, these two disorders may be highly comorbid.

Patients with impulse control disorders also have a high OCD comorbidity rate, though some impulse control disorders may be more comorbid with OCD than others. There has been a noticeable increase in diagnosis of comorbid OCD among trichotillomania patients (Christenson & Crow, 1996). One recent study found a prevalence rate of 30.4% for OCD among trichotillomania patients (Lochner, Simeon, Niehaus, & Stein, 2002), while another reported an OCD prevalence rate of 14.9% (du Toit, van Kradenburg, Niehaus, & Stein, 2001a). A study investigating comorbid OC spectrum disorders among OCD patients found that the OC spectrum disorders with the highest prevalence rates were both impulse control disorders, compulsive self-injury (22.4%) and compulsive buying (10.6%) (du Toit, van Kradenburg, Niehaus, & Stein, 2001b). The evidence of elevated comorbidity rates of OCD among pathological gamblers and sexual compulsions is much less conclusive. One epidemiological study reported a relative risk of 7.2 for OCD among pathological gamblers (Bland, Newman, Orn, & Stebelsky, 1993), while another study did not find an elevated odds ratio for OCD in pathological gamblers (Cunningham-Williams, Cottler, Compton, & Spitznagel, 1998). A recent study of OCD patients found that the prevalence of pathological gambling and sexual compulsions among OCD patients did not differ from that of controls (Jaisoorya, Reddy, & Srinath, 2003).

The key to understanding the link between OCD and impulse control disorders such as pathological gambling may lie in the subset of patients who suffer from comorbid attention-deficit/hyperactivity disorder (ADHD). ADHD may affect more than 20% of pathological gamblers (Specker, Carlson, Christenson, & Marcotte, 1995). Similarly, up to 30% of children and adolescents with OCD also have ADHD (Geller, Biederman, Griffin, Jones, & Lefkowitz, 1996). Tic disorders and ADHD may also be related. A longitudinal study comparing ADHD boys to controls found that the boys with ADHD exhibited more tic disorders than did the controls (Spencer et al., 1999). It has been suggested that the coexistence of OCD and ADHD may represent a distinct OCD subtype (Geller et al., 2003).

Studies suggest that somatoform disorders and OCD are highly comorbid. Two large-scale studies of BDD patients reported a lifetime OCD comorbidity rate of 30% (Gunstad & Phillips, 2003; Phillips & Diaz, 1997), while another study found that 14.5% of patients initially diagnosed with OCD had comorbid BDD (Phillips, Gunderson, Mallya, McElroy, & Carter, 1998). A recent study investigating the comorbidity of OCD and a variety of OCSDs reported that prevalence of BDD and hypochondriasis was significantly greater in OCD patients than in healthy controls (Jaisoorya et al., 2003).

There is also a relatively high degree of comorbidity for eating disorders and OCD. One study found an OCD comorbidity rate of 18.2% among patients with anorexia nervosa and bulimia, and further suggested that individuals with comorbid OCD and eating disorders had a higher lifetime prevalence rate than eating disordered patients without OCD (Lennkh et al., 1998). It may be that anorexia is more highly comorbid with OCD than is bulimia, as one study reported that 37% of anorexics and only 3% of bulimics met criteria for OCD (Thornton & Russell, 1997). However, other studies have suggested a higher comorbidity rate for bulimics. A study of bulimics found a lifetime prevalence rate of 32% for OCD and 24% for subthreshold OCD (Rubenstein et al., 1993).

Family History

Findings from family studies suggest that OCD may have a familial or genetic association with several but not all OCSDs. A recent study found an elevated prevalence rate of BDD, hypochondriasis, and pathological grooming conditions (such as trichotillomania, skin picking, and nail biting) among first-degree relatives of OCD patients as compared to relatives of case controls (Bienvenu et al., 2000). However, this study did not find elevated prevalence rates of eating disorders or impulse control disorders such as kleptomania, pathological gambling, or pyromania among relatives of OCD patients.

Though this study did not find a familial link between eating disorders and OCD, other studies have suggested that relatives of anorexics and bulimics do in fact have elevated lifetime prevalence rates of OCD. First-degree relatives of anorexic patients had significantly higher lifetime rates of OCD and obsessive-compulsive personality disorder as compared to relatives of bulimics and healthy controls (Lilenfeld et al., 1998). Another study found that the morbidity risk for OCD and tic disorders was higher among first-degree relatives of patients with anorexia and bulimia than among relatives of comparison patients (Bellodi et al., 2001).

Neurological disorders also appear to have a familial relationship to OCD. Relatives of OCD patients had a greater lifetime prevalence of tic disorders including transient tic disorder, chronic motor/vocal tics, and TS than did relatives of control subjects (Grados et al., 2001). Recent studies have suggested that two dimensions of OCD—aggressive, sexual, and religious obsessions and checking compulsions, and symmetry and ordering obsessions and compulsions—are transmitted within families, and it appears that these two dimensions are also correlated among sibling pairs who have been diagnosed with TS (Leckman et al., 2003). One study found that OCD and motor tics were significantly more common among relatives of autistic patients than among relatives of Down's Syndrome patients (Bolton, Pickles, Murphy, & Rutter, 1998). Furthermore, this study found that family members with OCD were significantly more likely to display impairments in social functioning and communication skills that were consistent with autism. The occurrence of OCD or obsessive-compulsive traits in parents of autistic children is significantly more likely if autistic children have a high occurrence of repetitive behaviors (Hollander, King, Delaney, Smith, & Silverman, 2003). One study of autism multiplex families suggests that there is an autism susceptibility gene on chromosome 1 and chromosome 6 that is associated with the severity of obsessive-compulsive behaviors (Buxbaum et al., in press).

Recent animal genetic studies have further supported the hypothesis that OCD and OCSDs have a genetic component. A study by Greer and Capecchi (2002) demonstrated that mutations in the *Hoxb8* gene in mice result in excessive grooming and hair removal consistent with the behavior of humans with trichotillomania. It is possible that the excessive grooming behaviors, ritualistic cleaning, and self-injurious behavior characteristic of many OCSDs is also a result of genetic mutations (Graybiel & Saka, 2002).

Neurocircuitry

Functional neuroimaging studies have elucidated the neurocircuitry of OCD, and yet few studies have defined the functional neurocircuitry of OCSDs. This section aims to examine similarities in the neurocircuitry of OCD and that of OCSDs.

OCD is believed to involve alterations in the metabolic activity of the orbitofrontal-subcortical circuits, which contain both a "direct" and an "indirect" pathway (Saxena & Rauch, 2000). The direct pathway projects from the cerebral cortex to the striatum, to the internal segment of the globus pallidus-substantia nigra pars reticulate (the main output of the basal ganglia), and back to the cortex. The indirect pathway also begins in the cortex and leads to the striatum, to the external segment of the globus pallidus, and then to the subthalamic nucleus, to the globus pallidus-substantia nigra pars reticulata, to the thalamus, and then back to the cortex. The direct pathway functions as an activator of the thalamic system, engaging it in a positive-feedback loop. The indirect pathway, in contrast, provides negative feedback. It is believed that these pathways balance each other and allow facilitation and suppression of complex motor programs.

OCD symptomatology includes a preoccupation with danger, violence, hygiene, order, and sex. These symptoms are believed to be mediated by the orbitofrontal-subcortical circuits and may involve an imbalance between the direct pathway and the indirect pathway. A lack of inhibition of the direct pathway (ie, hyperactivity of the thalamic system) may cause the patient to become obsessed with the danger, violence, hygiene, so forth, and to respond with ritualistic behavior. Some evidence points to structural abnormalities in the striatum as a possible cause for dysfunction (Bartha et. al., 1998). When the levels of *N*-acetylaspartate, a cerebral metabolite, was compared between 13 OCD patients and 13 healthy controls, it was found that the *N*-acetylaspartate levels from the left corpus striatum were significantly lower in patients with OCD. The results indicate reduced neuronal density in the striatum of OCD patients.

OCD not only involves the basal ganglia, but the limbic system as well. As discussed elsewhere in this chapter, OCD patients experience anxiety and fear and often must perform compulsive and ritualistic acts to relieve that tension. This symptomatology suggests that the limbic system, which is involved in emotions, also participates in the neurocircuitry of OCD. One symptom provocation study involving functional MRI showed that OCD patients showed significant amygdala activation after being confronted with visual representations of fearful expressions (Breiter & Rauch 1996, Breiter et al., 1996).

Similar abnormalities have been found in OCSDs and suggest that symptoms of these disorders arise from defects in the same area of the brain. A recent study examining clinical, laboratory, psychiatric, and magnetic resonance findings in patients with Sydenham's chorea (Faustino et al., 2003) revealed that patients with this disorder had alterations to their basal ganglia. The location of these changes is consistent with changes that were found in patients with OCD. Sydenham's chorea patients were also shown to have signal hyperintensity in the head of caudate nuclei. Furthermore, 3 of the 19 patients had prolonged duration of chorea, recurrence of the symptoms of the disorder, and suffered from a permanent lesion in the basal nuclei. The study also revealed that no correlation existed between lesion persistence and the severity of the chorea.

Sydenham's chorea is not the only tic disorder to show abnormalities in the basal ganglia. Another study showed that children with PANDAS had changes in their neuroanatomy similar to that of Sydenham's chorea (Giedd, Rapoport, Garvey, Perlmutter, & Swedo, 2000). This study hypothesized that the pathophysiology for children with PANDAS involved antibodies that were directed toward the group A beta-hemolytic bacteria cross-reacting with the basal ganglia of genetically

susceptible hosts, ultimately leading to OCD and other tic disorders. In the study, the MRIs of 34 children in whom there existed a relationship between neuropsychiatric symptoms and streptococcal infection were compared with those of 82 healthy subjects. The results revealed that components of the basal ganglia, such as the caudate, putamen, and globus pallidus, were enlarged in the PANDAS group. These results were consistent with the hypothesis of antibody-mediated inflammation of the basal ganglia being the cause of poststreptococcal OCD or tics. As with Sydenham's chorea, there was no correlation between basal ganglia size and symptom severity.

Gilles de la Tourette syndrome is another tic disorder that has been shown to have structural abnormalities similar to OCD. Recently, Peterson et al. (2003) studied abnormal anatomical characteristics of patients with TS using a large sample size to decrease error. Previous studies had not been very robust, possibly due to a small sample size or limitations of MRI. In this study, however, basal ganglia volumes were measured in 154 children and adults with TS and 130 healthy controls using high resolution MRI. The study found that volume of the caudate nucleus was decreased across all age groups in patients with TS, while the volumes of the putamen and globus pallidus nuclei were decreased on average only in adults. As with findings with Sydenham's chorea and PANDAS, the basal ganglia volumes did not significantly correlate with the severity of the tic symptoms.

Symptoms characteristic of autism, which maybe categorized as lying within the neurological cluster of OCSDs along with Sydenham's chorea and TS, likewise may be associated with basal ganglia dysfunction. Like OCD and TS, autism is characterized by stereotyped and repetitive behaviors, such as compulsive rituals and difficulties with change in routine or environment. Sears et al. (1999) used MRI to study basal ganglia abnormalities in autistic patients. In this study, the MRIs of 35 autistic subjects were compared to those of 36 healthy volunteers. The results showed significant enlargement of the caudate, although there was no significant difference in mean symmetry. The MRIs were also examined for any correlation between caudate volume and clinical features. The results showed that ritualistic or repetitive behavior, but not social or communication, significantly correlated with caudate volume.

Other OCSDs that do not involve tics also show similar abnormalities in the basal ganglia. Rauch et al. (2003) examined the validity of BDD as either an OCSD or an affective disorder. The authors hypothesized that if BDD was an OCSD, the MRIs of the patients should exhibit abnormal striatal and white matter volumes, as well as a shift in asymmetry. If BDD was an affective disorder, the MRIs should reveal reduced hippocampal volumes. The study compared the MRIs of eight women with BDD with that of eight healthy subjects. The BDD group showed a leftward shift in the caudate nucleus asymmetry and an increased total white matter volume. There were differences between the results of this study and the results of previous studies involving OCD. Previous studies had shown OCD patients to exhibit a reduced white matter volume and a rightward shift of the caudate nucleus, the opposite of that shown in BDD patients. However, as the authors pointed out, BDD and OCD are not identical disorders and these results suggest that BDD is an OCSD as originally hypothesized.

Eating disorders, such as anorexia nervosa, likewise exhibit basal ganglia abnormalities that suggest these illnesses also lie on the obsessive-compulsive spectrum. The data from one study indicated that anorexia nervosa might be linked to both

OCD and tic disorders that result from autoimmune processes (Havel, Hallett, Riggs, Vaz, & Kiessling, 2001). The study examined whether antiputamen antibodies were present in 22 adolescents with anorexia nervosa. The results showed that 27% of the girls had levels antibody levels greater than two standard deviations from the mean. These findings were similar to findings in movement disorders, such as TS, and also suggested that some anorexic nervosa patients suffered an autoimmune response my like PANDAS patients.

Anorexia nervosa further resembles OCD in its involvement of the limbic system. The limbic system, and specifically the amygdala, is concerned with feelings of fear. One study hypothesized that anorexia nervosa activates the "fear network" centered in the amygdala and examined the effects that body image distortions have on the amygdala (Seeger, Braus, Ruf, Goldberger, & Schmidt, 2002). Using functional MRI, the study compared three females diagnosed with anorexia nervosa with three healthy controls. The participants were shown distorted body images, such as increased thigh, stomach, or breast circumference, and their brains were monitored for activity in the amygdala. The results showed that AN patients exhibited right amygdala activation when confronted with their own distorted body images. These results suggest that the fear network is activated, leading to a preoccupation of body weight seen in patients with this disorder.

Impulse control disorders have also shown similar neuroanatomical abnormalities as OCD. Ritualistic behavior in OCD that appear to result from corticostriatal abnormalities have led researches to question whether impulsive behavior in impulse disorders also have roots in structural defects. One preliminary study examined the validity of that hypothesis in patients with trichotillomania, which is characterized by uncontrolled hair-pulling (O'Sullivan et al., 1997). The morphometric MRIs of 10 patients with trichotillomania were compared to those of 10 healthy controls, and the study showed that trichotillomania patients exhibited significantly decreased left putamen and left lenticular nuclei volumes. However, the data also suggest that these differences could be attributed to volumetric differences rather than structural abnormalities, though the small sample size could be the reason for these results.

Neurotransmitter Function

In addition to structural abnormalities, OCD and OCSDs also exhibit abnormalities in neurotransmitter function. In a normal human brain, excitatory axons predominantly use glutamate as a neurotransmitter, while inhibitory ones mainly utilize gamma aminobutyric acid. Dopamine and 5-hydroxytryptamine (5-HT), or serotonin, provide modification on these projections. Most of the evidence comes from treatment studies, which are elucidated elsewhere in this chapter. In brief, treatments which are effective in alleviating OCD symptoms also appear to be effective in ameliorating those of OCSDs. However, there have been other studies that demonstrate dopamine and serotonin involvement, and these will be discussed in this section.

Serotonin appears to play a prominent role in the pathophysiology of OCD. Numerous studies involving serotonin reuptake inhibitors have provided the most compelling evidence for serotonin involvement. Serotonin reuptake inhibitors (SRIs) and SSRIs have been shown to be very effective in reducing OCD symptoms. For example, the serotonin metabolite 5-hydroxy-indole acetic acid (5-HIAA) has been found to have higher levels in OCD patients; however, after treatment with clomipramine,

an SRI, the patients showed a reduction of CSF concentration of 5-HIAA, which correlated with an amelioration of their obsessive-compulsive symptoms (Thoren et al., 1980). The results of this study indicate that the antiobsessive effect of clomipramine may be connected to its ability to inhibit serotonin reuptake.

Other studies have examined serotonin receptor function. Zohar, Mueller, Insel, Zohar-Kadouch, and Murphy (1987) studied behavioral and neuroendocrine effects of *meta*-chlorophenylpiperazine (mCPP), a serotonin 5-HT2c and 5-HT1d agonist, in patients with OCD and healthy controls. mCPP was administered to 12 OCD patients along with 20 healthy controls, and the results showed that patients with OCD experienced an exacerbation of obsessive-compulsive symptoms. Another study compared mCPP to fenfluramine by comparing the behavioral and neuroendocrine responses of 20 OCD patients and 10 healthy controls (Hollander et al., 1992). The results showed that OCD patients experienced a worsening of OC symptoms following mCPP administration, but not fenfluramine. These patients also showed a blunted prolactin response, which inversely correlated with the degree of behavioral response.

Administration of sumatriptan, also a 5-HT1d agonist, likewise showed an exacerbation of OC symptoms (Stein et al., 1999). These studies indicate that there is a serotonergic dysfunction in OCD in which the serotonin receptors become hypersensitive. The effectiveness of SSRIs in the treatment of OCD may be due to a downregulation of serotonin receptors. In fact, although the initial response of sumatriptan is a worsening of symptoms, chronic administration of this agonist actually results in improvement in patients who had been previously unresponsive to previous pharmacological treatment (Stern, Zohar, Cohen, & Sasson, 1998).

The involvement of the 5-HT1d receptor is further supported by genetic studies. Mundo et al. (2002) examined whether the G861C polymorphism of the 5-HT1dbeta receptor was implicated in the pathogenesis of OCD. The study genotyped 121 families for the G861C and the T371G polymorphisms of the receptor and found that a significant linkage disequilibrium existed between the G861C polymorphism and the 5-HT1dbeta receptor and OCD. There was preferential transmission of the G allele to affected subjects. The results also showed that patients carrying the G681C allele had higher lifetime Y-BOCS obsession scores. This result did not reach statistical significance, but that could be due to methodology.

Similar studies have also been conducted to elucidate serotonin involvement in OCSDs. While SRIs and SSRIs have been shown to be effective in treating autistic symptoms (Hollander, Phillips, & Yeh, 2003), further studies have suggested that serotonin dysfunction plays a direct role in the development of autism (Chugani, 2002). Serotonin is involved in brain development and takes part in synaptogenesis, and a deficiency in this neurotransmitter may disrupt connections in the sensory cortices, such as a thalamocortical tract, as well as resulting in smaller hippocampal volumes. Chugani et al. (1999) compared the serotonin synthesis capacity of autistic children with that of nonautistic children and found that normal children showed a serotonin synthesis capacity greater than 200% of adults, whereas autistic children showed only a 1.5 times increase from that of adults. These data suggest that during childhood there is a period of high serotonin synthesis capacity, and that a disturbance in this developmental process can lead to autism.

The role of serotonin receptor dysfunction has also been implicated in autism. One study examined the relationship between severity of repetitive behaviors in autism and growth hormone response to sumatriptan in adult patients (Hollander et al.,

2000b). Eleven adults with either autism or Asperger syndrome were given a sumatriptan challenge and a placebo challenge, and blood samples for growth hormone were obtained as a measure of serotonergic function. The results showed that the severity of repetitive behaviors, but not other behavioral dimensions such as communication and social deficits, paralleled the sumatriptan-elicited growth hormone response. These findings suggest that the 5-HT1d receptor may also play a role in mediating a specific component of autism, as well as OCD.

Serotonin involvement has also been implicated in other OCSDs, such as eating disorders. In addition to modifying mood and impulsive control, serotonin also appears to modulate eating behavior. One study examined the role of serotonin in anorexia nervosa and bulimia nervosa by comparing serotonergic activity in eating disorder patients and healthy controls (Ramacciotti, Coli, Paoli, Marazziti, & Dell'Osso, 2003). In this study, platelet [3H] paroxetine binding was investigated in 25 eating disorder patients and 26 normal controls, and the results showed that 5-HT activity was lower in eating disorder patients compared to controls. The results also suggested that 5-HT dysregularity might be a vulnerability trait for an eating disorder as a result of 5-HT2 receptor supersensitivity.

Another study has supported the above hypothesis regarding 5-HT2 receptor supersensitivity and its role in eating disorders (Hu et al., 2003). In this study, it was hypothesized that when an individual diets excessively, 5-HT2c receptors become supersensitive, and that this sensitivity can vary depending on certain alleles. Specifically, the Ser23cys polymorphism can make supersensitivity more likely, causing individuals with that allele to have a predisposition for developing anorexia nervosa. The study examined 118 patients with anorexia nervosa and 244 controls for the presence of the ser23 allele, and found that there was a significant increase in frequency of the ser23 allele in the patient population. The results also indicated that there was a significant correlation between the genotype and minimum body mass index, suggesting that the ser23 allele also has an effect on the severity of the disease. These results are consistent with the hypothesis that serotonin is involved in anorexia nervosa, and that moderate dieting can cause 5-HT2c hypersensitivity, which exacerbates the eating disorder.

Dopamine is another neurotransmitter that appears to play a role in the pathogenesis of both OCD and OCSDs, and treatment studies have shown that drugs that block dopamine reuptake are effective in alleviating OC symptoms (see Treatment section). Due to dopamine's role in thalamocortical activation, many researchers have hypothesized that OCD patients suffer from an inappropriate amount of dopamine release, a hypothesis that has been supported by the fact that when dopamine-related drugs are administered, subjects show stereotypic behavior similar to obsessive behaviors in OCD, TS, and Sydenham's chorea patients. In a recent study, Kim et al. (2003) examined possible dysfunction in dopamine transporter density in the basal ganglia in OCD patients using single positron emission tomography. The study compared the dopamine transporter density of 15 OCD subjects with that of 19 normal adults and found that the specific/nonspecific dopamine transporter binding ratio was significantly higher in the OCD group in both the right and left basal ganglia. The results of this study suggests that dopamine dysfunction in the basal ganglia does participate in the pathophysiology of OCD.

The stereotypic and repetitive behaviors resulting from an excess amount of dopamine in OCD patients have led researchers to search for a similar dopamine

imbalance in certain OCSDs. Because TS patients exhibit the same tic abnormalities, one study hypothesized that dopamine also plays a role in this disorder and that abnormalities in post- and presynaptic function, such as supersensitive dopamine receptors, dopamine hyperinnervation, or abnormal presynaptic terminal function, gave rise to the verbal and motor tics characteristic of TS (Singer et al., 2002). In this study, seven adults with TS and five healthy controls received PET scans with [11C]raclopride (yielding highly specific intrasynaptic DA activity) after an intravenous injection of saline and again after an intravenous injection of amphetamine, a drug which enhances dopamine release and blocks reuptake. The results showed that the relative dopamine release in TS patients was significantly higher from that of healthy subjects.

Treatments

Since serotonin dysregulation has been implicated as one pathway in the pathophysiology of OC spectrum disorders, it follows that serotonin reuptake inhibitors (SRIs) have been utilized in the treatment of these disorders. In fact, OCD and OCSDs show a preferential response to treatment with SRIs and behavior therapy (Flament et al., 1985; Goodman et al., 1990; Hollander et al., 1999). Several important treatment studies of OCD have shown norepinephrine reuptake inhibitors are ineffective (Goodman et al., 1990; Hollander et al., 1999; Thoren et al., 1980). Treatment studies for OC spectrum disorders have been mostly open clinical trials. However, as described below, more recently placebo-controlled studies are being conducted and demonstrate significant therapeutic effect with SRIs.

Neurological disorders such as autism appear to be responsive to SRIs. Serotonin reuptake inhibitors have been shown to reduce the impulsive and aggressive behavior as well as compulsive and repetitive behavior in autistic individuals. A placebo-controlled double-blind study with fluvoxamine in adults by McDougle et al. (1996) resulted in decreased repetitive thoughts and behavior, decreased aggression, and improved social relatedness and language. Trials involving various SRIs shown that these medications are beneficial in the treatment of children with autistic spectrum disorders (Hollander et al., 2003). Fluoxetine appears to be a promising treatment in several domains, including repetitive/compulsive behaviors, social behavior, and global autism severity. The serotonin/norepinephrine reuptake inhibitor venlafaxine was also shown in a small open trial to improve repetitive behaviors in children, adolescents, and young adults with autistic spectrum disorders (Hollander, Kaplan, Cartwright, & Reichman, 2000).

Since SRIs appear to have both anti-compulsive and anti-impulsive effects, it follows that they would be used in the treatment of impulse control OCSDs such as pathological gambling and compulsive shopping (Sood, Pallanti, & Hollander, 2003). Though there are few studies investigating the efficacy of SRIs in the treatment of pathological gambling, those that have been conducted suggest that SRIs are effective for this disorder. Pilot open label studies suggest that citalopram improves symptoms associated with pathological gambling including urge to gamble, preoccupation with gambling, number of days spent gambling, and amount of money lost during gambling episodes (Zimmerman, Breen, & Posternak, 2002). Double-blind, placebo-controlled studies have found that fluvoxamine (Hollander et al., 2000a) and paroxetine (Kim, Grant, Adson, Shin, & Zaninelli, 2002) are also effective in treating

pathological gambling. However, SRIs may not be the optimal treatment of pathological gambling if there is comorbid ADHD or affective disorders.

Open studies have reported a significant clinical response with SRIs in compulsive shopping. One study indicated that open-label treatment with fluvoxamine resulted in a reduction of preoccupation with shopping, time spent shopping, and money spent (Black, Monahan, & Gable, 1997). Double blind, placebo-controlled trials of fluvoxamine in compulsive shopping, however, have failed to find a difference in efficacy between fluvoxamine and placebo (Black, Gabel, Hansen, & Schlosser, 2000; Ninan et al., 2000). Several open label studies found that compulsive shopping symptoms improved as a result of treatment with citalopram (Koran, Bullock, Hartston, Elliott, & D'Andrea, 2002; Koran, Chuong, Bullock, & Smith, 2003). In the Koran et al. (2003) study, 7 weeks of open-label treatment with citalopram was followed by a 9-week double-blind, placebo-controlled discontinuation trial, in which the participants who were randomized to placebo had a higher relapse rate than those who were randomized to citalopram.

In somatoform disorders, preliminary evidence suggests that hypochondriasis may respond to SRIs. Fallon et al. (1993) showed in a 12-week open-label study with fluoxetine that hypochondriacal patients were "much improved" after treatment with medication. Ten out of 14 patients experienced a decrease in measures of phobia and disease conviction. Patients with BDD also appear to respond well to SRIs. Open label studies indicate that open-label fluvoxamine (Hollander et al., 1994; Phillips, McElroy, Dwight, Eisen, & Rasmussen, 2001) and citalopram (Phillips & Najjar, 2003) are effective in treating BDD. A double-blind crossover study indicated that BDD patients responded better to treatment with clomipramine, an SRI, than to treatment with desipramine, a norepinephrine reuptake inhibitor (Hollander et al., 1999). The first published placebo-controlled medication trial in BDD indicated that fluoxetine was more significantly more effective than placebo (Phillips, Albertini, & Rasmussen, 2002).

Eating disorders also may be responsive to treatment with SRIs. The data regarding efficacy of SRIs in the treatment of anorexia nervosa is mixed. Some open label studies (eg, Strober, Pataki, Freeman, & DeAntonio, 1999) have suggested that SRIs have no beneficial effect on patients with anorexia nervosa, while others have demonstrated that SRIs reduce depressive symptoms, which results in weight gain (Gwirtsman, Guze, Yager, & Gainsley, 1990). It has been suggested that SRIs are ineffective in treating malnourished underweight anorexic patients (Ferguson, La Via, Crossan, & Kaye, 1999). Several controlled trials of SRIs, in which anorexic patients treated with SRIs were compared to a control group that did not receive medication, indicate that although weight gain was similar in the SRI and control groups, patients who received SRIs experienced greater improvement of depression, obsessive-compulsive symptoms, and impulsiveness (Fassino et al., 2002; Santonastaso, Friederici, & Favaro, 2001). In a recent double-blind, placebo-controlled trial of fluoxetine in anorexia nervosa, Kaye et al. (2001) found that fluoxetine was effective in increasing weight and reducing relapse of core eating disorder symptoms, obsessive thoughts, and depressed mood.

Among patients with binge eating disorder, several randomized, double-blind, placebo-controlled trials have indicated that citalopram (McElroy et al., 2003), fluoxetine (Arnold et al., 2002), fluvoxamine (Hudson et al., 1998), and sertraline (McElroy et al., 2000) are associated with significant reductions in binge frequency and weight as compared to placebo.

Although serotonin reuptake inhibitors have been most extensively studied in OCD and OCSDs, studies involving atypical antipsychotics that block postsynaptic dopamine and serotonin receptors have shown efficacy as an augmentation strategy added to SRIs in OCD or as monotherapy or augmentation in OCSDs. McDougle, Epperson, Pelton, Wasylink, and Price (2000) showed that risperidone augmentation can prove beneficial to OCD patients who had previously been refractory to SRI treatment alone. In this double-blind, placebo-controlled study, 70 patients were treated with an SRI for 12 weeks. Thirty-six patients were refractory to the SRI and from these patients, 18 were placed on risperidone for 6 weeks and 18 were placed on placebo. The results showed that risperidone augmentation was superior to placebo in reducing OCD symptoms.

Treatment with atypicals has also been shown to be effective in treating various OCSDs. As with OCD, risperidone augmentation of SRIs may be beneficial in the treatment of patients with SRI-refractory trichotillomania and TS (Stein, Bouwer, Hawkridge, & Emsley, 1997), especially in the reduction of tic symptoms. Epperson, Fasula, Wasylink, Price, and McDougle (1999) reported three cases in which patients with SRI-refractory trichotillomania were given a systematic addition of risperidone. All three patients showed a significant decrease in hair-pulling, suggesting that risperidone is an effective treatment for the compulsive behaviors or tics associated with trichotillomania.

Tourette's syndrome also appears to respond well to risperidone. In double-blind, placebo-controlled trial, 34 patients were given risperidone for 8 weeks, and the results showed that risperidone appears to be safe and effective in short-term treatment of tics (Scahill, Leckman, Schultz, Katsovich, & Peterson, 2003). There was a reduction in both tic symptoms and tic severity.

Atypicals such as risperidone also appear to be beneficial for autistic patients with severe behavioral problems (McCracken et al., 2002). In a multisite, randomized, double-blind trial of risperidone, 101 children between the ages of 5 and 17 were placed on risperidone or placebo for 8 weeks. The results showed the risperidone was an effective and well-tolerated treatment for autistic disorder with severe tantrums, aggression, or self-injurious behavior. Two-thirds of the children with positive responses at 8 weeks continued to have benefits after 6 months.

IMPLICATIONS AND LIMITATIONS

Similarities between the aforementioned disorders and OCD in comorbidity, age of onset, clinical course, family history, neurocircuitry, neurotransmitter function, and treatment response provide suggestive evidence for the existence of an OC spectrum. The evidence for the existence of such a spectrum will contribute to future discussions over whether or not to remove OCD from the anxiety disorders category and create a new category for obsessive-compulsive related or spectrum disorders.

However compelling the evidence, it is clear that more work must be done to further our understanding of these disorders and the putative relationship between these disorders. Though a number of studies have found that certain OCSDs, including the somatoform disorders, eating disorders, tic disorders, and trichotillomania are highly comorbid with OCD, we are in need of additional field trials and epidemiological studies of patients who suffer from OCSDs. Unfortunately, the national comorbidity studies have not systematically assessed for OCSDs.

Despite similarities in phenomenology and treatment response, the evidence for familial transmission and comorbidity of impulse control disorders such as pathological gambling is somewhat mixed in comparison to that of other disorders that fall in the OC spectrum. We argue that a certain subset of OCD patients, those who have comorbid ADHD, are more likely to exhibit oppositional defiant, conduct disorder and subsequent impulse control disorders. Future epidemiological and family studies should take this comorbidity of ADHD and OCD into account and evaluate the relationship of the impulsive OCSD and OCD with and without comorbid ADHD.

A number of family studies of OCD and OC spectrum disorders have concluded that family members of OCD patients are significantly more likely to display OC spectrum disorders such as eating disorders, tic disorders, somatoform disorders, autism, and trichotillomania. That these disorders have a familial relationship to OCD appears to be established. There is now a need for studies investigating whether there are common genetic contributions to OCD and OC spectrum disorders. Ultimately, to determine a fundamental relationship between OCD and the OCSDs will require demonstration of a shared etiology.

Evidence of similar abnormalities in neurocircuitry and neurotransmitter function between OCD and OCSDs also suggest that these disorders may be closely related in their etiologies. While not identical, comparable responses to drug challenges and the involvement of similar neurotransmitter systems point to an overlap in the underlying causes of these disorders. It can be argued that it is these very differences that lead to the distinct symptoms that characterizes each disorder. However, more research is needed before a complete understanding of the mechanisms involved in the pathogenesis of OCD and OCSDs can be achieved.

In the past, it has been argued that more double-blind, placebo-controlled medication trials for OC spectrum disorders must be carried out. We applaud the recent effort to address this concern and hope that the trend of double-blind, placebo-controlled studies of SRIs and atypical antipsychotics in OCSDs continue.

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Chapter 6

OBSESSIVE-COMPULSIVE DISORDER: ESSENTIAL PHENOMENOLOGY AND OVERLAP WITH OTHER ANXIETY DISORDERS

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Very few of the emotional disorders described in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 1994) are as devastating as obsessive-compulsive disorder (OCD). Individuals suffering from OCD are likely to have difficulty with work or school, falter in maintaining social and emotional relationships, and struggle with daily life events that others take for granted. They are more likely than patients with other anxiety disorders to suffer from severe depression, require inpatient treatment, and undergo psychosurgery (Barlow, 1988). Additionally, the psychopathology in OCD is among the most complex of the emotional disorders. Intense and often senseless and bizarre thoughts that run contrary to the individual's logic, sense of self, and core values come to mind, often at the worst possible times. More overt, yet equally impairing signs of OCD include profound avoidance and performance of senseless and excessive compulsive rituals that can dominate the sufferer's day.

How are the clinician and researcher to understand this enigmatic and often disabling condition? Is OCD a thought disorder? Is it a disturbance of repetitive behavior? Is it a problem with impulse control? Is it one of anxiety? How one answers these questions has major implications for how they conceptualize OCD, how they study the disorder, and how they evaluate and treat affected individuals. In this chapter, we describe a conceptualization of OCD phenomenology that is logically and theoretically consistent, supported by evidence from research on its signs and symptoms, and consistent with the most effective and specific form of treatment for this problem. Phenomenological parallels between OCD and other anxiety disorders are also discussed along with the conceptual significance of specific, effective treatment procedures for OCD. To begin with, we present two cases that illustrate the complexity and heterogeneity of OCD and to which we will refer throughout this chapter.

ANDREW'S CASE

Andrew was a 48-year-old, single, university professor who had suffered with OCD in one form or another since he was a young boy. He had previously been treated with medications and "talk" therapy, yet finding these interventions more or less unhelpful, became resigned to living with his symptoms. Andrew presented to our clinic in response to an advertisement for research participants with OCD. Andrew's primary symptoms involved hoarding his garbage. Everything he discarded—empty boxes, milk cartons, envelopes, even dental floss and used tissue—he carefully placed into a "temporary trash can." When this container was full, every item had to be carefully checked to ensure that nothing important was being permanently thrown away. This meant closely inspecting empty boxes, cans, and envelopes and sorting through papers, dirty tissues, and dirty paper towels. Nothing could leave the house for the weekly garbage collection until it had been thoroughly inspected and deemed safe to discard by Andrew.

Unfortunately, things had come to the point where Andrew, plagued by doubts that perhaps by mistake he had thrown away something important, would get stuck checking the same garbage over and over. Sometimes, he would spend hours at a time sorting through piles. Other piles would be left for "later," but never actually confronted. Thus, his home had become filled with garbage. He described awful odors, rooms that were unsuitable for entry, and even complaints from neighbors. When asked about what important items he might throw away with his garbage by mistake, Andrew could not say for sure. Further, he acknowledged never finding anything important in the trash that he checked. Yet he reported feeling extremely uncomfortable just knowing that he "could" or "might" discard something important by mistake. He described this sense of uncertainty as "excruciating." Although Andrew was able to hold his job at the university, his OCD symptoms increasingly interfered with his social functioning. He had not had guests to his home in years and felt uncomfortable dating or developing close relationships.

MICHELLE'S CASE

Michelle, a 25-year-old married woman with a healthy 19-month-old daughter named Lauren, was referred to our clinic for evaluation. Michelle's symptoms, which began a few days after Lauren's birth, included bizarre, unwanted thoughts and ideas of deliberately injuring her young child. For example, one day while her husband was at work, Michelle had an unwanted impulse to put Lauren in the microwave. Other intrusive thoughts included images of drowning Lauren in the bathtub and of pushing her stroller into traffic. Michelle also had intrusive doubts concerning awful acts that she may have previously committed, such as "what if I molested Lauren without realizing it?" and "what if I fed her poison by mistake?" Michelle reported that these terribly upsetting recurrent thoughts were taking up to 2 h each day despite desperate efforts to suppress them. By the time she presented in our clinic, she even doubted whether she was really the good-hearted, compassionate person she had once considered herself. After looking forward to being a mother for so long, she worried whether she was even fit to raise children. "What would my family think if they found out about these terrible thoughts," she worried. Until her evaluation, Michelle had concealed these "horrifying secretive thoughts" from others. She felt that saying the ideas out loud might make

them come true. Of course, these thoughts had never actually led to any harmful behavior.

However, obsessional thoughts were not Michelle's only problem. She was also spending several hours performing compulsive praying rituals, which included asking God to keep her from acting on her terrible thoughts. If another stray thought interrupted her praying, she had to start from the beginning to assure herself that she had prayed "completely and perfectly." If, during any activity, Michelle had obsessional thoughts about harming her daughter, she felt compelled to repeat the activity until she could get through it without having the obsessional thought. On one occasion, it took her over 2 h (and 15 pieces of stationary) to write a simple thank-you note to a friend. She was also getting "stuck" having to repeat routine actions such as opening or closing doors, standing up out of a chair, or dialing telephone numbers since these actions had to be completed without any bad thoughts. If unwanted thoughts came to mind, Michelle had to "cancel" them out by imagining a red line through the thought. Along with these compulsive rituals, Michelle was avoiding many situations to prevent herself from having stressful thoughts. For example, she had enrolled in a night class at a local community college to avoid having to give Lauren her evening baths. Michelle said that she performed all of these compulsive and avoidant behaviors because she felt it reduced her chances of acting on the horrible thoughts.

CONCEPTUAL APPROACHES TO OCD THROUGH HISTORY

Not surprisingly, varied approaches have been taken through history to understand the complex symptom picture in OCD, beginning with early accounts of demonic possession and treatment by exorcism. The first medical (as opposed to religious) explanations held that obsessions and compulsions developed from "psychic fatigue"—a "diminution" of available mental energy that was thought to prevent individuals from controlling their thoughts or actions (Janet, 1903). Freud and the psychoanalysts viewed obsessions as unconscious impulses, and compulsions as defensive responses to these impulses (Salzman & Thaler, 1981). Although not widely supported by research, these early conceptualizations represent attempts to understand the phenomenology of the problem.

Behavioral psychology continued the focus on phenomenological mechanisms by applying *functional analysis*, which clarified observable (and therefore measurable) antecedents and consequences of obsessions and compulsions (eg, Dollard & Miller, 1950; Mowrer, 1960; Rachman & Hodgson, 1980). Behavioral (learning) models of OCD derived from clinical observation were subsequently tested in the laboratory using elegant experimental approaches (eg, Rachman & Hodgson, 1980). According to learning theory, obsessions are viewed as classically conditioned anxiety responses, and compulsive behavior, as attempts to escape from obsessional anxiety that are negatively reinforced by the reduction in distress that they engender (Dollard & Miller, 1950). Thus, OCD was viewed as a set of self-perpetuating habits of thinking and behaving. There is empirical support for key components of this model (eg, that compulsive behavior is negatively reinforced) and it therefore remains a basis for conceptualizing and treating OCD symptoms to this day (as we will discuss later in this chapter).

Cognitive theorists have delved even further into the phenomenological mechanisms of OCD by identifying and studying maladaptive thinking styles considered to underlie obsessional fears and urges to perform compulsive behavior (Obsessive-Compulsive Cognitions Working Group, 1997; Salkovskis, 1985, 1989, 1999). Synthesizing cognitive and behavioral research, investigators from this perspective have proposed and critically examined an idiopathic (as opposed to nomothetic) approach to understanding OCD symptoms that begins with the fact that such symptoms occur on a continuum from "normal" obsessions and rituals to unremitting clinical symptoms (eg, Rachman & de Silva, 1978). Cognitive theory contends that mechanisms underlying the development and persistence of clinically significant OCD symptoms are highly individualized and involve, to some extent, one's early experiences (eg, Salkovskis, Shafran, Rachman, & Freeston, 1999). For example, a child whose fleeting thoughts about the death of his dog are coincidentally followed by the dog's actual death might (erroneously) conclude that his thoughts had something to do with the event. Experiences such as this could lead to the belief that negative thoughts must be controlled or avoided since they might increase the probability of disastrous consequences.

Unfortunately, the theory-driven and empirically based cognitive and behavioral approach to OCD phenomenology is at variance with the present enthusiasm over largely atheoretical diagnostic criteria such as those presented in the *DSM*, which emphasize disorders that are comprised of lists of signs and symptoms. Because of the shift toward a medical model of psychiatric disorders, a conceptual or theoretical understanding of psychological mechanisms is replaced by a more superficial "checklist" approach that simply collects behaviors according to *form* or topography, as opposed to *function*. Such an approach, inaugurated with the *DSM-III* operational criteria, made good sense given that the original aim was to improve upon the diagnostic unreliability of *DSM-II*. However, improvement in diagnostic reliability has come with unfortunate side effects. In particular, attempts to define OCD solely on the basis of overt signs and symptoms have made it easy to neglect the more complex *functional* relationships between obsessional and compulsive phenomena. We argue here that this has fostered a superficial and incomplete understanding of OCD as merely a collection of grossly abnormal, repetitive, and bizarre thoughts and behaviors. Moreover, it has blurred the distinction between symptoms of OCD and those of various other disorders that also include excessive or repetitive thinking or behavior as diagnostic criteria.

OCD ACCORDING TO THE DSM-IV

In the DSM-IV (American Psychiatric Association, 1994), OCD is categorized as an anxiety disorder and defined by the presence of *obsessions* or *compulsions* that produce significant distress and cause noticeable interference with various aspects of functioning. *Obsessions* are defined as intrusive thoughts, ideas, images, impulses, or doubts that the person experiences in some way as senseless and that evoke affective distress. *Compulsions* are defined as repetitive behavioral (eg, checking, washing) or mental rituals (eg, praying) in response to obsessions. As with obsessions, compulsive behavior is senseless or excessive.

LIMITATIONS OF THE DIAGNOSTIC CRITERIA

Research on the phenomenology of OCD suggests three limitations of the current diagnostic criteria that may lead to confusion over the disorder's cardinal features. These include (*a*) the implied assumption that the presence of obsessions *or* compulsions is necessary and sufficient for a diagnosis, (*b*) the emphasis on *form* as opposed to *functional* characteristics of symptoms, and (*c*) the failure to recognize certain covert, yet phenomenologically significant, signs and symptoms. As we describe below, the meaningful patterns of phenomenology that are key to understanding the essential features of OCD are lost on the DSM's symptom checklist approach. Instead, this approach fosters a concern for less consequential signs and symptoms, such as the repetition of thoughts and behavior; and this has resulted in confusion between the symptoms of OCD and those of other disorders that also happen to involve repetitive or stereotyped thoughts and behavior.

Obsessions or Compulsions

The DSM-IV, in stating that either obsessions *or* compulsions are necessary and sufficient for a diagnosis of OCD, leaves the impression that these symptoms are independent phenomena and that OCD should be diagnosed if the clinician can identify any striking, excessive, bizarre, stereotyped, repetitive, or impairing thoughts or behaviors. This approach might also lead to drawing parallels between OCD symptoms and symptoms of other disorders on the basis of these superficial characteristics. Excellent examples are *compulsive* gambling, *compulsive* stealing, *compulsive* use of pornography, and *obsessive* jealousy, which clinicians frequently subsume under the OCD umbrella. In addition to parallels based on repetition, OCD has also been likened with psychosis on the basis of the bizarreness of its symptoms. For example, the physician who referred Andrew to our clinic had been treating him with an antipsychotic medication for his "psychotic-like garbage hoarding ritual." In her case notes conceptualizing Andrew's problem, there were references likening this behavior to "animal models in which some species of birds save their waste matter."

However, there are considerable differences between Andrew's compulsive behavior and the hoarding observed in birds. Whereas birds save their waste (and other materials) as a matter of adaptive instinct (it helps in building protective nests), Andrew's behavior occurs in the context of thoughts (fears) of mistakenly discarding important items and it is often resisted and recognized as senseless. This is consistent with Rachman's (2002) assertion that OCD patients with checking rituals engage in checking to reduce obsessional distress evoked by situations in which they fear being responsible for negative outcomes. The parallel with psychotic behavior is also inaccurate: people with psychosis do not typically have obsessional fears; nor do they recognize the senselessness or excessiveness of their thinking and behavior. Instead, they engage in strange behavior because they have lost touch with reality and fall victim to delusional thinking.

This superficial conceptual approach to OCD as a collection of bizarre or repetitive symptoms is also at odds with research findings on the phenomenology of OCD. For example, Foa and Kozak (1995) found that 96% of 411 OCD patients reported

both obsessions *and* compulsions on the Yale-Brown Obsessive-Compulsive symptom checklist, whereas only 2.1% reported predominantly obsessions and only 1.7% reported predominantly compulsions. Moreover, a growing literature has identified dimensions and "subtypes" of OCD in which obsessions and compulsions load together on the same symptom-based factors and clusters (eg, Leckman et al., 1997) as well as on measures of symptom severity (Amir, Foa, & Coles, 1997). Thus, as much as the distinction between obsessions and compulsions is intuitively appealing and endorsed by the DSM, research suggests that OCD phenomenology does not necessarily distill neatly into these two categories.

Form Versus Function

The DSM's emphasis on the *form*, as opposed to the underlying *functional properties*, of obsessions and compulsions is a related limitation of the definition. A series of simple yet elegant laboratory studies has closely examined the phenomenology of compulsive behavior in OCD. The research paradigm included exposing patients to stimuli that provoked urges to engage in compulsive behavior and recording subjective levels of anxiety and urges to ritualize before and after exposure, and after performing the compulsive ritual. The findings of these studies can be summarized as follows. For patients with washing rituals evoked by fears of germs, exposure to contaminants led to an increase in subjective anxiety and urges to ritualize, while completion of a washing ritual rapidly reduced the distress and urges. A more gradual spontaneous reduction in both anxiety and compulsive urges was observed when the performance of rituals was delayed for 30 min (Hodgson & Rachman, 1972). Similar results were obtained in two studies of patients with checking rituals evoked by exposure to potentially harmful stimuli such as knives (Roper & Rachman, 1976; Roper, Rachman, & Hodgson, 1973) and in a study of patients with mental compulsions evoked by intrusive unacceptable thoughts (de Silva, Menzies, & Shafran, 2003).

These findings show that in OCD, compulsive behavior is performed in response to specific cues such as particular personally relevant situations or thoughts that evoke anxiety concerning feared outcomes. Moreover, anxiety and compulsive urges quickly subside after completion of the ritual. The evocation of compulsive urges by obsessional fear, the immediate reduction in anxiety following compulsive behavior, and the eventual reduction of anxiety and compulsive urges (even if the ritual is not performed) are the hallmarks of OCD. Thus, whereas the most readily *observable* signs of OCD may be repetitive, "bizarre," or stereotyped obsessions and compulsions, the most *consequential* aspect of OCD is the functional relationship *between* obsessions—which evoke distress—and efforts to reduce this distress (ie, compulsions). Understanding this relationship (and the rich patterns of phenomenology present in OCD in general) requires a rigorous assessment of thoughts and behaviors (ie, functional analysis) that is beyond the cursory symptom checklist approach advocated in DSM-IV.

Functional assessment also leads to the realization of substantial differences between OCD and other disorders sometimes considered to be related to OCD; trichotillomania being an excellent example (Stein, Simeon, Cohen, & Hollander, 1995). As the work of Rachman and his colleagues suggest, and as is illustrated by the cases of Andrew and Michelle above, patients with OCD experience thoughts that (at least early on in the disorder) are experienced as unacceptable and distressing. They then

attempt to control or neutralize these thoughts with some other thought or action (eg, doubts about discarding important items lead to excessive saving and checking; repugnant thoughts about harm lead to avoidance, praying, and repeating). Thus, compulsive behavior in OCD leads to a sense of escape or relief from obsessional anxiety. Yet this relationship between obsessional thinking and repetitive behavior does not exist in trichotillomania. In fact, evidence suggests that patients with trichotillomania pull their hair repeatedly in response to feelings of general tension, depression, boredom, frustration, indecision, or fatigue—but not obsessional fear as in OCD (Christenson, Ristvedt, & Mackenzie, 1993; Stanley & Mouton, 1996). Studies also demonstrate that hair pulling in trichotillomania leads to pleasurable feelings, a phenomenon not observed with rituals in OCD (Stanley, Swann, Bowers, & Davis, 1992). Thus, the phenomenology (ie, function) of the repetitive thought and behavior in OCD and trichotillomania is unmistakably distinct.

Tourette's syndrome (TS), which involves repetitive motor or vocal tics is also sometimes conceptualized as related to OCD, at least partially on the basis of the presence of repetitive, stereotyped behavior in both conditions (Hollander & Wong, 1995, 2000). Nevertheless, research has pointed out important functional differences between tics in TS and compulsive rituals in OCD, clearly differentiating these two phenomena. For example, Miguel, Coffey, Baer, Savage, Rauch, and Jenike (1995) found that whereas compulsions were preceded by obsessional fears, tics were preceded by sensory phenomena such as inner tension or urges to release energy that were not present in OCD patients. They also found that whereas OCD patients reported cognitive phenomena and autonomic arousal, TS patients did not endorse such symptoms. Finally, OCD patients reported mounting autonomic anxiety symptoms when they were prevented from performing compulsive rituals, yet TS patients did not report such symptoms. Thus, whereas compulsive rituals are deliberate and serve as an escape from obsessional distress, tics are spontaneous (sudden), and performed to reduce sensory discomfort or tension—not to neutralize obsessional fear or anxiety (Miguel et al., 1995; Shapiro & Shapiro, 1992). As is the case with trichotillomania, a relationship between TS and OCD has been assumed to exist at least initially on the basis of superficial description of the behavior as "repetitive" or "compulsive," (Hollander & Wong, 1995, 2000) as opposed to a closer investigation of the function of the behavior (ie, its antecedents and consequences).

Beyond Overt Compulsions

In focusing on the repetitive and stereotyped *form* of compulsive behavior, at the expense of its *functional* properties, the DSM definition of compulsions overlooks additional tactics that patients use in response to their obsessional fears. Along with stereotyped behavioral rituals such as washing and checking, most patients engage in inconspicuous strategies such as avoidance, concealment, neutralizing (eg, mentally "crossing out" unwanted thoughts), and thought suppression (eg, Ladouceur et al., 2000a). As Michelle's case illustrates, these covert strategies are performed with the intent of reducing distress associated with (*a*) unwanted obsessional thoughts and/or (*b*) the probability of feared outcomes. Thus, they are equivalent to overt compulsive acts such as washing, checking, and repeating actions in that their function is identical. The presence of covert neutralizing strategies that serve the same purpose as compulsive rituals, further suggests that the phenomenological importance of compulsive rituals

in OCD lies not in their bizarreness, repetition, stereotypy, or even their visibility; but instead in the purpose or *function* of the behavior or mental act. Overt compulsions are simply one type of strategy used by patients to reduce obsessional distress.

OCD PHENOMENOLOGY: BEYOND DIAGNOSTIC CRITERIA

As is illustrated by the cases of Andrew and Michelle, OCD is characterized by an internally consistent pattern of thinking and behaving, which is unique and can be readily identified with proper assessment. First, individuals experience involuntary thoughts and doubts (obsessions) that they find unacceptable and anxiety evoking (Rachman & de Silva, 1978). To deal with the obsessional distress, patients resort to purposeful behavioral or mental activity (Roper et al., 1973). Obsessive-compulsive disorder is therefore best conceptualized as a problem in which an individual comes to fear, and magnify the significance of, intrusive thoughts, images, urges, and doubts, leading to obsessional preoccupation. Below, we describe phenomenological research supporting this conceptual approach to OCD.

Obsessions

Evidence from research on obsessional phenomena supports a dimensional, as opposed to a categorical, approach to understanding these symptoms. Most convincingly, results from numerous investigations converge to indicate that 80 to 90% of the population at large experience senseless intrusive thoughts, the content of which is indistinguishable from that reported by individuals with OCD (eg, Abramowitz, Schwartz, & Moore, 2003; Rachman & de Silva, 1978; Salkovskis & Harrison, 1984). However, whereas most people easily brush such thoughts aside or consider them senseless, those with OCD (mis)appraise them as highly significant, unacceptable, and dangerous, leading to obsessional anxiety (eg, Salkovskis, 1985, 1989, 1999).

A specific example of how people with OCD ascribe excessive significance to otherwise meaningless intrusive thoughts is by equating thoughts with actions (ie, *thought–action fusion*). Several studies have found that compared to healthy controls and patients with other anxiety disorders, those with OCD believe that thoughts about negative events (*a*) are equivalent to the corresponding behavior and therefore morally reprehensible; and (*b*) make such events more likely (eg, Abramowitz, Whiteside, Lynam, & Kalsy, 2003). Holding such beliefs about thoughts leads to the perception that certain thoughts are harmful and must be kept in check, or that a feared outcome must be prevented. This is clear in the clinical examples provided above. Andrew experienced his absurd doubts about discarding important belongings as meaningful and necessitating reassurance. Michelle perceived unwelcome violent thoughts toward her inculpable infant as morally reprehensible and likely to lead to violent acts.

Compulsive Rituals

It is well known that one response used by individuals with OCD to deal with obsessional distress is compulsive ritualizing. As discussed above, research has

established that rituals (eg, checking, repeating, washing) result in an immediate (albeit short-term) reduction in anxiety and a decrease in urges to ritualize further (eg, Hodgson & Rachman, 1972). More recently, the significance of mental compulsive rituals has also been realized. Foa and Kozak (1995), for example, found that ~80% of individuals with OCD engage in mental compulsive rituals such as praying and mentally reviewing. de Silva et al. (2003) found that mental compulsions are also performed deliberately with the purpose of reducing obsessional anxiety and are therefore functionally equivalent to behavioral compulsions such as washing and checking.

Although they are intended to reduce discomfort immediately, a significant consequence of both behavioral and mental rituals is that in the long-term, they produce a self-perpetuating cycle of obsessional anxiety and ritualizing. Recognition of this process is essential in understanding why compulsive rituals in OCD are repetitious. One factor that contributes to this self-perpetuating mechanism is the way in which people with OCD interpret the outcome of their rituals as somehow preventing negative consequences. For example, as long as Michelle prays according to special ritualistic rules, she will never find out that she is unlikely to act on her upsetting aggressive thoughts even if she does not perform this ritual. Thus, her compulsive behavior serves to maintain (*a*) her unfounded fear that thoughts will cause her to act violently and (*b*) her belief that she had better ritualize in order not to commit such acts. Similarly, Andrew believed the reason he did not routinely discard important items was that he checked his garbage carefully. As long as he continues to carefully check, he is robbed of any opportunity to obtain evidence to disconfirm this belief.

Another reason that compulsive rituals become self-perpetuating is that they are ineffective at providing a sense of complete assurance that feared catastrophes will not occur. Individuals with OCD, and especially those with checking rituals, display an intolerance for uncertainty (Tolin, Abramowitz, Brigidi, & Foa, 2003) and they perform rituals as a means of gaining reassurance of safety. Because the actual occurrence of many obsessional fears (eg, whether germs are present, whether one is going to hell) can never really be verified with absolute certainty, no amount of ritualizing will bring about sufficient reassurance. Further, in the case of checking rituals, research suggests that these compulsions lead to a decline in memory confidence (van den Hout & Kindt, 2003; Tolin, Abramowitz, Brigidi, Amir, Street, and Foa, 2001), thus evoking further urges to check.

Neutralization

Unfortunately, the DSM's acknowledgement of patients' responses to obsessional distress emphasizes repetitive compulsions. This is likely the result of DSM's focus on the *form* or *phenotype*, as opposed to the *function*, of these signs and symptoms. However, researchers have identified a variety of "neutralizing strategies" present in OCD that do not necessarily meet DSM criteria for compulsions, but which do possess identical functional properties (Rachman & Shafran, 1998). That is, although some of these strategies would not be identified as stereotyped or ritualistic (eg, "canceling" an unacceptable thought with a more acceptable one), they are intended to neutralize obsessional thinking, reduce the perceived probability of feared consequences, and reduce anxiety (eg, van den Hout, van Pol, & Peters, 2001). Therefore, neutralization

strategies are the phenomenological equivalent of compulsive rituals. Neutralizing strategies include reassurance seeking, overanalyzing and rational self-talk (ie, to convince oneself of the unimportance of the thought), mentally replacing a "bad" thought with a different "good" thought, performing a brief mental or behavioral act, intentional distraction, and attempts to suppress or control unwanted thoughts (Ladouceur et al., 2000a). The choice of strategy used in a given situation may be influenced by the intensity of the obsessional thought, the context in which it occurs, how the thought is appraised, and how well particular strategies have "worked" in the past (Freeston & Ladouceur, 1997; Ladouceur et al., 2000a).

A series of experiments on the phenomenology of neutralizing strategies has also revealed that, like overt compulsions, these maneuvers are self-perpetuating. Salkovskis, Westbrook, Davis, Jeavons, and Gledhill (1997) found that trying to neutralize upsetting intrusive thoughts led to increased discomfort associated with the thought and urges to continue to neutralize. Numerous studies point to the paradoxical effects of attempted thought suppression, whereby trying to suppress unwanted thoughts actually results in increased thought frequency (for a review see Abramowitz, Tolin, & Street, 2001). Our research group has also found a link between OCD symptom severity and certain ways in which people respond to and attempt to control intrusive unwanted thoughts, namely by punishing themselves and worrying about the thought (Abramowitz, Whiteside, et al., 2003).

Summary: What Is OCD?

Investigations of the functional properties of OCD symptoms therefore suggest that OCD can be conceptualized as a problem in which an individual excessively fears their own (normally occurring) intrusive negative thoughts and doubts, leading to preoccupation with such thoughts. To deal with obsessional fear, individuals habitually deploy responses such as rituals, neutralizing, avoidance, and thought suppression, which have the short-term effect of providing escape from distress, but which paradoxically maintain the obsessional fear and increase urges to ritualize and neutralize in the long run. Such strategies also increase the perceived salience of intrusive thoughts. Perhaps the fact that many neutralizing strategies are inconspicuous (as compared to overt compulsive behavior) contributes to the fact that they are a less well-recognized phenomenon in OCD. However, to the patient, these behaviors all serve the same purpose and have the same effects. Evidence that compulsive rituals represent just one of many strategies used by patients to escape from obsessional distress suggests that the phenomenologically important aspect of compulsive behavior in OCD is not its bizarreness, stereotypy, or repetition, but instead its *function*.

Is OCD an Anxiety Disorder?

One aim of diagnostic classification is to organize clinical problems into groups sharing common characteristics. The classification of OCD has been a matter of some debate, with some authors asserting that it is incorrectly classified among the anxiety disorders (eg, Enright, 1996). The basis for this assertion is often that compared to other anxiety disorders, OCD more often begins during childhood (Karno, Golding, Sorenson, & Burnam, 1988), is associated with greater functional impairment, and has higher rates of psychiatric comorbidity (especially with depression; Steketee,

Grayson, & Foa, 1987). Other reasons OCD is sometimes seen as separate from other anxiety disorders include the presence of strange, intrusive, and repetitious obsessions and compulsions; and the occasional presence of poor insight into the senselessness of these symptoms. In fact, on the basis of these characteristics, some have proposed that OCD is more similar to schizophrenia than to other anxiety disorders (eg, Enright, 1996). Below, we assert that OCD *is* correctly conceptualized as a primary anxiety disorder on the basis of shared phenomenological mechanisms and similar responses to specific treatment procedures that target this mechanism.

Phenomenological Similarities

Despite the topographical differences between OCD and other anxiety disorders mentioned above, there are clear similarities in the underlying phenomenological mechanisms that characterize these disorders; and these similarities suggest that OCD is appropriately categorized as an anxiety disorder. As is shown in Table 6.1, OCD, specific and social phobia, panic, posttraumatic stress disorder (PTSD), and generalized anxiety disorder all involve fear that occurs in the context of disorder-specific situations or stimuli. Research also demonstrates that across disorders, this fear is maintained by distorted perceptions regarding the dangerousness of such situations, sensations, or mental events (also shown in Table 6.1). Interestingly, careful clinical assessment reveals that poor insight about the irrationality of fear is not specific to

TABLE 6.1. Feared stimuli, dysfunctional cognitions, and behavioral processes underlying the maintenance of different anxiety disorders

Diagnosis	Fear evoking stimuli	Underlying beliefs	Safety behaviors
OCD	Intrusive thoughts	Thoughts are equivalent to actions, inflated responsibility for preventing harm (eg, Obsessive-Compulsive Cognitions Working Group, 1997)	Checking, hoarding, washing, ordering/arranging, covert neutralizing
Specific phobia	Snakes, heights, injections	Overestimation of the likelihood or severity of danger from the feared stimulus (Beck & Emery, 1985)	Drinking alcohol before flying, distraction during injections
Social phobia	Social situations	Other people are highly judgmental, negative evaluation is intolerable (Clark & Wells, 1995)	Speaking softly, using alcohol
Panic disorder and agoraphobia	Arousal-related body sensations	Heart palpitations signify a heart attack, dizziness leads to fainting (Clark, 1986)	Sitting down, going to emergency room, drinking water, calling a safe person
PTSD	Memories of traumatic events	Nowhere is safe, I could have prevented the trauma (Foa & Rothbaum, 1999)	Distraction, relying on others for safety, carrying a flashlight
Generalized anxiety disorder	Images of low probability catastrophes	Intolerance of uncertainty, the world is a dangerous place (Wells, 2000)	Calling loved ones to verify safety, asking for reassurance

OCD: individuals with other anxiety disorders also hold their illogical beliefs quite firmly and sometimes do not recognize their irrationality. Excellent examples of "poor insight" in other anxiety disorders include the panic patient who visits the emergency room daily, believing he is having a heart attack; the social phobic who remains housebound with the belief that others will make fun of him; and the person with a fear of flying who is convinced that her next flight will end in a crash. Why poor insight has been included in the DSM as a diagnostic specifier for OCD but not for other anxiety disorders is not clear.

There is also phenomenological similarity in how patients with different anxiety disorders respond when confronted with feared stimuli, or with the prospect of exposure to such situations. In a broad sense, when danger is perceived, taking action to reduce or escape the potential threat is normal and adaptive (ie, the "fight or flight" instinct). Indeed, we observe this kind of avoidance and "safety-seeking" behavior across the anxiety disorders (eg, Clark, 1999). However, the behaviors of clinical patients are (by definition) in response to *irrational* fears of unlikely or un-costly negative consequences. Examples include resting to prevent heart attacks during panic episodes, the agoraphobic's use of a "safety person" to avoid losing control, and overrehearsal of a speech by someone with social anxiety who fears that any mistake would result in public humiliation. Analogously, in OCD, we might observe the use of a paper towel to open bathroom doors because of a fear of "urine germs," repeated checking of the roadside motivated by a doubt of unknowingly hitting a pedestrian with one's car, or repetitive washing because of the fear of illness. Although these responses appear topographically diverse, each is phenomenologically linked to an overestimation of threat that is characteristic of each disorder. Moreover, at least to the patient, the safety response functions to reduce the perceived probability of feared consequences. The far right column in Table 6.1 displays examples of safety behaviors observed in the various anxiety disorders. As we have discussed, compulsive rituals in OCD, although *superficially* unique from other forms of safety behaviors, are functionally equivalent to safety seeking as observed in other anxiety disorders (eg, Salkovskis, 1999).

If OCD possesses psychological mechanisms similar to those of the other anxiety disorders, what accounts for the "bizarre" thoughts, functional impairment, and relationship with depression that are most apparent in OCD? With respect to bizarreness, several studies have found that the content of clinical obsessions is indistinguishable from that of normally occurring intrusive thoughts that are reported by the majority of the population at large (Rachman & de Silva, 1978; Salkovskis, & Harrison, 1984). "Normal" intrusive cognitions identified by nonclinical participants included thoughts about violent or "unnatural" sexual behavior, impulses to abuse, attack, or say rude things to loved ones, and thoughts of contamination (Rachman & de Silva, 1978). Thus, the assertion that individuals with OCD have a particular problem in which their brain generates strange and bizarre thoughts (as in schizophrenia) is unsupported.

Turning to the high comorbidity with depression and associated functional impairment, most likely these phenomena are a by-product of the intense fear of, and inability to control, involuntary yet unacceptable intrusive thoughts. Tolin, Abramowitz, Hamlin, Foa, and Synodi (2002) found that among individuals with OCD, the failure to suppress target thoughts was associated with negative self-related beliefs characteristic of depression and hopelessness (eg, "I am mentally weak," "I am sick"). Thus, it is not surprising that Demal, Lenz, Mayrhofer, and Zapotoczky (1993) reported

finding that depressive symptoms typically begin *following* OCD onset (as opposed to vice versa), and that depressive symptoms are related to the severity of obsessions but not compulsions (Ricciardi & McNally, 1995).

A Common Response to a Specific Treatment

Cognitive-behavior therapy is a set of procedures used to modify patients' pathological beliefs/assumptions and behaviors that maintain their emotional disorders. A strength of this approach is that the active ingredients in treatment are highly specific and derived from empirically supported models of psychopathology. As Table 6.1 suggests, for anxiety disorder patients, this means that treatment must target the disorder-specific erroneously high estimates of risk and the safety behaviors that function to maintain the faulty estimates. In particular, research suggests that this is best accomplished using therapeutic exposure to feared stimuli (sometimes aided by logical discussions regarding dysfunctional cognitions) and elimination of safety behaviors (Clark, 1999; Salkovskis, 1999). We argue that the similarity of theoretically derived and specific effective treatment techniques/programs for OCD and the various other anxiety disorders provides evidence that OCD is closely related to these other anxiety disorders.

In recent decades, the prognosis for people with OCD has improved dramatically and this is due in large part to the development and dissemination of effective cognitive-behavioral treatment programs (eg, Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000; Steketee, 1993). Obsessive-compulsive symptoms respond preferentially to specific therapeutic procedures that are derived from cognitive and behavioral models of the disorder as described above. In particular, what is required in treatment is (*a*) systematic repeated/prolonged exposure to cues that produce obsessional anxiety and (*b*) restraint from performing compulsive or neutralizing behaviors (response prevention) to reduce the anxiety (Abramowitz, 1997). While the precise mechanism of change with exposure and response prevention (ERP) is debated (eg, Foa & Kozak, 1986), it is well established that exposure weakens pathological overestimates of the likelihood or severity of danger associated with obsessional thoughts, and response prevention weakens urges to perform compulsive behavior (Foa, Steketee, & Milby, 1980; McLean et al., 2001). Correctly implemented, these procedures help patients learn that their obsessional fears are unfounded and that they need not neutralize or perform rituals to prevent obsessionally feared disastrous consequences.

Cognitive-behavioral treatment programs for other anxiety disorders contain highly similar procedures. Phobic patients are instructed to systematically confront their feared stimuli (eg, needles, social situations) while giving up safety behaviors (eg, avoidance, distraction, alcohol use; Turk, Heimberg, & Hope, 2001). Patients with panic disorder and agoraphobia are helped to confront feared internal states that cue panic attacks (eg, dizziness, racing heart) via interoceptive exposure, and feared situations (eg, shopping malls) via in vivo exposure, while simultaneously eliminating safety behaviors such as using benzodiazepine medication or resting to reduce a rapid heart rate (Clark, Salkovskis, Hackmann, Middleton, Anastasiades, & Gelder, 1994). Individuals with PTSD are helped to confront unwanted intrusive memories of their traumatic experiences using imaginal exposure (reliving) while ceasing avoidance of situational reminders of the trauma (eg, Foa & Rothbaum, 1999). Recently, Ladouceur, Dugas, Freeston, Leger, Gagnon, and Thibodeau (2000b) developed and evaluated an exposure-based treatment for generalized anxiety disorder in which

patients were exposed in imagination (via audio tape) to images of disastrous consequences that were the focus of their worries. At the same time, patients were instructed to resist performing any voluntary activity directed at neutralizing the upsetting images.

As we have described above, OCD and the other anxiety disorders share fundamental psychological processes such as overestimation of threat, avoidance, and safety-seeking behaviors. Moreover, OCD appears only to differ from other anxiety disorders at a descriptive level and in terms of the content or focus of the fear and the form of the safety-seeking behavior used to deal with the perceived threat. Accordingly, the effective cognitive-behavioral treatment procedures for OCD and each of the other anxiety disorders all contain elements of ERP. These procedures target a common essential underlying maintenance process: a self-perpetuating mechanism of dysfunctional catastrophic beliefs that produce anxiety and responses to anxiety that reinforce the erroneous beliefs. This fundamental maintenance process is no different in OCD than in other anxiety disorders.

SUMMARY AND CONCLUSIONS

Current trends in conceptualizing OCD within Psychiatry and Psychology increasingly emphasize a symptom-based checklist approach as is advocated by DSM diagnostic criteria. However, this focus on the superficial *form*, as opposed to the underlying *functional aspects*, of OCD symptoms has resulted in a cursory conceptualization of this problem as one characterized by repetitive or bizarre thought and behavioral patterns. This has led to the illusion that OCD overlaps with other disorders that involve repetitious behavior or bizarre thought.

In this chapter, we have argued that the essence of OCD is *not* to be found in the repetitive nature or bizarreness of obsessions or compulsions; instead, what is important about OCD is the cognitive mediation and functional relationship *between* these symptoms. There is compelling evidence that people with OCD experience thoughts that they misperceive as highly significant and foreboding of danger. They then engage in attempts to reduce the chances of danger, or control the thought itself, with tactics such as overt and covert (mental) compulsive rituals, avoidance, or other neutralizing strategies. These strategies become habitual because they result in immediate reduction of obsessional fear; yet in the long-term, they maintain the fear. This may give the illusion of a disorder involving behavioral inhibition or bizarre thought processes, yet research clearly shows that compulsive behavior is quite deliberate, that obsessional thoughts are similar in content to normal intrusions, and that anxiety leads to preoccupation with the feared stimulus (in this case, thoughts).

Not only are these key phenomenological processes internally valid and accessible via proper assessment, they are also similar to the processes involved with fears characteristic of other anxiety disorders such as social phobia, panic, and PTSD. In all of these conditions, there is the perception that some unlikely or un-costly feared outcome will occur and the use of maladaptive strategies to deal with the perceived threat. Although the focus of the fear and maladaptive safety behaviors vary across anxiety disorders (eg, in OCD it is intrusive thoughts, in social phobia it is social situations), the cognitive mediational processes overlap. Each anxiety disorder involves overestimates of threat and subsequent responses that prevent realization that the

fear is groundless. Moreover, treatment based on this conceptualization, which aims to demonstrate that appraisals of threat are incorrect, is highly effective for OCD and for other anxiety disorders. In conclusion, it is our position that OCD can mislead the clinician and/or researcher who is overly engrossed with overt signs and symptoms. Such a focus on repetition and bizarreness in OCD is as superficial as it is seductive. Instead, we stand to gain a greater understanding of this complex condition by turning our attention to the links between thinking and behavior, and understanding OCD as we do other anxiety-based disorders.

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Reply to Abramowitz and Deacon:

BEYOND ANXIETY: ETIOLOGICAL AND FUNCTIONAL OVERLAPS BETWEEN OCD AND OC SPECTRUM DISORDERS

Eric Hollander and Chin-Chin Yeh

Abramowitz and Deacon point out that OCD is an extremely complex disease that has so far defied a definitive categorization and is often described inaccurately. In DSM-IV, the American Psychiatric Association has defined OCD as a disorder that is characterized by obsessions, which are recurrent and intrusive thoughts, impulses, or images that cause marked distress, and/or compulsions, which are repetitive behaviors performed in response to an obsession (American Psychiatric Association, 2000). It is understandable why Abramowitz and Deacon have chosen to consider OCD as an anxiety disorder. Approaching this illness from the point of view of someone who practices cognitive-behavior therapy, OCD does respond to similar cognitive-behavioral techniques as other anxiety disorders, such as panic disorder or social phobia. However, in our understanding of the relationship between OCD and obsessive-compulsive spectrum disorders (OCSDs), we seek to find shared characteristics that illuminate the etiology of these disorders, and evidence suggests that a reconceptualization may be in order. It is, therefore, our belief that while OCD shares some symptom characteristics with other anxiety disorders (all share the symptom of anxiety), it might be conceptualized as its own category. Studies examining familial transmission, comorbidity, and treatment response support the idea that an obsessive-compulsive spectrum exists.

If there is any weakness to Abramowitz and Deacon's argument, it is one of exclusion. While Abramowitz and Deacon thoroughly explore the functional aspects of OCD, they have ignored the relationship between OCD and other disorders that bear similarities in comorbidity, family history, neurocircuitry, neurotransmitter function, and treatment response. These spectrum disorders were not included simply for their similarities in signs and symptoms, but because of similarities that suggest an underlying functional overlapping of these disorders. Recognition of these characteristics will allow us to better understand the etiology of these disorders and help us devise treatment plans with selective medications that are safe and effective.

One of the most compelling pieces of evidence that OCD is fundamentally different from other anxiety disorders involves the neurocircuitry. Neuroimaging studies have shown that OCD symptoms might result from alterations in the metabolic activity of the orbitofrontal-subcortical circuits (Saxena and Rauch, 2000). A lack of inhibition of the pathway leading from the cortex to the striatum to the globus pallidus-substantia nigra pars reticulata back to the cortex may cause the patient to become obsessed with danger, violence, hygiene, and sex. There is also evidence that abnormalities of the striatum are involved in dysfunction as well (Bartha et al. 1998). While the neurocircuitry of OCD appears to center on cortical regions, that of anxiety disorders involves the amygdala and limbic system. Studies have shown that there is a similarity between the physiological and behavioral response to a conditioned fear stimulus and that of panic attacks (Gorman, Kent, Sullivan, & Coplan, 2000). Both involve stimulation of the central nucleus of the amygdala, and it is hypothesized that panic disorder is a result of defects in the cortical processing pathways that lead to misinterpretation of sensory information and inappropriate activation of the "fear network." Rather than the increased frontal-cortical activity seen in OCD, these other anxiety disorders have characteristically decreased frontal activity.

Neuroimaging studies of OCSDs, in contrast, suggest that these disorders share similar neurobiological abnormalities as OCD. One study compared the basal ganglia volumes of 154 children and adults with TS with that of 130 healthy controls and found that volume of the caudate nucleus was decreased across all age groups in TS patients (Peterson et al. 2003). Another study compared the magnetic resonance imagings of 35 autistic subjects with that of 36 controls (Sears, Vest, Mohamed, Bailey, Ranson, & Piven, 1999). The results showed that the patients had significant enlargement of the caudate volume, which correlated with ritualistic and repetitive behavior.

Another point that Abramowitz and Deacon do not address is the high level of comorbidity that occurs in patients with OCD and/or OCSDs. This finding suggests that there is a relationship between OCD and these disorders that extends beyond mere coincidence. One study compared 36 OCD patients without comorbid putative OCSDs with 49 OCD patients with comorbid OCSDs (du Toit, van Kradenburg, Niehaus, & Stein, 2001). The study showed that lifetime prevalence rates of anorexia nervosa, bulimia nervosa, hypochondriasis, and compulsive buying tended to be higher in OCD patients, indicating that there is a relatively high rate of comorbidity between OCSDs and OCD.

Family history studies have also been performed that indicate a genetic component does exist between OCD and OCSDs. In the Hopkins Family Study, Bienvenu et al. (2000) investigated comorbidity and familial relationships between OCD and somatoform disorders (body dysmorphic disorder [BDD] and hypochondriasis), eating disorders (anorexia nervosa and bulimia nervosa), and pathologic "grooming" conditions (nail biting, skin picking, and trichotillomania). Using the Schedule for Affective Disorders and Schizophrenia Lifetime Anxiety, clinicians in this study interviewed 80 OCD patients and 343 of their first-degree relatives and 73 healthy controls and 300 of their first-degree relatives. The results showed that BDD, hypochondriasis, any eating disorder, and any grooming condition occurred more frequently in OCD patients than they did in healthy controls. Also, first-degree relatives of OCD patients exhibited higher rates of BDD, hypochondriasis, and any grooming condition regardless of the diagnosis of the patient. These findings indicate that certain somatoform and pathological grooming conditions may be part of a familial OCD spectrum.

Another study examined anticipation of age of onset in 40 probands affected with OCD and compared their age of onset with that of the parental generation (Cavallini, Albertazzi, Bianchi, & Bellodi, 2002). Anticipation has been documented in other illnesses and is believed to be caused by the extension of repeating trinucleotides that are passed on from one generation to the next. The results of this study showed that both OCD and TS patients have an earlier age of onset with succeeding generations. These findings suggest that these disorders may be heritable and share a common genetic etiology.

Another study examining morbidity risk also suggests that OCD and OCSDs share genetic similarities (Bellodi, Cavallini, Bertelli, Chiapparino, Riboldi, & Smeraldi, 2001). This study attempted to determine whether eating disorders, such as anorexia nervosa and bulimia, are familial by comparing the morbidity risk for OCSDs in first-degree relatives of 136 patients with eating disorders with first-degree relatives of 72 healthy controls. The results showed that morbidity risk was indeed higher in the families of patients. The study concluded that obsessive and compulsive signs should not be considered an additional diagnosis in patients with eating disorders, but as part of the eating disorder itself. These findings suggest that there is a genetic link between OCD and eating disorders, which should be considered part of a spectrum.

In their chapter, Abramowitz and Deacon argue that OCD rightfully belongs to the anxiety disorders category and thus should be distinguished from disorders that bear only a superficial resemblance of repetitive acts. They point out specifically the example of trichotillomania and contend that this illness is not a spectrum disorder because it does not exhibit the same obsessional anxiety that is characteristic in OCD. However, Lochner et al. (2002) have shown that trauma, and the anxiety that results from it, can lead to trichotillomania. In their study, the Childhood Trauma Questionnaire was administered to 74 OCD patients, 36 trichotillomania patients, and 31 normal controls to assess physical, emotional, and sexual abuse as well as physical and emotional neglect. The results revealed that childhood trauma was significantly higher in patients with OCD and trichotillomania, suggesting that trichotillomania may have a common etiology with OCD and can be affected by anxiety.

Anxiety also plays a role in other spectrum disorders, such as TS. One study hypothesized that comorbidity with anxiety disorders would predict tic severity in youths with TS (Coffey et al., 2000). These findings showed that anxiety disorder, particularly separation anxiety, may be significantly associated with tic severity in patients with TS. As the paper pointed out, the link between TS and anxiety disorders could have clinical implications and lead to appropriate intervention strategies that are aimed at reducing anxiety and that are different from those used to treat tic disorders. Understanding of these overlaps may eventually lead to better tic management.

In conclusion, the inclusion of these disorders on a spectrum related to OCD does not imply that they are identical to OCD. Indeed, the comorbidity and associated symptom domains among these disorders may make them distinct from each other. The use of a spectrum is an attempt to recognize that these disorders share various overlapping features that may help us illuminate both OCD and OCSDs and allow us to design better treatment strategies. Therefore, the category of an OC spectrum in DSM-V would not be meant to simply come up with a checklist of signs and symptoms, but to ultimately reflect etiology, including genetic, phenomenological, and neurobiological factors that may be common to these disorders.

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Reply to Hollander et al.:

THE OC SPECTRUM: A CLOSER LOOK AT THE ARGUMENTS AND THE DATA

Jonathan S. Abramowitz and Brett J. Deacon

Hollander et al.'s notion of an OCD spectrum contrasts sharply with our functionally based conceptualization of OCD as sharing fundamental characteristics with other anxiety disorders (which are conspicuously absent from Hollander et al.'s spectrum proposal). Accordingly, we believe Hollander et al.'s approach ignores critical similarities between OCD and other anxiety disorders that have implications for delivering effective treatment, and perhaps for understanding etiology. Moreover, their approach overlooks distinctions between OCD and proposed Obsessive-Compulsive Spectrum Disorders (OCSDs). Finally, most of the criteria Hollander et al. use for including disorders the OC spectrum lack sufficient specificity to tell us much about the shared etiology of OCD and proposed OCSDs. We expound upon these criticisms below.

YOU CANNOT JUDGE A BOOK . . .

Advocates of the OCD spectrum approach include disorders in the spectrum primarily on the basis of topographical similarities with OCD: namely all involve perseverative "obsessional" thinking and/or repetitive, stereotyped, "compulsive" behavior. For example, like checking in OCD, tics in Tourette Syndrome (TS) and hair pulling in trichotillomania (TTM) involve repetitious behaviors. One way of conceptualizing relationships between disorders is to focus on similarities in overt symptom presentation and speculate associations (or even etiological overlaps) on this basis. Unfortunately, though, this conceptual approach is superficial and it overlooks contributions by behavioral and cognitive psychology that have led to a richer understanding of OCD and putative OCSDs (as we have discussed in Chapter 6). In particular, careful studies of behavior and cognition have established that obsessional thoughts in OCD have characteristics that are unique (and readily identifiable) from symptoms of other problems sometimes colloquially labeled as "obsessive" (eg, jealousy). Obsessions in OCD are experienced as unwanted and intrusive, distressing, and they are resisted. Similarly, the repetitive behaviors in OCD have important characteristics that differentiate them from other repetitive behaviors sometimes colloquially labeled "compulsive" (eg, hair-pulling). In OCD, compulsions are performed deliberately to attain

reassurance and escape from obsessional fear and/or reduce the perceived probability of catastrophe.

Carefully conducted behavioral and cognitive research suggests that *some* OCSDs proposed by Hollander et al. do share characteristics with OCD. Body dysmorphic disorder (BDD), for example, involves anxiety-evoking thoughts about physical appearance and attempts to gain reassurance (eg, checking mirrors). Similarly, hypochondriasis (HC) involves illness-related fears and checking for reassurance of health status. However, this special relationship between anxiety-evoking thoughts and anxiety-reducing behavior is absent from the impulse control disorders, neurological disorders, and other body-focused disorders that Hollander et al. identify as related to OCD. Research that has closely examined urges to pull hair in TTM reveals that such urges are not precipitated by obsessional fears, but instead by feelings of general tension, depression, anger, boredom, frustration, indecision, or fatigue (Christenson, Ristvedt, & Mackenzie, 1993). Moreover, behavioral analysis shows that hair pulling leads to *pleasurable* feelings, a phenomenon not observed with rituals in OCD (Stanley, Swann, Bowers, & Davis, 1992).

Other impulse-control disorders show similar distinctions from OCD: their repetitive behaviors have qualitatively different functions than do compulsive rituals in OCD. Individuals with kleptomania report a "rush," "thrill," or "manic high" associated with their stealing and those with compulsive buying describe a "high like taking cocaine" when purchasing products (McElroy et al., 1995). Similarly, patients with pathological gambling report pleasure or gratification during and after gambling (Hollander & Wong, 1995). The drive to perform these behaviors, and the emotional experiences associated with their completion, are vastly different from those present in OCD. Researchers have also clearly differentiated compulsive rituals in OCD from tics (as in TS): whereas compulsive rituals are deliberate and serve as an escape from obsessional fear, tics are spontaneous (sudden), and performed to reduce sensory discomfort or tension (Miguel, Coffey, Baer, Savage, Rauch, & Jenike, 1995).

Whereas anxiety and guilt might be present in OCSDs to some extent (eg, people with kleptomania may feel guilty after stealing or anxious about being caught), Hollander et al.'s spectrum approach ignores critical differences in the cognitive mediation underlying anxiety in OCD and the various OCSDs. For example, thought–action fusion, the need to control unwanted thoughts, and an inflated sense of responsibility for harm play a role in the maintenance of obsessional anxiety in OCD (eg, Frost & Steketee, 2002; Salkovskis et al. 2000). However, these cognitive biases are irrelevant in OCSDs such as impulse control, neurological, and body-images disorders where qualitatively different cognitive factors (such as catastrophic thoughts about being perceived in a negative way) mediate any anxiety that might be present.

In delineating the OCD spectrum, Hollander et al. assume that relationships exist between OCD and the putative OCSDs because *repetition* is present, as opposed to a careful analysis of the underlying function and mediation of the repetitive behavior. The functional approach, which uses clinical experimentation to carefully examine the phenomenology (ie, antecedents and consequences) of cognition and behavior, provides greater resolution and specificity for understanding behavior disorders as compared to relying merely on overt and superficial behavioral manifestations or diagnostic criteria. We believe that if Hollander et al. more thoughtfully

embraced the behavioral analytic approach, and more cautiously examined the symptoms of putative OCSDs, they too would agree that impulse control disorders, eating disorders, and Tourette's, for example, bear little phenomenological similarity to OCD.

COMORBIDITY

Hollander et al. propose that OCSDs are related to OCD based on a high degree of comorbidity. However, several large-scale OCD comorbidity studies do not support the claim that patients with OCD often exhibit symptoms of the putative OCSDs. For example, in the Johns Hopkins OCD family study, Bienvenu et al. (2000) reported the following rates of OCSDs among 80 individuals with OCD: HC = 16%, BDD = 15%, anorexia nervosa = 9%, bulimia = 4%, TTM = 4%, kleptomania = 3%, pathologic gambling = 0%, and pyromania = 0%. In a larger study, Jaisoorya, Reddy, and Srinath (2003) found comorbidity rates of only 3% for TS, 3% for TTM, 0.4% for sexual compulsions, compulsive buying, and anorexia, and 0% for bulimia and depersonalization among 231 OCD patients.

We interpret the findings presented above to suggest OCSDs are quite *uncommon* among patients with OCD. This is perhaps more dramatically illustrated if we say that 97% of patients with OCD *did not* have Tourette's in the Jaisoorya et al. study. Interestingly, and consistent with the functional approach we advocate, HC and BDD appear to be exceptions. However, even if we grant that the OCSD comorbidity rates are higher than chance, a more serious problem for the OC spectrum notion is that comorbidity rates between OCD and anxiety and mood disorders are considerably higher. For example, data from the Hopkins Study indicate that 13% of OCD patients also met criteria for generalized anxiety disorder, 20.8% met criteria for panic disorder, 16.7% for agoraphobia, 36% for social phobia, 30.7% for specific phobias, and 54.1% for recurrent major depression (Nestadt et al., 2001). Therefore, the comorbidity argument that Hollander et al. use to support their contention that OCSDs are etiologically related to OCD is actually more in line with our own view that OCD is more closely related to other anxiety disorders (on the order of 5- to 10-fold closer).

Another problem with Hollander et al's appeal to comorbidity is that although comorbidity signifies a relationship between disorders at *some* level, this relationship may not be etiologically meaningful. Indeed, comorbidity is common in most major mental disorders and there are numerous explanations for this phenomenon that do not require co-occurring conditions to be considered part of the same spectrum. For instance, alcohol dependence, depression, and PTSD are all more highly comorbid than with one another would be expected by chance. While it is easy to recognize several potential reasons for the co-occurrence of these disorders, few would suggest alcohol dependence, depression, and PTSD are part of the same spectrum (PTSDSDs?). Not surprisingly, Summerfeldt, Hood, Antony, Richter, and Swinson (2004) found that although OCD was associated with elevated levels of impulsivity compared to nonclinical controls, this relationship was not unique to OCD since all of the anxiety disorders showed increased impulsivity relative to the control group. All of this data suggest comorbidity is of limited value in understanding the uniqueness of OCD and its links to OCSDs.

FAMILY HISTORY

A similar argument applies to Hollander et al.'s claim that OCD has a familial or genetic association with OCSDs. Not only is this association admittedly unclear for several proposed OCSDs, but the rates of other anxiety disorders among first degree relatives of people with OCD are far higher than the rates of OCSDs among relatives of OCD sufferers (eg, Bienvenu et al., 2000; Nestadt et al., 2001). Therefore, Hollander et al.'s assertion that familial pattern is good evidence for a relationship between OCD and other disorders actually supports the notion that OCD is much more strongly related to other anxiety disorders than to proposed OCSDs. These data are also more consistent with the notion of shared genetic vulnerability among OCD and the other anxiety disorders, as opposed to a genetic link between OCD and the putative OCSDs.

NEUROCIRCUITRY

In their chapter, Hollander et al. contend that functional neuroimaging research has elucidated the neurocircuitry underlying OCD, but that few studies have addressed the neurocircuitry of OCSDs. If so, we ought not to be making broad conclusions about similarities in neurocircuitry until more data have been collected. Preferentially, this data should come from controlled studies that directly compare brain regions of interest in OCD and OCSDs. Such studies are scarce, and most of the existing research is based on very small sample sizes as Hollander et al. describe (eg, Seeger, Braus, Ruf, Goldberger, & Schmidt, 2002). Moreover, two magnetic resonance imaging studies of TTM (O'Sullivan et al., 1997; Stein et al., 1997) found results that were inconsistent with magnetic resonance imaging studies of OCD patients.

A second concern is how Hollander et al. have interpreted the available functional neuroimaging data. They imply that such data indicate the presence of "abnormalities," "imbalances," and "defects" that play a role in the etiology of OCD. However, these neuroimaging studies are cross-sectional and they only present data on observed *differences* between people with and without OCD. Data from cross-sectional studies address only whether the identified brain regions are in *some sense involved* in OCD; such study designs do not permit one to conclude that observed differences are related to etiology. To infer such a causal relationship requires prospective research in which brain activity in specific regions of interest are experimentally manipulated, resulting in the development (or exacerbation) of symptoms; and this has not been done. In the absence of such prospective studies, conclusions regarding OCD and neuroimaging findings must be restricted to those allowed by correlational data. Therefore, three possible explanations for the current findings are: (*a*) alterations in functioning in certain brain regions cause OCD; (*b*) OCD causes alterations in functioning as observed in certain brain regions; or (*c*) a third variable causes both phenomena. Interestingly, data from symptom provocation studies, which measure the effects of the environment on the brain in a prospective fashion, indicate that OCD patients and non-patients both evidence higher regional cerebral blood flow when exposed to anxiety-evoking, as opposed to neutral, stimuli (eg, Cottraux et al., 1996). This leads to the conclusion that increased brain activity in OCD patients compared to controls (eg, Rauch et al., 1994) is merely due to the differences in state and trait anxiety between individuals with and without OCD. Thus,

given the inconsistent findings, paucity of comparison studies, and misinterpretation of neuropsychiatric data, the case for inclusion of OCSDs on the basis of common neurocircuitry is unconvincing.

NEUROTRANSMITTER FUNCTION

Hollander et al.'s position that a relationship between OCD and OCSDs exists on the basis of an overlap in abnormal neurotransmitter function also suffers from weaknesses. We agree that the most consistent data relevant to this issue come from treatment studies; indeed, the findings from biological marker and pharmacological challenge studies of the serotonin system in OCD have been remarkably inconsistent (Gross, Sasson, Chorpa, & Zohar, 1998). However, the appeal to similarly selective responses to serotonin reuptake inhibitor medication (SRIs) is only a compelling argument for the OC spectrum if this response profile is both sensitive and specific to OCD and OCSDs. If other, non-OC spectrum, disorders respond selectively to SRIs, than this appeal to neurotransmission is of little value as a means of identifying OCSDs. Ironically, an important scientific problem with SRIs is that they appear to improve such a wide variety of disorders (including most anxiety disorders and depression) that there is little chance of meaningful pharmacological dissection of disorders with them.

A related issue is that OCD's (and the OCSDs') preferential response to SRIs does not indicate than an *abnormally* functioning serotonin system is involved in the cause of OCD (or OCSDs). This is because specific models of etiology cannot be derived solely from knowledge of successful treatment response. Inferring such a relationship is an example of the logical error known as *ex juvantibus* reasoning, or "reasoning backward from what helps," (a variation of the fallacy known as *post hoc ergo propter hoc*, or "after this, therefore because of this") and represents a gross oversimplification of how neurotransmitters (and SRIs) work. The problem with such reasoning is clear in the following example: "*When I take aspirin, my headache goes away. Therefore, the reason I get headaches is that my aspirin level is too low.*"

Just as there may be many possible mechanisms by which aspirin makes headaches go away, there may be many possible interacting mechanisms by which SRIs decrease OCD symptoms. From an epistemological standpoint, successful response to a treatment derived from a particular conceptual framework may in some instances provide *clues* to etiology; however, definitive conclusions regarding causes of, and relationships between, disorders are generally not warranted on the basis of treatment response. Undoubtedly, the behavior observed in OCD and putative OCSDs *involves* the serotonin system (one is hard pressed to identify many human processes that do not); yet existing evidence suggests neither that these problems are *caused* by an abnormal serotonin system, nor that an overlap in serotonin involvement justifies a spectrum.

TREATMENT RESPONSE

Finally, we come to treatment response, which should be a litmus test for any OCD spectrum proposals as it is an ultimately successful treatment that we seek by dealing with matters of phenomenology and etiology. Here again, Hollander et

al.'s OC spectrum proposal encounters serious problems and may even lead people with OCD *away from* the best treatment for their condition. Hollander et al. strongly tout evidence for a preferential response to SRIs in OCD and the OCSDs as supporting the spectrum conceptualization. However, the appeal to this preferential response is only clinically useful in delineating an OCD spectrum if three conditions are met: (*a*) preferential response to SRIs is observed uniformly in OCD and the OCSDs, (*b*) the preferential response to SRIs is not observed in other disorders that are not characterized as OCSDs, and (*c*) SRIs are the best treatment available for OCD and OCSDs. Unfortunately, none of these parameters have empirical support.

First, whereas OCD responds preferentially to SRIs, the claim of a similar preferential response across the OCSDs is not supported by the data. Very few controlled double-blind studies in which an SRI and non-SRI are directly compared have been reported for the various OCSDs. This means that the assertion of preferential treatment response in most OCSDs is based on open-trial study results that are not designed to answer the question of relative efficacy of medication. We find it troubling that such broad speculations about treatment response are made, given the lack of convincing data and implications for clinical management. A close look at the data even suggests that non-SRIs are helpful for many of the proposed OCSDs such as kleptomania (McElroy et al., 1995), compulsive shopping (McElroy et al., 1991), and pathological gambling (Moskowitz, 1980) to name a few. In addition, neuroleptic medications (eg, haldol) that are ineffective as monotherapies for OCD are often used in the treatment of TS (Leckman, Hardin, Riddle, Stevenson, Ort, & Cohen, 1991).

Second, SRIs demonstrate at least equivalent efficacy to other medications in the treatment of depressive disorders (eg, Nemeroff & Schatzberg, 1998) and other anxiety disorders including panic disorder (eg, Boyer, 1995) that are left out of Hollander et al.'s OCD spectrum. Here again, the spectrum argument runs into the lack of specificity problem: because SRIs help so many disorders, the observation that a group of disorders responds preferentially to these drugs does not tell us anything special about these disorders.

Third, it is now well acknowledged that cognitive-behavior therapy using exposure and response prevention (ERP) is the most effective treatment for OCD (Foa et al., 2005 Jenike, 2004; Kozak, Liebowitz, & Foa, 2000; see Kozak & Coles, Chapter 15). Not only is ERP more effective than SRIs (average symptom reduction rates for ERP are 60–70% versus only 20–40% with SRIs; Jenike, 2004), but also ERP was developed from a specific and empirically demonstrated conceptualization of OCD as an anxiety disorder in which compulsive rituals are performed to reduce inappropriate fear of obsessional stimuli. Because ERP is based on the specific relationship between obsessional fear and compulsive behavior, this treatment is irrelevant for most OCSDs proposed by Hollander et al. For example, because TTM involves neither obsessional fears nor urges to perform compulsive rituals designed to escape or neutralize anxiety, there would be no logic in using ERP in the treatment of this disorder. Hair pulling in TTM is not evoked by obsessional anxiety, but rather by general tension, fatigue, or boredom. Hair pulling in TTM is also not performed to reduce the probability of danger, as is observed with compulsions in OCD.

As can be seen, TTM involves considerably different behavioral mechanisms than does OCD. Thus, the therapeutic procedures used in reducing this behavior are necessarily different. Functional analysis logically leads to the use of treatment procedures

that hinder attempts to pull (stimulus control), such as wearing mittens, covering hair, or remaining around other people. Procedures that compete with pulling, such as handling a rubber ball, are also implemented along with repeated practice in "high risk" situations. Finally, procedures that help patients avoid strong urges to pull (eg, avoidance of cues, relaxation training) are employed. Similar procedures that aim to complicate the performance of specific undesirable behaviors are used to reduce other disorders of impulse control such as binge eating, pathological gambling, and pathological sexual behavior. Behavioral therapy for alcohol and other forms of substance abuse (which are curiously left out of the OCD spectrum) also features similar procedures.

CONCLUSIONS

As we have shown, Hollander et al.'s case for the OC spectrum is undermined by (*a*) the lack of a fine-grained conceptualization of OCD and OCSDs (which would clearly differentiate these conditions from one another on a phenomenological basis), (*b*) reliance on high base rates of overlapping features with poor sensitivity and specificity (eg, comorbidity, response to SRI medications), (*c*) misinterpretation of data from neuroimaging and treatment outcome research, and (*d*) omission of data that largely do not support the spectrum concept (eg, differential response to ERP). Because of this, Hollander et al.'s OCSD model is over-inclusive. A more valid means of generating an OC spectrum would be to start by identifying the fundamental (specific) phenomenological features of OCD, such as the functional relationship between anxiety-evoking stimuli and feared consequences on the one hand, and strategies used to reduce anxiety on the other hand. Next, *a priori* hypotheses about similarities with other disorders could be examined experimentally to determine which disorders share these features. Applying this more cautious approach would likely lead to inclusion of very few of the OCSDs proposed by Hollander et al. (perhaps only BDD and HC). Moreover, the phenomenological similarities between OCD and other anxiety disorders would become more apparent.

We therefore have little confidence that the OCD spectrum model proposed by Hollander et al. would help clarify the etiology and treatment of OCD or putative OCSDs. Furthermore, we see little utility in reclassifying OCD, or reclassifying other disorder as OCSDs. If, as we have argued in Chapter 6, OCD and the other anxiety disorders all involve problems with catastrophic misinterpretations of objectively nondangerous stimuli and counterproductive attempts to deal with the resulting anxiety, then a number of common processes across these problems (which do not turn up as key features of other disorders) would be expected. Despite expected topographic variability depending on the nature of the feared stimuli, these processes should include phenomena such as selective attention to threat cues, passive avoidance of threat cues, safety-seeking behaviors, and response to treatment using exposure to fear cues and prevention of safety-seeking behaviors. These processes are observed across the anxiety disorders (and perhaps BDD and HC), but not in the other putative OCSDs.

One final comment. Hollander et al.'s over-inclusive OCSD model also presents problems for clinical management and perpetuates the notion that OCD is about repetitive behavior. Individuals who present themselves at OCD treatment programs convinced

their "obsessive" jealousy or anger, or their "compulsive" eating, hair-pulling, masturbation, gambling, or nail biting are forms of OCD, and should be treated as such, invoke the OC spectrum concept in support of their convictions. For example, we routinely receive referrals from health care providers who cite the spectrum notion when referring a wide variety of problems for the treatment of "OCD." An unfortunate perception among many consumers and treatment providers is that the data supporting the efficacy of SRIs and ERP for OCD are similarly applicable to the proposed OCSDs. Yet as we have seen, this is not the case.

EDITOR'S NOTE

Because such a wide scope of disorders has been proposed as part of the obsessive-compulsive spectrum, we have included six brief chapters that specifically address a number of these conditions. Authors of each chapter were asked to evaluate and comment on the relationship between OCD and the putative spectrum condition. Chapters in this section address trichotillomania, compulsive shopping, hypochondriasis and body dysmorphic disorder, TS, and compulsive sexual behavior.

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Chapter 7

TRICHOTILLOMANIA: AN OBSESSIVE-COMPULSIVE SPECTRUM DISORDER?

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In this chapter, we argue that it is a useful heuristic to conceptualize trichotillomania (TTM) as belonging to an obsessive-compulsive spectrum of disorders. Viewing TTM from this perspective provides the researcher with several fertile hypotheses with which to explore the psychobiology of this condition, and it provides the clinician with useful potential strategies for assessment and treatment. At the same time, viewing TTM as *related* to obsessive-compulsive disorder (OCD) does not in any way imply that there are not crucial differences between the symptoms, pathogenesis, and management of the two disorders. Similarly, a view of TTM as an OCD spectrum condition does not exclude the possibility that TTM may be more closely related to a range of other conditions that are characterized by stereotypic or self-injurious behaviors, than it is to OCD.

HISTORICAL PERSPECTIVE

Hair-pulling has long been ascribed to frustration and grief, with depictions of such behavior in the bible, Homer, and Shakespeare (Christenson & Mansueto, 1999). Similarly, Hippocrates advised clinicians to include hair-pulling in their routine mental health examination, and described a patient with hair-pulling in the apparent context of depression. The first detailed case report of pathological hair-pulling, and the coining of the term "trichotillomania," came towards the end of the 19th century (Hallopeau, 1889). Perhaps the first systematic study relevant to hair-pulling was conducted in 1939, and discussed 311 patients (DeBakey & Ochsner, 1939). However, this paper was authored by surgeons primarily interested in the gastro-intestinal sequelae of trichophagia (the eating of one's hair), and gave short shrift to the phenomenology and treatment of hair-pulling.

Much of the subsequent literature was comprised of case reports and small series. A particularly important approach to hair-pulling emerged from the work of Azrin, Nunn, and colleagues (Azrin & Nunn, 1973; Azrin, Nunn, & Frantz, 1980) on a method of "habit reversal" for decreasing a range of unwanted repetitive habits. Their conceptual framework included hair-pulling as one of many habits that patients may suffer from. This spectrum of habits included tics, and is therefore arguably relevant to the current interest in TTM as an obsessive-compulsive spectrum disorder today. Nevertheless, these authors were more interested in stereotypic behaviors per se, than in classical obsessions and compulsions. This work continues to influence current approaches to the cognitive-behavioral treatment of TTM (Keuthen, Aronowitz, Badenoch, & Wilhelm, 1999; Stemberger, Stein, & Mansueto, 2003).

In 1980, TTM was included in the DSM system, but arguably the most important impetus to research on TTM came later that decade, when researchers with interests in OCD raised the question of whether medications that had recently been found useful for the repetitive symptoms of that disorder, could also be effective in TTM. Whereas depression responded to both clomipramine, a serotonin reuptake inhibitor (SRIs), and to desipramine, a noradrenaline reuptake inhibitor, OCD was unusual in that it responded much more robustly to clomipramine than desiprame (Zohar & Insel, 1987). Swedo and colleagues reported that TTM, like OCD, showed a significantly more robust response to clomipramine (Swedo et al., 1989).

This report was seminal in raising the interests of clinicians and of consumer advocates in TTM, in suggesting that TTM (like OCD) was a disorder with particular psychobiological underpinnings, and in proposing a novel line of intervention for its management. Indeed, the finding that TTM responded selectively to clomipramine helped trigger a range of subsequent studies on TTM, a number of which specifically attempted to address the question of its relationship to OCD (Stein, Simeon, Cohen, & Hollander, 1995; Swedo, 1993). It is important to emphasize that Swedo and colleagues were not arguing that TTM was a form of OCD per se; nevertheless, they had developed a perspective which continues to be useful in thinking about new studies of people with TTM and in working with patients who are seeking help for hair-pulling.

SYMPTOMATOLOGY

The symptoms of TTM and OCD are in one way entirely different; OCD may be characterized by a range of obsessions and compulsions, whereas TTM does not typically involve obsessions and the behaviors focus primarily on the activity of hair-pulling. Some authors have suggested that hair-pulling is more reminiscent of comorbid tics in OCD than of compulsions per se. OCD symptoms are often precipitated by exposure to feared stimuli (eg, sources of contamination), whereas TTM is frequently precipitated by particular affective states (eg, boredom) (Christenson, Ristvedt, & Mackenzie, 1993). In our experience, whereas OCD frequently occurs at different times of the day, TTM is often worse at night.

At the same time, there are some important similarities between the symptoms of OCD and TTM. Both compulsions and hair-pulling are repetitive, unwanted, and ritualistic (hair-pulling rituals may include playing with the hair, selecting a hair to pull, the pulling itself, and then mouthing, biting, swallowing, or other disposal of the hair). Both can be preceded by an urge; in OCD obsessions triggers compulsions,

while in TTM there is frequently a preceding somatic sensation (eg, scalp itchiness) or an urge to pull out hair. Symptom dimensions such as time spent, accompanying distress when the behavior is prevented, and lack of control, all of which are reliably anchored by the Yale-Brown Obsessive-Compulsive Scale (Goodman et al., 1989), are useful in assessing the severity of both OCD and TTM.

Both OCD and TTM can involve a concern with symmetry. Some OCD patients wash symmetrically or have multiple ordering and arranging compulsions while some hair-pulling patients carefully pull on both sides of the scalp or carefully ensure that eyebrows or eyelashes are symmetrical. Both conditions are characterized by a sense of shame and embarrassment; the person with OCD worries that people will think him or her crazy because the compulsions are disproportionate to reality and the person with TTM berates himself or herself for the fact that their hair-pulling is self-inflicted. This self-blame not only exacerbates accompanying distress, but contributes to delayed help-seeking (Seedat & Stein, 1998; Soriano et al., 1996).

A number of other clinical features deserve mention in considering the relationship between OCD and TTM. Both disorders have a prevalence of around 2% or more (Christenson & Mansueto, 1999). Prevalence of both conditions in dermatology clinics may be even higher. Infant hair-pulling and toddler or early child compulsions are normal phenomena that subsequently often disappear. Hair-pulling most commonly begins at the time of puberty, with OCD typically beginning either somewhat earlier or later than puberty, but also during or after pregnancy. Hair-pulling is significantly more common in females, but in both early onset OCD and early onset TTM, the relative proportion of males increases. Both disorders are seen in all socio-economic classes and in all ethnic groups (although rigorous and representative community surveys of TTM remain to be done).

Comorbidity with other psychiatric conditions is high in both disorders (Christenson & Mansueto, 1999). In particular, both disorders frequently have comorbid mood, anxiety, eating, and substance use disorders. Lower comorbidity in childhood and adolescent samples of both OCD and TTM suggests the possibility that some of the comorbidity results as a sequel of the primary condition, and might be prevented by earlier, more rigorous intervention. There is also some evidence that certain personality disorders are increased in both disorders, although this question has not been as well studied in TTM and it is difficult to reach firm conclusions at this point in time. Small studies directly comparing comorbid symptoms and disorders across the two disorders have, however, indicated that comorbidity is higher in OCD than in TTM (Himle, Bordnick, & Thyer, 1995; Stanley, Swann, Bowers, Davis, & Taylor, 1992; Tukel, Keser, Karali, Olgun, & Calikusu, 2001).

While many psychiatric disorders are disabling, it is relevant to note that the prevalence, chronicity, comorbidity, and morbidity associated with both OCD and TTM have often been underestimated. There is growing evidence that the costs of OCD, particularly in terms of functional impairment and associated disability, are amongst the highest of those associated with any general medical disorder (Mogotsi, Kaminer, & Stein, 2000). TTM, while perhaps not as disabling as OCD, can be associated with surprisingly high levels of distress and impairment (Seedat & Stein 1998; Soriano & Stein, 1996) and trichophagy may have fatal consequences (Bouwer & Stein, 1998). These findings highlight the need for increased community outreach programs to ensure earlier diagnosis and treatment of both disorders.

PSYCHOBIOLOGY

The psychobiology of OCD has been more frequently and rigorously investigated than that of TTM, so that drawing definitive conclusions about the relevant similarities and differences between these two conditions ultimately requires much more additional study. Nevertheless, based on the limited set of studies to date, a number of preliminary comments can be made about the neuroanatomy, neurochemistry, neuroimmunology, and neurogenetics of OCD and TTM. In our view, these studies provide some support for the argument that there is a relationship between OCD and TTM, although the two disorders are distinct in many respects, and TTM may ultimately be best conceptualized as lying on a spectrum of stereotypic or self-injurious conditions.

There is growing evidence of the importance of corticostriatal-thalamic circuits in OCD (Rauch & Baxter, 1998). A range of different data point to this, but perhaps the most persuasive is from structural and functional brain imaging. There is increased activity in corticostriatal-thalamic circuits prior to treatment and a number of studies have found changes in caudate volume in OCD subjects. In TTM, there is evidence of decreased volume in the left putamen (O'Sullivan et al., 1997) but not in the caudate (O'Sullivan et al., 1997; Stein, Coetzer, Lee, Davids, & Bouwer, 1997); and this is consistent with the more motoric nature of hair-pulling symptoms. During treatment with either SRIs or cognitive-behavioral therapy (CBT), there is normalization of neuronal activity in OCD. Similarly, during SRI treatment of TTM, there is normalization of perfusion in frontal circuits (Stein et al., 2002). Furthermore, although baseline functional imaging seems to differ in OCD and TTM, decreased frontal activity may predict response to SRIs in both disorders (Stein et al., 2002; Swedo, Rapoport, Leonard, & et al., 1991).

Brain imaging studies are consistent with much earlier work demonstrating that OCD can be associated with a range of neurological insults to the basal ganglia (Cheyette & Cummings, 1995; Cummings & Cunningham, 1992), and that conversely, patients with OCD demonstrate specific neuropsychiatric and neuropsychological impairments. There is much smaller literature on neurological lesions resulting in hair-pulling symptoms, on neurological soft signs in TTM, and on neuropsychological impairment in these patients, therefore, conclusions cannot be drawn with certainty at this stage. Nevertheless, the work on TTM does provide some evidence (including data on visual-spatial impairment in TTM) to support the hypothesis that as in the case of OCD, corticostriatal-thalamic circuits do play a role in underpinning TTM symptoms (Stein, O'Sullivan, & Hollander, 1999).

Corticostriatal-thalamic circuitry incorporates a range of different neurotransmitter systems including the serotonin and dopamine systems. The selective response to serotonergic agents has led to a great deal of work on the role of the serotonin system in OCD. Although there is little definitive evidence that serotonergic dysfunction underlies OCD, it is clear that the serotonin system plays a key role in mediating symptoms. During effective treatment with SRIs, for example, there is a decrease in cerebrospinal fluid levels of 5-hydroxyindoleacetic acid, a primary metabolite of serotonin (Thoren, Asberg, & Bertilsson, 1980). This is reminiscent of the finding that high cerebrospinal fluid 5-hydroxyindoleacetic acid predicts response to SSRIs in TTM (Ninan, Rothbaum, Stipetic, & et al., 1992). There is some evidence that administration of the

serotonin agonist metachlorophenylpiperazine results in symptom exacerbation in a proportion of OCD patients, but in a "high" feeling in TTM, suggesting overlapping but differential involvement of the serotonin system in both disorders (Stein et al., 1995).

Dopamine also plays a role in mediating OCD. Dopaminergic agonists can exacerbate compulsions, and dopamine blockers are used to augment SSRIs in the treatment of OCD (Goodman, McDougle, & Lawrence, 1990). The dopaminergic system is strongly implicated in Tourette's disorder (TS), and in OCD patients with tics there is evidence of worse response to SSRIs, but responsivity to the combination of a dopamine blocker and an SSRI (Hawkridge, Stein, & Bouwer, 1996; McDougle, Goodman, & Leckman, 1994). Similarly, in TTM, there is exacerbation of hair-pulling by dopamine agonists, and dopamine blockers can be useful in augmenting the treatment response to SSRIs (Stein et al., 1997). Although hair-pulling is a common comorbid symptom in TS, tics are less common in TTM than in OCD (Lochner et al., unpublished data). On the other hand, when prescribed without SSRIs, dopamine blockers do not appear effective for OCD, but may be useful in TTM (Stewart & Nejtek, 2003).

A broad range of neurochemical systems other than serotonin and dopamine may mediate both OCD and TTM. The role of steroidal hormones is suggested by data such as the frequent onset of OCD during or after pregnancy, and the common exacerbation of both OCD and TTM symptoms during menstruation. The opioid system has also been implicated in both conditions. Both disorders also deserve more study with regard to a range of different neuropeptides that may play a role in mediating stereotypic behavior (Leckman, Goodman, & North, 1994). Ultimately, it will be necessary to characterize the second and third messenger pathways involved in the mediation of these and other OCD spectrum conditions.

An ultimate goal of research in this area is to determine the precise genetic and environmental factors that cause disruption in CTSC circuits. There is good evidence from family studies for the heritability of OCD, but the heritability of TTM remains unclear. Nevertheless, there is also some evidence for increased prevalence of OCD in the families of TTM probands. Ultimately, the specific genetic variants that may contribute to OCD and TTM need to be determined. A recent report noting that a homeobox gene is required for the mediation of grooming behavior in rodents certainly encourages such work to proceed (Greer & Capecchi, 2002). Genetic factors may also play a role in the susceptibility of certain species of animal to develop grooming problems, including hair-pulling and feather-picking (Hugo et al., 2003).

Autoimmunity has recently been hypothesized to contribute to CTSC damage in OCD and spectrum disorders. This idea is based on the observation that OCD symptoms and tics may develop after Streptococcal infection (Leonard & Swedo, 2001). Such patients may have elevated expression of a marker of susceptibility to rheumatic fever, the B lymphocyte antigen D8/17. To date there is no evidence that D8/17 is higher in OCD than in TTM and healthy controls (Niehaus et al., 1999). However, there is some evidence that hair-pulling may relapse after streptococcal infection, and an interesting case report documented the onset of hair-pulling in the context of Sydenham's chorea. The question of whether particular genetic variables contribute to vulnerability for such auto-immune processes, the extent to which D8/17

is a valid marker, and the proportion of OCD or TTM cases in which auto-immunity plays a role, remains to be clarified.

Although recent literature has focused on neuropsychiatric factors in OCD, a possible role for psychological factors in precipitating or exacerbating symptoms should not be ignored. Psychodynamic theories emphasizing the role of such factors have not, however, received a great deal of empirical attention. Data from our group found that scores on a childhood trauma scale were increased in OCD and TTM compared with normal controls, and this issue therefore requires further study (Lochner et al., 2002). There is growing recognition that adverse childhood environments may be associated with specific neurobiological sequelae, and it is possible that these in turn are associated with vulnerability to the development of stereotypic symptoms (Martin, Spicer, Lewis, Gluck, & Cork, 1991).

TREATMENT

OCD responds in 40–60% of cases to treatment with an SSRI. In TTM, open-label studies of SSRIs were promising, but controlled studies have yielded negative results (O'Sullivan, Christenson, & Stein, 1999). Nevertheless, it is possible that a subgroup of patients with TTM does respond to treatment with these agents, and they continue to be used in clinical practice. Although the early report by Swedo and colleagues has not been replicated, it is possible that clomipramine is particularly useful in TTM. Interestingly, there is also some evidence from metaanalyses of the OCD clinical trials database that clomipramine is also particularly effective in that disorder (Stein, Spadaccini, & Hollander, 1995). While the superior efficacy of clomipramine in OCD and TTM is far from proven, it is interesting to speculate that the relatively non-specific actions of this agent (including dopaminergic effects) may contribute to its efficacy.

Treatment efficacy is typically maintained over time in OCD. Nevertheless, there are some patients in whom response to SSRI "poops out." Again, in TTM, the data are more inconsistent; although there are some data that response is maintained, there is also an impression that early response to SSRI is often lost over time (O'Sullivan et al., 1999). Should future prospective studies confirm that the duration of response to pharmacotherapy differs in OCD and TTM, this would again provide an interesting departure point for considering the exact range of overlapping and distinguishing features that characterizes the neurobiological intersection between these conditions. Some authors have argued that loss of response to SSRIs over time is seen in those disorders that lie on the more impulsive pole of the putative compulsive-impulsive OCD spectrum of conditions.

Augmentation with antipsychotic medication is useful in ~50% of OCD cases (McDougle, Epperson, Pelton, & et al., 2000). There is some evidence from case studies and series that a similar strategy may also work in TTM, although further work is needed (O'Sullivan et al., 1999). It is possible that whereas refractory OCD patients, or OCD patients with tics, respond to a combination of serotonergic and dopaminergic agents, in TTM there is a response to dopaminergic drugs alone (Stewart & Nejtek, 2003). Such data once again underscore the central contention of this chapter; that although OCD and TTM are clearly not the same phenomenon, work on one disorder may be useful in informing clinical practice and research studies on the other.

What about psychotherapy? The best studied intervention for OCD is exposure and response prevention, although cognitive techniques may also be effective. In TTM, the principles of cognitive-behavioral therapy are rather different, with the emphasis instead on habit reversal as well as a range of associated techniques (Keuthen et al., 1999; Stemberger et al., 2003). These differences reflect the possibility that in many cases of OCD, negative reinforcement is important; whereas in many (but not all) cases of TTM, positive reinforcement is key. At the same time, there is also some overlap in the cognitive-behavioral therapy techniques used to treat these conditions; self-monitoring, for example, may play an especially important role in the cognitive-behavioral therapy of both OCD and TTM. Whereas pharmacotherapy and psychotherapy for OCD may be similarly effective, in TTM there is some evidence for the relative superiority of behavioral therapy (Minnen, Hoogduin, Keijsers, Hellenbrand, & Hendriks, 2003).

CONCLUSION

Clearly, OCD and TTM are two entirely different disorders. Nevertheless, they are characterized by a range of overlapping phenomenological and psychobiological features, and approaches to the assessment and treatment of the two conditions can usefully inform one another. At the same time, however, the extent of overlap between OCD and TTM may, in the larger scheme of things, be relatively small. In our experience, TTM patients feel uncomfortable participating in OCD self-help groups, and vice versa, highlighting the differences in phenomenology between these two conditions. There are also crucial differences in pharmacotherapeutic and psychotherapeutic approaches to OCD and TTM. It is also important to emphasize the heterogeneity of both disorders; this chapter has not addressed in detail the possibility that certain subgroups of OCD and TTM (du Toit, van Kradenburg, Niehaus, & Stein, 2001) have a particularly close relationship.

Trichotillomania may be related more closely to other disorders characterized by stereotypic and self-injurious behaviors than to OCD. In particular, there is phenomenological overlap between TTM, skin-picking, and stereotypic movement disorder in adults of normal intelligence (Lochner, Simeon, Niehaus, & Stein, 2002). Significantly, the prevalence of stereotypic behaviors other than hair-pulling in TTM is high. There are also important neurobiological and treatment overlaps across these conditions (Stein & Simeon, 1998), although much remains to characterize them further. It is also worth considering the phenonomenology and psychobiology overlaps and contrasts between TTM, impulsive symptoms, and the other impulse control disorders (eg, pathological gambling) (Stein et al., 1995).

In our view, it is useful to consider TTM and related stereotypic disorders, as forming one pole of an OCD spectrum of disorders. This provides a heuristic framework for both research studies and clinical intervention. Indeed, this framework has already provided the impetus for a range of studies on the neurobiology and pharmacotherapy of TTM. Without this framework, data such as those on decreased putamen volume in TTM would likely not have been sought or found. Similarly, investigators may well have been slower to explore the role of antipsychotic agents in the treatment of TTM. In the future, when more is known about the neurobiology of both OCD and the stereotypic disorders (such as skin-picking), this heuristic may,

however, no longer be useful and may well need to be replaced with a different framework.

Is it time to consider removing OCD from the anxiety disorders, and including it together with TTM, TS and a number of other conditions in an obsessive-compulsive spectrum section of DSM? Clearly, the current DSM classification reflects historical contingencies, and many have argued that OCD is not an anxiety disorder (Montgomery, 1993). Although the architects of DSM-IV did make changes to DSM-III-R on the basis of new evidence, the kind of evidence needed for moving disorders from one section to another was not specified particularly rigorously, and it is therefore difficult to provide an unequivocal answer to this question.

Nevertheless, a section on OCD spectrum disorders would remind clinicians of the phenomenological and psychobiological overlaps between, say, OCD and TS. It would remind clinicians to assess TTM in OCD and TS, to assess skin-picking and OCD in body dysmorphic disorder, and so on. In our experience, many TTM patients appreciate learning about the concept of an OCD spectrum, this helps destigmatize their symptoms, and helps them combat self-blame. Finally, the spectrum concept helps clinicians to think about management approaches. Despite the lacklustre performance of SSRIs in TTM, they are still useful treatment option for some patients. Dopamine blockers are an important option in OCD, TTM, and a number of spectrum disorders. Cognitive-behavioral therapy is crucial in both OCD and TTM, and many OCD therapists are also skilled in the treatment of TTM. Thus, combining TTM and OCD in a single section in future classification schemes has some appeal.

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Chapter 8

OVERLAP OF BODY DYSMORPHIC DISORDER AND HYPOCHONDRIASIS WITH OCD

Fugen Neziroglu and Sony Khemlani-Patel

The development of the obsessive-compulsive spectrum disorders as a group of conditions linked by similar core symptomatology has inspired much debate among scholars. In light of the current trend to seek empirically validated and effective treatment strategies, this concept has become more useful. Similar symptomatology suggests possibly similar treatment approaches, which is particularly helpful for disorders that have traditionally been a challenge to treat, such as body dysmorphic disorder (BDD) and hypochondriasis (HC).

The current chapter will explore the similarities between obsessive-compulsive disorder (OCD) and BDD and HC in an attempt to conceptualize them within the obsessive-compulsive spectrum disorders.

BODY DYSMORPHIC DISORDER

Symptoms

Body dysmorphic disorder, previously referred to as "dysmorphophobia," is defined as an exaggerated preoccupation with an imagined or slight defect in physical appearance causing marked distress and interference with daily functioning (American Psychiatric Association, 2000). Although the word "preoccupation" is used to define BDD, one can easily substitute the word "obsession" in place of "preoccupation." In BDD, there is an obsession with either one's general physical appearance or with a specific body part. Although any body part can be involved, the most commonly observed typically include the head area, such as skin, hair, and nose (Neziroglu & Yaryura-Tobias, 1993a; Phillips, 1996a; Phillips, McElroy, Keck, Pope, & Hudson, 1993). Body dysmorphic disorder involving the muscularity of the entire body is termed "muscle dysmorphia." (Phillips, O'Sullivan, & Pope, 1997; Pope, Gruber, Choi, Olivardia, & Phillips, 1997).

Individuals with BDD spend a significant amount of time thinking about their appearance. These thoughts interfere with daily functioning, cause distress. People

with BDD often review others' reactions/observations of their appearance and are obsessed with the image of their current appearance and the way they would like it to be. Their mood state is generally depressed, but they also exhibit anxiety over exposing their defect in public. Ideas of reference are common as well, with individuals believing that others are taking special note of their defect. They may also misinterpret ambiguous events in the environment (eg, someone laughing) as directly referring to them (eg, "the laughter must be because of the way I look"). The anxiety elicited by external stimuli and the desire to avoid them is similar in both BDD and OCD. The misinterpretation of events is also similar, although in BDD it is to others' reaction to their appearance and in OCD it is the presence of danger where there is none.

Repetitive behaviors that significantly interfere with daily living and resemble the ritualistic behaviors found in OCD are also observed in BDD. One of the most common behaviors is body checking using mirrors or reflective surfaces (Phillips, 1996a; Veale & Riley, 2001). Mirror checking is more common than avoidance of mirrors and can include brief but numerous episodes as well as prolonged staring. It is common for individuals with BDD to seek methods to alter their appearance, either through camouflaging or improving the body part of concern through excessive grooming and the use of beauty products. Patients may also consider more permanent changes through cosmetic or dermatological procedures (Sarwer, Wadden, Pertschuk, & Whitaker, 1998), and in some cases, even attempt to implement "do it yourself" surgery (Veale, 2000). Affected individuals may spend hours researching and reading about the way that certain body parts "should" appear, and/or possible ways to alter their appearance. Seeking reassurance about appearance is also a commonly seen symptom. Many individuals with BDD avoid social or performance situations (McElroy, Phillips et al., 1993), which can lead to impairment in various areas of functioning.

Similarities in Symptoms: BDD and OCD

Similar to obsessions in OCD, the thoughts about appearance in BDD are repetitive, intrusive, difficult to control or resist, and quite distressing (Brady, Austin, & Lydiard, 1990; Hollander, Cohen, & Simeon, 1993; Neziroglu & Yaryura-Tobias, 1993a, 1993b; Rosen, 1995). Magical thinking, a symptom found in OCD, can also be present in BDD. For example, the belief that the imagined defect has changed and/or disappeared (Neziroglu, Anderson, & Yaryura-Tobias, 1999), sometimes even within minutes of having checked the mirror (leading to subsequent mirror checking).

Although there is debate that the difference between obsessions in OCD and beliefs in BDD is one of ego-syntonicity versus dystonicity, not all patients with BDD describe beliefs with delusional intensity, nor do all OCD patients demonstrate good insight. Perhaps the question of ego-syntonicity is one of *degree of insight*, with the various spectrum disorders existing on a continuum. There may also be fluctuation of insight during the course of any one of the disorders, with individuals fluctuating between obsessional, overvalued ideation, and delusionality (McKay, Neziroglu, & Yaryura-Tobias, 1997). Thus, BDD thought content may resemble OCD obsessions, but differ in the level of insight the person has into its senselessness.

The behaviors observed in BDD are similar to OCD compulsions in terms of frequency, duration, intensity and degree of reported control (Hollander et al., 1993).

Individuals with BDD, similar to those with OCD, report that they are driven to engage in repetitive behaviors. Further, functional analysis reveals similarities between repetitive behaviors in both conditions. For example, mirror checking is performed to reduce anxiety levels and provide reassurance, similar to compulsive checking (eg, of door locks) in OCD.

The degree of avoidance based on fear and apprehension is similar in nature in OCD and BDD, and is based on a negative reinforcement paradigm in both disorders. For example, hand washing in OCD may reduce obsessional fears of AIDS contamination; likewise in BDD, wearing a hat may reduce fears that others will notice and/or negatively evaluate a "defective" hairline. Thus, in both conditions, avoidance occurs in situations where *perceived* threat is high. Reassurance seeking also occurs in both conditions. Like OCD, individuals with BDD may repeatedly ask questions of others to gain reassurance regarding their appearance (eg, "do you think my hair looks OK?")

Only two empirical studies have directly compared the symptoms of OCD and BDD. One investigation found that the two disorders were similar in terms of the severity of obsessions, compulsions, depression, and trait anxiety; yet that BDD patients had more overvalued ideation and lower levels of physiological reactivity (McKay et al., 1997). These results suggest that the two disorders are related; with BDD perhaps a more severe variant. The second study reported similar findings: BDD and OCD patients had similar levels of obsessive and compulsive symptom severity and overall functional impairment (Saxena et al., 2001).

Epidemiology

Similar to OCD, BDD is shown to affect both genders equally (Perugi et al., 1997; Phillips & Diaz, 1997), although we find a predominance of young men in our own practice. Onset of BDD is most frequently in the adolescent years (McElroy et al., 1993; Neziroglu et al., 1999; Phillips, McElroy, Keck, Pope, & Hudson, 1993; Veale et al., 1996a), probably because of the heightened exposure to social activities which occurs at this age. Similarly, OCD onset is typically before age 25 in 65% of cases; and in women seems to have a bi-modal distribution with onset occurring either during adolescence or during pregnancy or its immediate aftermath (Neziroglu, Anemone, & Yaryura-Tobias, 1992). Body dysmorphic disorder is reported to occur in up to 2% of the general population (Rich, Rosen, Orosan, & Reiter, 1992) and OCD in about 2.5% (Karno, Goldin, & Soreman, 1988; Reiger, Boyd, & Burke, 1988). It should be noted that the lower prevalence rates in BDD may be an artifact of the tendency to underreport body image concerns (Hollander, Cohen, & Simeon, 1993).

Associated Features

Overvalued ideation, as it is currently defined, refers to how strongly one holds a particular belief along a continuum from rational thought to delusional (Kozak & Foa, 1994). Although it has traditionally been thought that individuals with OCD recognize the senselessness of their obsessions, research suggests a continuum of insight with some patients demonstrating poor insight and delusionality (Eisen & Rasmussen, 1993; Insel & Akiskal, 1986). Although compared to OCD, BDD seems to be associated

with a higher degree of OVI, both disorders can be viewed on a continuum of insight ranging from good to delusional intensity (Hollander et al., 1993; McKay et al., 1997). This suggests that BDD is a more severe variant of OCD.

Comorbidity rates are similar in both disorders, with other anxiety disorders and depression as the most commonly observed conditions (Hollander et al., 1993; Phillips et al., 1993). In addition, there are high rates of comorbidity between OCD and BDD (Simeon, Hollander, Stein, Cohen, & Aronowitz, 1995), ranging from 16% (Bienvenu et al., 2000) to 37% of OCD patients meeting criteria for BDD (Hollander et al., 1993). Studies have also found that the lifetime incidence of OCD in a BDD population ranges from 30% (Gustad & Phillips, 2003) to 37% (Phillips et al., 1993). Interestingly, one recent comorbidity study found that depression tended to onset *following* the onset of BDD, possibly suggesting that depression is a complication of BDD (Gustad & Phillips, 2003). However, we find a substantial amount of comorbid depression in BDD that appears unrelated to the BDD, whereas in OCD the depression is usually secondary. Family history studies suggest that first-degree relatives of patients with BDD have high rates of OCD (Phillips et al., 1993).

The high comorbidity between depression and BDD, as well as the high levels of suicidal ideation characteristic of BDD, has led to the hypothesis that BDD may be better conceptualized as an "Affective Spectrum Disorder" rather than an obsessive-compulsive spectrum disorder (Phillips, McElroy, Hudson, & Pope, 1995). Findings from a morphometric magnetic resonance imaging study indicated that BDD patients displayed a leftward shift in caudate nucleus asymmetry as well as increased total white matter volume, which is consistent with findings for OCD (Rauch et al., 2003). Individuals with major depression, on the other hand, demonstrate reduced hippocampal volumes, rather than the striatum (Bremner et al., 2000).

Findings from one neuropsychological study comparing BDD patients to a sample of OCD patients, schizophrenia patients, and healthy individuals demonstrated that BDD and OCD display similar deficits in executive functioning dependent on the prefrontal cortex (Hanes, 1998). This is consistent with previous findings of executive functioning deficits in OCD (Abbruzzesse, Ferri, & Scarone, 1997).

Treatment Response

Although treatment research is limited, the psychological treatments effective for OCD also show promise for BDD. Behavioral treatment involving exposure and response prevention (Braddock, 1982; Campisi, 1995; Marks & Mishan, 1988), cognitive therapy (Geremia & Neziroglu, 2001), as well as the combination (Neziroglu & Khemlani-Patel, 2002, 2003; Neziroglu & Yaryura-Tobias, 1993b; Veale et al., 1996b) result in symptom improvement. Pharmacological studies have demonstrated consistent results with the use of selective serotonin reuptake inhibitors (SSRIs), which are also helpful for OCD. Response to SSRIs seems to be positive even in cases of BDD with very poor insight (Phillips, McElroy, Dwightman, Eisen, & Rasmussen, 2001b; Phillips, McElroy, Keck, Hudson, & Pope, 1994). Augmentation strategies are often required for BDD, and success with buspirone and neuroleptics has been reported (Phillips, Albertini, Siniscalchi, Khan, & Robinson, 2001a).

Response to SSRIs alone does not necessarily imply a relationship between OCD and BDD since these medications are used to treat a variety of psychiatric conditions

and the exact mechanism by which they bring about symptom reduction remains unknown. However, exposure and response prevention is a specific set of treatment procedures designed to weaken anxiety and fear that is maintained by negative reinforcement. So, the fact that exposure therapy is useful with BDD does suggest a similar phenomenology to that of OCD. Thus, in light of similar symptomatology, familial history, and comorbidity data, the comparable treatment response profiles provide further evidence for the inclusion of BDD within the OC spectrum.

HYPOCHONDRIASIS

Symptoms

The essential feature of HC is a fear of having, or a conviction that one has, a serious disease based on misinterpretation of bodily symptoms (American Psychiatric Association, 2000). The belief persists despite receiving adequate medical information and reassurance to the contrary, which is one of the most distinct characteristics of the disorder (Fava & Mangelli, 2001). Symptoms must be present for 6 months in order to qualify for the diagnosis, since transient HC can occur within other psychiatric diagnoses, under conditions of stress, or during changes in physical health or life events (Barsky, Wyshak, & Klerman, 1990a; Robbins & Kirmayer, 1996).

There has been much debate regarding how best to conceptualize HC (Noyes, 2001); and it has been categorized in various ways, including as a personality trait (Tyrer, Fowler-Dixon, Ferguson, & Kelemen, 1990), a primary Axis I disorder (Barsky & Klerman, 1983; Costa & McCrae, 1985), and a symptom of anxiety and mood disorders. There is also a lack of clear distinction within the literature in the differentiation between the concepts of "disease phobia" and "disease conviction," with the former often defined as the fear of developing an illness, and the latter as a belief that one has a particular illness. In both cases, there is a preoccupation with illness and avoidance behavior, however "disease phobia" may be better conceptualized as somatic obsessions in OCD whereas "disease conviction" is specific to HC.

Individuals with HC present with an impending sense that their wellness is being threatened, excessive concern with health, a desire to remain healthy, and a preoccupation with morbidity (Starcevic, 2001). In addition, patients possess false beliefs about physical symptoms and disease, increased attention to bodily sensations, and distrust of physicians' opinions (Warwick & Salkovskis, 1990).

Salkovskis and Warwick (2001) proposed a cognitive-behavioral model of HC in which dysfunctional beliefs about health and illnesses are maintained by four main processes: (1) information processing bias, (2) hypervigilance to physiological reactions, (3) safety-seeking behaviors, and (4) changes in affect. Once an individual actively believes that they are suffering from a particular disease, evidence that supports that belief is gathered. Information processing bias refers to the selective attention to information given by medical professionals, as well as an increased attention to bodily sensations such as slight variations in any physical experiences. Similarly to OCD, individuals with HC seek complete certainty that they are healthy. Intense anxiety can invariably lead to increased physiological arousal, which inadvertently provides more evidence for the belief that one is suffering from an illness. Misinterpretation of

these symptoms leads to increased anxiety, which evokes further physical sensations (ie, sympathetic arousal) and the development of a vicious cycle. Safety-seeking behaviors are intentional responses to perceived danger, which are performed in order to decrease the (erroneously high) perceived likelihood of threat. Typically, individuals with HC engage in repetitious health checking behaviors such as seeking excessive medical consultation and examination, closely monitoring bodily processes (eg, heart rate), manipulating and inspecting the body, and reading medical textbooks or information from the Internet. Individuals may also avoid situations believed to exacerbate the illness, such as physical exercise or sexual contact. Perhaps the most prominent symptom in HC is reassurance seeking behavior through repeated medical consultations and repetitive questioning towards family and friends. Lastly, disturbance in thought content leads to increased disturbance in mood.

Similarities in OCD and HC Symptoms

Symptomatology in HC shares many similarities with OCD, suggesting its inclusion within the OC spectrum. Researchers have proposed that the preoccupation with illness in HC is similar to obsessional thinking in that both are difficult to resist, intrusive, and lead to increased anxiety (Warwick & Salkovskis, 1990). Similar to OCD and other anxiety disorders, the cognitive distortions in HC include a misinterpretation of ambiguous stimuli or situations as more threatening than they really are (Salkovskis & Warwick, 2001). This tendency to overestimate the degree of threat is central to the experience and maintenance of anxiety disorders (Beck, Emery, & Greenberg, 1985). Among the cognitive processes identified in OCD (Obsessive-Compulsive Cognitions Working Group, 1997, 2001) intolerance for uncertainty, inflated sense of responsibility, and over-appraisal of threat are all present in HC (Salkovskis & Warwick, 2001; Starcevic, 2001) along with the desire to attain control over the body (Starcevic, 2001). In HC and in OCD, beliefs concerning responsibility for harm appear to motivate checking and reassurance behaviors.

Similar to the dysfunctional thinking patterns present in OCD, patients with HC seem highly concerned with the *meaning* of symptoms and with seeking an explanation for these symptoms rather than with seeking obtaining appropriate treatment (Barsky & Klerman, 1983; Kellner, 1987). Paradoxically, patients with HC do not engage in healthier behaviors compared to a group of family practice patients; they do not smoke less and take other health precautions such as avoiding unhealthy foods (Kellner, 1987). Researchers have also noted that HC individuals can suffer from distressing thoughts and images of death (Starcevic, 2001), similar to patients with OCD with morbid concerns. Similarly, OCD can include concerns about health and fears of illness, injury or contamination (Fallon, Javitch, Hollander, & Liebowitz, 1991). Rasmussen and Eisen (1992) indicate that such somatic obsessions may be are indistinguishable from HC, except that OCD includes other unrelated obsessions and compulsions (eg, blasphemous intrusive thoughts and praying rituals), whereas patients with HC are singly obsessed with health issues.

As noted above, HC involves many safety-seeking behaviors. Similar to compulsions, these behaviors have a driven and irresistible quality, are excessive, and are intended to relieve anxiety (Noyes, 2001). In addition, these behaviors appear identical to compulsive rituals in OCD in their intensity, duration, and frequency. Their

purpose is to avoid or escape a feared consequence, similar to people suffering from anxiety disorders (Salkovskis, 1991, 1996a, 1996b). People with HC attempt to reduce health anxiety by seeking reassurance, but similar to OCD compulsions, the anxiety increases again until further reassurance is required (Warwick & Salkovskis, 1990). Repetitive behaviors in both conditions also share the qualities of being limited in range and repertoire (Barsky, 1992a, 1992b).

Despite hypothesized similarities between OCD and HC, there is little empirical research directly comparing the disorders on symptomatology, course, prognostic variables, or treatment response. One recent study compared a group of outpatients with HC alone, OCD alone, and the combination of HC and OCD (Neziroglu, McKay, & Yaryura-Tobias, 2000). The groups did not differ in their severity of depressive symptoms, state and trait anxiety, or physiological symptoms of anxiety. Furthermore, there were no significant differences in the severity of obsessions. However, HC patients demonstrated a higher degree of overvalued ideation than did OCD patients, and the OCD group demonstrated higher levels of compulsivity; although the latter finding may have been due to the assessment tool rather than true differences between the disorders. These findings suggest that the HC, similar to BDD, may be a part of the OC spectrum, with high OVI as a variable that distinguishes disorders within the spectrum (Neziroglu et al., 2000).

Epidemiology and Prevalence

The onset of HC typically occurs in early adulthood. As noted above, HC symptoms can occur transiently in normal populations (Kellner, 1987), although typically when a person is under stress, seriously ill or recovering from a serious illness, or following the loss of a family member (Barsky & Klerman, 1983). HC symptoms have also been observed among medical students; in 70–79% in one study (Hunter, Lohrenz, & Schwartzman, 1964).

Whereas most studies have found an equal prevalence of HC across genders (Barsky & Wyshak, 1989), some report a higher prevalence among women (Barsky et al., 1990a), although this finding may be due to increased treatment-seeking among women in general. The prevalence of HC in general medical outpatient clinics is estimated to be between 3% and 6.5% (Barsky, Wyshak & Klerman, 1990a; Escobar, Rubio-Stipec, Canino, & Karno, 1998). On the basis of the National Institute for Mental Health Epidemiological Catchment Area (ECA) studies, the prevalence of HC in the general population is estimated to be between 9% and 20% (Escobar, Rubio-Stipec, Canino, & Karno, 1989).

Associated Features

Similar to BDD, patients with HC display high levels of OVI, making them reluctant to admit that their problem is due to psychological causes rather than physical (Neziroglu et al., 2000; Pilowsky, 1970; Sims, 1988; Warwick & Salkovskis, 1989). This results in repeatedly visiting doctors and undergoing medical procedures and tests even when the findings are negative (Barsky & Klerman, 1983). As in BDD and other proposed OC spectrum disorders, HC is characterized by a continuum of insight. Yet,

patients with HC seem to display a higher degree of OVI than do those with other disorders, and this difference has been cited as one crucial distinction between OCD and HC.

Research indicates a high comorbidity between HC and the anxiety and mood disorders. The lifetime prevalence of any anxiety disorder in HC patients was 86% in a sample of 42 participants (Barsky, Wyshak, & Klerman, 1992). Noyes et al. (1994) reported that 22% of 50 HC patients had comorbid anxiety disorders. Lifetime prevalence of HC among OCD patients ranges from 13% (Jaisoorya, Janardhan Reddy, & Srinath, 2003) to 15% (Bienvenu et al., 2000).

Treatment Response

Despite the attention it has received for over 2000 years, there are very few treatment studies of HC. Treatment has consisted of a variety of methods including supportive therapy, explanatory therapy, psychodynamic approaches, cognitive and behavioral treatment, and psychopharmacology. Hollander (1993) has proposed that HC is part of the OCD spectrum based on symptom profile and selective response to CBT and pharmacotherapy.

Case reports and open label studies suggest that SSRI medication (at higher dosages typically recommended for OCD) is associated with positive outcome in HC (Fallon et al., 1991, 1993; Viswanathan & Paradis, 1991). Hypochondriacal patients, however, tend to be resistant to taking psychotropic medications due to increased fears and hypervigilance to the side effects. Their resistance is also based on the belief that they have a medical, not a psychiatric, condition.

Positive response has also been shown with exposure and response prevention (Logsdail, Lovell, Warwick, & Marks, 1991; Warwick & Marks, 1988) and a combination of cognitive and behavioral therapy techniques (Salkovskis, Warwick, & Deale, 2003; Visser & Bouman, 1992), including a wait list controlled trial of cognitive-behavioral treatment (Warwick, Clark, Cobb, & Salkovskis, 1996). One pilot study comparing the efficacy of cognitive versus behavioral (exposure) strategies found no differential response for either treatment method (Bouman & Visser, 1998): both modalities resulted in improvement in illness attitude and depression after 12 sessions.

Variables identified as predictors of poor treatment outcome include a higher degree of HC symptoms, higher degree of cognitive distortions related to bodily functioning, greater psychosocial impairment, more somatization symptoms, greater degree of general psychopathology, and more utilization of health care services (Hiller, Leibbrand, Rief, & Fichter, 2002). In summary, studies demonstrating similar symptomatology and treatment response suggest that, as with BDD, HC can be classified as an OC spectrum disorder. However, further research comparing the OC spectrum disorders may shed further light on the conceptualization of this construct.

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Chapter 9

CONTRASTING NONPARAPHILIC SEXUAL ADDICTIONS AND OCD

Stefanie A. Schwartz and Jonathan S. Abramowitz

One group of behaviors typically included in the proposed obsessive-compulsive (OC) spectrum is the "nonparaphilic sexual addictions" (NPSAs; Goldsmith, Shapira, Phillips, & McElroy, 1998; Hollander & Wong, 1995), which are sometimes referred to as sexual compulsions. Nonparaphilic sexual addictions are defined as "repetitive sexual acts involving conventional, normative, or nondeviant sexual thoughts or behaviors that the person feels compelled or driven to perform, which may or may not cause distress (Goldsmith et al., 1998)." Nonparaphilic sexual addictions are not formally described in the *DSM*; however, a diagnosis of impulse-control disorder not otherwise specified (NOS) may be given if a person with this behavioral pattern experiences an interference in functioning (eg, relationships, work, etc) for at least six months.

Examples of NPSAs include the excessive patronage of exotic dance bars and frequent use of pornography at the expense of one's romantic relationship. One patient we evaluated could not resist the urge to meet and engage in sex with prostitutes despite the fact that this regular activity was resulting in difficulty maintaining his attendance and grades in graduate school. If one focuses solely on the excessiveness and repetitiveness of sexual urges and behaviors among such patients, there appears to be little that distinguishes NPSAs from the repetitive thinking and behavior that occur among individuals with OCD, especially given that both problems may involve sexual themes. In our experience, we have observed that individuals with NPSAs are often given the diagnosis of OCD based on the repetitive and senseless qualities of the sexual thoughts and behaviors that are conceptualized as "obsessive" and "compulsive" (for a review see Goldsmith et al., 1998).

Despite claims of overlapping features between NPSAs and OCD, no research has been conducted to compare these two conditions. One reason for the dearth of research on NPSAs is that most sufferers would just as well not disclose their behavior due to potential embarrassment. As a result, NPSAs remain a somewhat unidentified problem (Goldsmith et al., 1998). Clinical observations, however, suggest that the repetitive sexual thoughts in NPSAs typically concern erotic themes and bear similarities to sexual fantasies. In contrast, repetitive sexual thoughts in OCD are usually experienced as highly unacceptable, aversive, and met with intense resistance. Gordon (2002) has differentiated "compulsive" activity in NPSAs from that

in OCD. He observed that although the compulsive behaviors in NPSAs often involve performing the very sexual acts contemplated in the erotic repetitive thoughts, compulsive rituals in OCD represent attempts to neutralize sexual obsessional thoughts (eg, via praying or mental neutralizing).

The observations discussed above suggest that OCD and NPSAs both involve repetitive thinking and behavior, but that the content of the thoughts and behavior may differ in many important ways. To further highlight these similarities and differences, we present two clinical case examples.

OCD Patient with Sexual Obsessions

S.D. was a 20-year-old unmarried, female college student who described an exclusively heterosexual dating history. However, she reported recurrent, upsetting, intrusive thoughts that she might be (or is becoming) a lesbian. These fears were triggered by seeing attractive females in person, in print, or on television. She was avoiding watching certain television shows or looking at certain fashion magazines or catalogs for fear of having lesbian thoughts, as she was concerned that such thoughts indicated sexual interest in females. Her compulsive behaviors included seeking reassurance by mentally assessing her own attraction to men (eg, repeating the phrase "I think he's cute" when she saw an attractive male), reviewing her dating/sexual history, and sometimes asking for reassurance from parents or friends that she is not a homosexual.

NPSA Patient

D.G. was a 30-year-old divorced male who was employed as a salesman. He reported frequent overwhelming thoughts and urges to visit exotic dance bars during the workday. He was unable to focus on his work as he was constantly thinking about his next visit to the bar and how he would pay for sexual favors from the various exotic dancers. He visited exotic dance bars on an almost daily basis and spent in excess of $500.00 per visit on lap dances and tips. His recent divorce was the result of his wife's intolerance of this behavior, which also included arriving home very late each day. Although D.G. acknowledged feeling guilty for his excessive behavior, he continued to frequent dance bars and pay for sexual favors.

REPORT OF A STUDY

In light of the observations discussed above, and the lack of empirical investigation, we designed a study to more systematically compare the phenomenological characteristics of sexual thoughts and behaviors in individuals with NPSAs to those present in OCD (Schwartz & Abramowitz, 2003). Our aim was to clarify the extent to which NPSAs might be related to OCD, and thus might be considered part of the OC spectrum. Given that both conditions involve repetitive thoughts and behavior, the focus of the study was on *functional* characteristics of these thoughts and behaviors. That is, we sought to compare and contrast the antecedents and consequences of sexual thoughts and behaviors in OCD and NPSAs. If repetitive thinking and behavior serves the same purpose in OCD and in NPSAs, there is good reason to suspect that these two conditions are related to one another.

Participants in our study included 12 patients referred to our OCD/Anxiety Disorders Clinic with the primary complaint of "sexual obsessions." Referral sources included physicians (including psychiatrists) and advertisements for research on OCD. Each participant was given a structured diagnostic interview as well as the Yale–Brown obsessive-compulsive scale (Y–BOCS; Goodman et al., 1989a, 1989b). The Y–BOCS first includes a fairly comprehensive listing of 40 obsessions and 29 compulsions, each categorized broadly by content (eg, contamination obsessions, washing compulsions, etc). Patients are asked whether or not they experience each of the specific obsessions and compulsions. Questions about sexual obsessions are included in the checklist. Next, the Y–BOCS severity scale is administered; this is a 10-item interview in which obsessions and compulsions are rated on their time, interference, distress, resistance, and control. Each severity scale item is rated from 0 (no symptoms) to 4 (severe) to produce a total score ranging from 0 to 40.

Based on the above diagnostic interviews, six patients (3 males and 3 females) were found to meet *DSM-IV* criteria (APA, 1994, Diagnostic and statistical manual of mental disorders) for OCD, whereas the other six (all males) had NPSAs and met *DSM-IV* criteria for impulse-control disorder NOS (as described above), but not OCD.

Each of the 12 participants was next given a semistructured interview designed specifically for the purposes of this study. This instrument consisted of five questions addressing phenomenological aspects of sexual thoughts and related behaviors. Specifically, the patients were asked (*a*) how much anxiety or distress their sexual thoughts caused them, (*b*) the extent of avoidance associated with the thoughts, (*c*) the strength of the urge to perform the compulsive behavior, (*d*) the level of sexual arousal, and (*e*) the amount of sexual gratification from performing the compulsive behavior. Interviewers rated the participant's responses on a scale from 0 (none or never) to 8 (extremely or always).

Participants also completed both the state and trait versions of the state–trait anxiety inventory (STAI; Spielberger, Gorsuch, Lushene, Vaag, & Jacobs, 1983). The first 20 items of the STAI assess trait anxiety, or how the participant feels "generally." The second 20 items assess state anxiety, or how the participant feels "right now." Before completing the state version, the participants were asked to evoke their most vivid sexual obsessional thought. Depression was assessed using the Beck depression inventory (BDI; Beck, Ward, Mendelsohn, Mock, & Erlbaugh, 1961), a 21-item self-report scale that assesses the severity of affective, cognitive, motivational, vegetative, and psychomotor components of depression.

As can be seen in Figures 9.1 and 9.2, sexual obsessions among OCD participants were associated with much stronger fear and avoidance responses than for participants with NPSAs. In fact, when such thoughts were evoked, individuals with NPSAs evidenced very little associated fear and instead reported feeling sexually aroused. Sexual arousal was not a consequence of such thoughts among participants with OCD. This suggests that while sexual obsessions occurring in the context of OCD are experienced as unwanted, aversive, and threatening, those occurring in NPSAs are experienced as sexually arousing. Between-group differences were significant at the $p < 0.01$ level. Differences were also observed in the function of repetitive behaviors in each group. Although both diagnostic groups were equally driven to perform repetitive behaviors, NPSAs experienced significantly more sexual pleasure when performing their compulsive behavior than did OCD patients, who experienced very little sexual pleasure. As is shown in Figure 9.2, patients with OCD also reported higher levels of

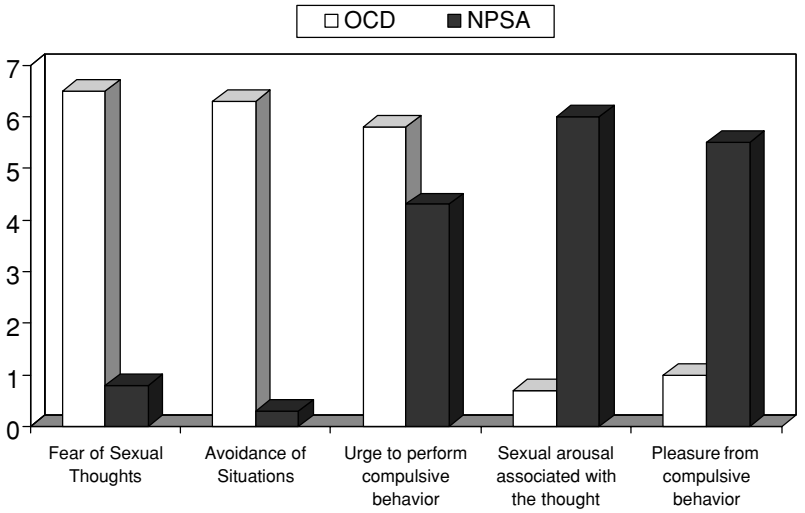

FIGURE 9.1. Mean scores for patients with OCD and NPSAs on measures of fear, avoidance, and compulsive behaviors.

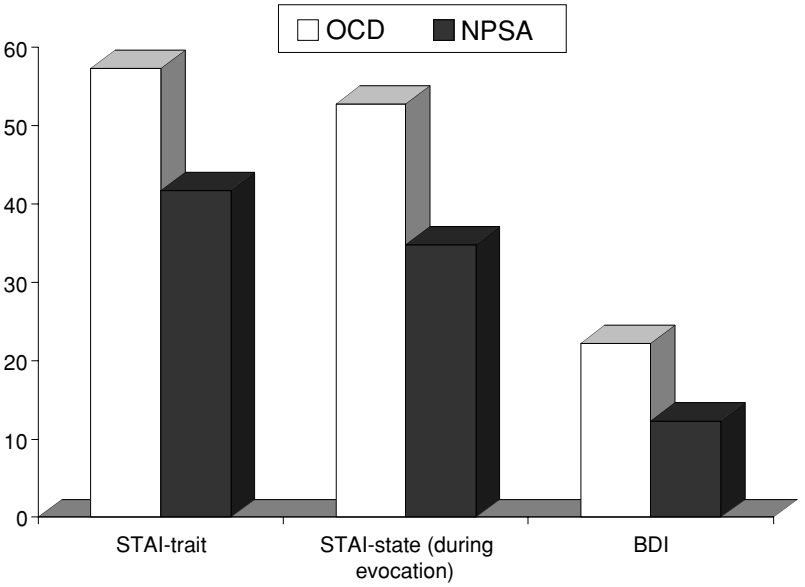

FIGURE 9.2. Mean scores for patients with OCD and NPSAs on measures of depressive and anxiety symptoms.

trait anxiety and depression compared to those with NPSAs, who scored within the subclinical range of both the BDI and STAI-trait. Taken together, the findings from this small study suggest, at least preliminarily, that sexual thoughts and compulsive behaviors in NPSAs involve remarkably different functional characteristics and clinical presentations than those observed in individuals with OCD.

Three specific qualitative distinctions between OCD and NPSA patients were observed that shed further light on our findings. First, clinical interviews revealed

that in cases of NPSAs, sexual thoughts and compulsive behaviors were of greater concern to those other than the patient (ie, the patient's spouse); yet in OCD, these thoughts and behaviors were of greatest concern to the patient himself/herself. This supports our finding that individuals with NPSAs do not experience the related depressive and anxious symptoms to the extent that those with OCD do. Second, we noted that in addition to sexual symptoms, five of the six OCD patients in our study reported other unrelated types of obsessions and compulsions (eg, contamination, religious, aggressive, miscellaneous) on the Y–BOCS checklist. In contrast, for all six NPSA participants, excessive sexual thoughts or behaviors were the lone symptom.

A third phenomenological difference between NPSAs and OCD concerned the link between repetitive thoughts and compulsive behaviors. Consistent with the diagnosis of impulse control disorder, individuals with NPSAs reported that they deliberately acted upon urges to engage in the sexual activities that they repetitively thought about in order to attain sexual gratification. In contrast, OCD participants in our study reported beliefs such as "thinking the sexual thoughts makes me immoral," or "if I think about rape it means I am a rapist." Moreover, compulsive behaviors among OCD participants included a range of strategies (both overt and covert) aimed at resisting or neutralizing this fear; and in no instance did OCD patients engage in the actual sexual behavior described in their obsessional thoughts. Thus, the phenomenological link between thought and behavior in OCD appears qualitatively distinct from that in NPSAs, further suggesting that these are unrelated conditions.

Our findings speak about conceptual approaches to understanding possible relationships between OCD and other disorders. Such overlaps may occur at various levels. On a superficial level, OCD and other conditions may be perceived as related based on the shared presence of repetitious thinking or behavior. This is the basis for including NPSAs among the OC spectrum disorders. Indeed OCD and NPSAs both involve repetitive thinking and behavior. However, as we have demonstrated, the repetitive phenomena in NPSAs have qualitatively distinct phenomenological properties from those present in OCD.

Thus, a more meaningful approach to understanding relationships between OCD and other disorders is to look for parallels at the level of phenomenological mechanisms involved in the maintenance of the problem. In the case of sexual obsessions in OCD, the compulsive behavior is linked to misinterpretations of unwanted intrusive sexual thoughts as threatening. The individual worries that their sexual idea, image, or impulse is in some sense significant, and that action must be taken to reduce the probability of some dreaded outcome. Thus, the individual "neutralizes" their thoughts (and thus their subjective distress) via some other thought or action (compulsive ritual). As we have discussed above, this cognition-behavior link is not present in NPSAs.

CLINICAL IMPLICATIONS

The most important reason for conceptualizing OCD as separate from NPSAs is that this distinction has implications for the choice of treatment procedures. This view is in contrast to that held by some advocates of the OC spectrum approach who have argued that the spectrum disorders overlap in their response to

"antiobsessional behavioral therapies" (Hollander & Wong, 1995, p. 3). However, this argument oversimplifies behavior therapy and fails to consider the wide range of behavioral procedures for a wide array of problems. As we discuss further below, the specific behavioral procedures that are effective in reducing OCD and NPSA symptoms are quite distinct in their form and function.

Effective use of behavioral (and cognitive) therapy techniques relies on precise information about the phenomenological and functional aspects of the undesirable thoughts and behaviors to be eliminated. Assessment of antecedents and consequences of symptoms (functional [behavioral] analysis; Wolpe, 1958, 1969) is critical in making logical choices of specific therapeutic procedures. From a behavioral perspective, obsessional fear is an excessive response to nondangerous stimuli, and compulsive urges function as an escape from obsessional distress. Thus, treatment of OCD must involve (*a*) exposure to obsessional cues to weaken the excessive anxiety, and (*b*) blocking of rituals (response prevention) to reduce the association between compulsive behaviors and the reduction of fear. Hence, behavioral therapy for OCD is not "antiobsessional." Rather its purpose is to weaken the association between otherwise innocuous intrusive thoughts and pathological anxiety.

While exposure and response prevention procedures are often effective in reducing OCD symptoms, functional analyses suggest that NPSAs (and many other disorders included in the OC spectrum) could not be appropriately treated using these procedures. This is because NPSAs involve neither fear-evoking obsessional thoughts nor excessive rituals designed to escape or neutralize anxiety. Functional assessment of NPSAs reveals appealing sexual thoughts and tendencies to engage excessively in sexual behaviors experienced as pleasurable. This conceptualization leads to the use of treatment procedures known as stimulus control (ie, habit reversal), in which the patient reduces access to stimuli involved in the sexual behavior and instead practices more appropriate responses to urges to engage in excessive sexual behavior (eg, distraction, appropriate sexual behavior, etc).

Rachman (1998) and Salkovskis (1999) have proposed a cognitive model of repugnant obsessions that further distinguishes OCD from NPSAs. This conceptualization begins with the understanding that most people normally experience intrusive undesirable thoughts, including those of a sexual nature. However, when individuals misconstrue these benign thoughts as having unacceptable consequences (ie, my 'perverted' thoughts mean I'm becoming a pervert), the result is increased preoccupation with the thought, difficulty controlling the thought, increased thought frequency, and negative mood. This, in turn, leads to efforts to neutralize the thought (via compulsive rituals), analyze its meaning, or try to keep them from entering consciousness (via avoidance) to forestall feared consequences. Inevitable failures to control such thoughts are further misperceived as evidence for the significance of the thoughts and the need for further restraint. Inevitable doubts about the meaning of such intrusions leads to uncertainty and fosters further preoccupation. Thus, whereas preoccupation with sexual thoughts may be prevalent in both OCD and NPSAs, the increased frequency of such thoughts in OCD patients is related to attempts to neutralize or control these thoughts. In contrast, preoccupation in NPSAs is more deliberate and occurs because the thoughts are experienced as sexually arousing.

From a cognitive standpoint, therapy for unwanted sexual obsessions in OCD must help patients to view their sexual thoughts as normal and not a sign of moral demise or untamed perverted impulses. Treatment therefore includes challenging

faulty beliefs about thoughts using educational procedures, cognitive restructuring, and behavioral experiments (eg, Beck, 1976). For example, a patient with OCD feared he would act upon obsessional thoughts of violently raping his wife while she slept. To ensure against impulsively raping, he had insisted his wife sleep in a different room with the door locked. Cognitive therapy involved developing an idiosyncratic model of the factors that maintain the obsessional problem (ie, faulty appraisals of harmless thoughts, avoidance), and identifying evidence that he was misinterpreting the thought's presence and meaning. At one point in treatment, the following behavioral experiment was undertaken: the patient was asked to remain in the room with his sleeping wife and deliberately think about raping her. The fact that no rape ever occurred was discussed as evidence that the patient would not impulsively act on these sexual thoughts; indeed he agreed that were he a ruthless rapist, he would not have let such an opportunity get away. Clearly, such treatment procedures would not be indicated for the treatment of NPSA patients since their problem is that they do act on their sexual thoughts.

CONCLUSIONS

To provide a more rigorous criterion for inclusion of disorders in an OCD spectrum that is sensitive to differences in treatment approaches described above, we recommend the use of empirically driven functional analytic studies. Such studies have been conducted previously in the cases of hypochondriasis (eg, Neziroglu, McKay, & Yaryura-Tobias, 2000) and body dysmorphic disorder (eg, McKay, Neziroglu, & Yaryura-Tobias, 1997), which seem to be functionally similar to OCD. Conversely, such analyses have indicated that Tourette's syndrome (Miguel et al., 1995) and trichotillomania (Tukel, Keser, Karali, Olgun, & Caliksu, 2001) are phenomenologically distinguishable from OCD. An advantage of this kind of empirical scrutiny is that it may result in homogeneous groups of disorders for which effective treatments can be more readily developed and applied.

By focusing on the repetitiousness of symptoms, one may miss the fundamental feature of OCD: the relationship between obsessions and compulsions. This relationship is unique to OCD. OCD is an anxiety disorder in which unwanted, upsetting, irrational thoughts (obsessions) give rise to intense distress and urges to reduce this distress through overt or covert compulsive behaviors. Compulsive rituals in OCD therefore function as an escape from distress. Since this pattern is not present in NPSAs, it is difficult to argue that NPSAs represent a form of OCD.

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Chapter 10

COMPULSIVE BUYING: A DISORDER OF COMPULSIVITY OR IMPULSIVITY?

Lorraine A. Swan-Kremeier, James E. Mitchell, and Ronald J. Faber

Although compulsive buying has only become the focus of clinical attention and research investigation in the last few decades, its presence has been noted for almost a century. Emil Kraepelin (1915) was first to define the problem of compulsive buying in 1915 in his description of "oniomania" or "buying mania." Kraepelin's original description was later expanded by Bleuler (1924), who commented on the uncontrollable and impulsive nature of the symptoms. Recent decades have brought a reemergence of interest in describing, defining, and classifying compulsive buyers in both the consumer behavior and psychiatric literature. Although some of the literature uses the term "compulsive shopper," we will use the term "compulsive buyer" which seems to better capture the behavior of interest that results in significant psychosocial problems.

It is estimated that 1.8–8.1% of the general adult population are compulsive buyers (Faber & O'Guinn, 1992). Compulsive buying appears to have a usual onset in late adolescence or early adulthood (although typically it is not recognized as problematic until much later in life), with a fairly chronic course (Black, Gabel, Hansen, & Schlosser, 2000; Black, Monahan, & Gabel, 1997; Christenson et al., 1994; Koran, Bullock, Hartston, Elliott, & D'Andrea, 2002). Compulsive buying is associated with significant financial, emotional, social, and at times, legal consequences, and is typically associated with expenditure of excessive amounts of both time and money (Christenson et al., 1994; McElroy, Keck, Pope, Smith, & Strakowski, 1994). It is estimated that 80–95% of compulsive buyers are female (Black, 2001). Compulsive buying is associated with high rates of psychiatric comorbidity including depressive disorders, anxiety disorders, substance use disorders, eating disorders, and other disorders of impulse control (Faber, Christenson, de Zwaan, & Mitchell, 1995; Koran et al., 2002; Ninan et al., 2000; Schlosser, Black, Repertinger, & Freet, 1994).

Numerous definitions of compulsive buying appear in contemporary literature. In attempts to describe the attitudes and behaviors of compulsive buying, O'Guinn and Faber (1989) put forth the following definition:

> Chronic, repetitive purchasing that occurs as a response to negative events or feelings. The alleviation of these negative feelings is the primary motivation for engaging in the behavior. Buying should provide the individual with short-term positive rewards, but result in long-term negative consequences. (p. 149).

Monahan, Black, and Gabel (1996) characterized compulsive buying as an "irresistible urge to buy and some form of tension relief (or gratification) after a purchase" (p. 59). McElroy et al. (1994) suggested specific diagnostic criteria for compulsive buying that include maladaptive preoccupation with buying or shopping that is "irresistible, intrusive, and/or senseless" (p. 247), frequent buying of more or for longer periods than was intended and beyond one's financial means. Furthermore, these authors suggest that preoccupations, impulses, and behaviors cause significant distress or interference and are not attributable to symptoms of hypomania or mania. Despite concerted attempts to define and describe compulsive buying as a psychiatric condition of clinical significance, as of yet it is not recognized as a distinct diagnostic entity, and currently is diagnosed under Impulse Control Disorder—Not Otherwise Specified in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (American Psychiatric Association, 1994). As defined in the *DSM-IV*:

> The essential feature of Impulse-Control Disorders is the failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person or to others . . . the individual feels an increasing sense of tension or arousal before committing the act and then experiences pleasure, gratification, or relief at the time of committing the act. Following the act there may or may not be regret, self-reproach, or guilt. (p. 609).

As researchers have struggled to define compulsive buying, a debate has emerged as to whether compulsive buying is really a disorder of impulsivity or of compulsivity. Christenson et al. (1994) found that all of the compulsive buyers in their study reported "irresistible urges, uncontrollable needs, or mounting tension that could be relieved only by buying" (p. 7) with a sense of gratification and tension release following a shopping episode which is consistent with an impulse control disorder. McElroy et al. (1994) also found that most subjects in their study reported "recurrent, intrusive, and irresistible urges or impulses to buy" (p. 246) and most experienced relief or pleasure after shopping. Other research identifying the presence of concomitant impulse control problems also lends support to compulsive buying being defined as a disorder of impulse control. Schlosser et al. (1994) found that concomitant impulse control disorders such as kleptomania, intermittent explosive disorder, and problems with gambling were common among compulsive buyers, again suggesting overlap between compulsive buying and impulse control disorders. They suggested that compulsive buying seems to have some commonalities with what are now classified as impulse control disorders, given that shopping behavior, similar to the behavior in other impulse control disorders, is initially experienced and perceived as pleasurable; however, in time the behavior results in significant negative consequences, distress, and impairment. Christenson et al. (1994) also found a high rate of concomitant impulse control disorders in their sample.

Others have argued that there appears to be a strong overlap between compulsive buying and anxiety disorders, particularly obsessive-compulsive disorder (OCD), suggesting that compulsive buying may be more accurately defined as a disorder of compulsivity. Christenson et al. (1994) found that two-thirds of their sample experienced urges or thoughts related to buying as intrusive, and 91.7% attempted

to resist urges, which are suggestive of obsessive-compulsive symptomatology. Furthermore, compulsive buyers scored significantly higher on a measure of compulsiveness. However, few subjects had other obsessions or compulsions, which led the authors to conclude that although there were similarities between compulsive buying and obsessive-compulsive symptomatology, there were differences as well. On the basis of the results of their study, Schlosser et al. (1994) reported many similarities between OCD and compulsive buying, including the "repetitive and problematic spending, intrusive thoughts about spending, and resistance to such thoughts and behavior" (p. 210). In addition, 22% of subjects in their study met criteria for obsessive-compulsive personality disorder. Frost et al. (1998) reported that compulsive buyers scored higher on a measure of OCD symptomatology. Several studies have reported concomitant OCD in compulsive buyers, although the prevalence rates have been highly variable. McElroy et al. (1994) found that 35% of their sample had a lifetime diagnosis of OCD. In contrast Christenson et al. (1994) found a prevalence rate of 12.5% and Schlosser et al. (1994) reported rates of OCD in their sample as only 4.9%.

Reports of pharmacological investigations have also drawn parallels between compulsive buying and OCD. Black et al. (1997) studied the effects of fluvoxamine, a serotonin reuptake inhibitor often used with OCD patients, on compulsive buying preoccupations and behaviors. Similar to OCD patients, participants reported the presence of intrusive thoughts. There were significant reductions in preoccupations and buying behavior following the initiation of fluvoxamine for most patients, and a gradual return of symptoms when medication was discontinued. Ninan et al. (2000) conducted a placebo-controlled study also looking at the potential efficacy of fluvoxamine in treating compulsive buyers. Although, there were significant reductions in symptomatology on active medication, a similar response was seen with placebo. In another study, Black et al. (2000) also found similar rates of improvements on fluvoxamine and placebo. Koran et al. (2002) found that citalopram, another SSRI, produced rapid, significant, and sustained reductions or remission in preoccupations and compulsive buying behavior in 71% of their sample, although this study was limited by a small sample size and an absence of a placebo control.

The literature on hoarding can also be interpreted as lending support to a connection between compulsive buying and OCD. Hoarding can be defined as "the acquisition of, and failure to discard, possessions that appear to be useless or of limited value" (Frost & Gross, 1993, p. 367) and is often observed in patients with comorbid OCD and obsessive-compulsive personality disorder. Black et al. (1997) found that many of their compulsive buyers were also hoarders. Frost et al. (1998) reported on two studies examining the association between hoarding and compulsive buying, both studies finding significant association between compulsive buying and hoarding. McElroy et al. (1994) also reported high rates of hoarding purchases in their study of compulsive buyers.

Taken together, these results suggest that compulsive buying does share some characteristics of impulse control disorders (irresistible urges, mounting tension previous to and an experience of relief following buying behavior) as well as with OCD (intrusive thoughts and uncontrollable urges related to buying). Yet neither diagnostic category seems to fully capture the phenomenology of this condition. Thus, research seems to suggest that the relationships among OCD, impulse control disorders and compulsive buying remain interesting but unclear (Christenson et al., 1994; McElroy et al., 1994; Schlosser et al., 1994). Given the apparent heterogeneity among compulsive

buyers, it would seem more relevant to explore the presence of subtypes within this condition and consider where on the spectrum from impulsivity to compulsivity they might be placed.

The work of Faber and colleagues (Faber, 2000a, 2000b, 2003; Faber & Vohs, in press) offers alternative perspectives for classifying different types of compulsive buyers and for differentiating buying behavior based on frequency of buying, motivation, and self-control. For example, Faber (2000a, 2000b) has reported that compulsive buyers differ in the frequency of buying behavior. Some feel compelled to shop every day and experience mounting anxiety on days they do not shop or buy. Other compulsive buyers report only occasional buying binges; typically triggered by some negative event in their life. The number of purchases made also can differ (Faber, 2000b). Some compulsive buyers report making multiple purchases of a specific item in one shopping trip, for example eight tee-shirts or three raincoats. Compulsive buyers also appear to differ in their desire for interpersonal interaction during or after buying. Some report desirable interactions with sales personnel (O'Guinn & Faber, 1989) while others dislike being interrupted by anyone while shopping (Elliott, 1994; Faber, 2000b). After purchasing, some compulsive buyers get pleasure from showing others what they bought, while many others hide their purchases from other people. Finally, Faber (2000b) has pointed out several different motivations that exist for some, but not all, compulsive buyers. Differences in motivation may be another important way of distinguishing different types of compulsive buyers.

Recent work on self-regulation has also suggested important ways in which compulsive buying may differ from impulse buying (Faber, 2003; Faber & Vohs, in press). Impulse buyers tend to engage in episodic purchasing, while compulsive buyers tend to "suffer from a chronic loss of impulse control that develops into a repetitive pattern marked by much more dire consequences than that experienced by the impulse shopper" (O'Guinn & Faber, 1989, p. 150).

Impulsive buying and compulsive buying also differ as to the motivation for purchasing. Impulse buying generally refers to a reaction to external stimuli experienced as an immediate desire for a specific item. Thus, desire for an object is typically the critical motivator (Faber & Vohs, 2004). In contrast, the motivation for compulsive buying often lies outside of the object itself (O'Guinn & Faber, 1989). Several studies report accounts of purchases going unused, returned or given as gifts (Christenson et al., 1994; O'Guinn & Faber, 1989; Schlosser et al., 1994), supporting the hypothesis that compulsive buying is not necessarily motivated by desire for an object.

There is mounting evidence that the motivation for compulsive buying is highly influenced by the desire to improve mood, escape negative mood states, and alter arousal levels (Christenson et al., 1994; Faber, 2000a; Miltenberger et al., 2003; O'Guinn & Faber, 1989). Buying is a way of coping with unpleasant feelings, and over time becomes the primary response when faced with negative emotions (O'Guinn & Faber, 1989). Miltenberger et al. (2003) similarly found that compulsive buyers were likely to engage in problematic buying when experiencing negative mood states.

Impulsive buying may primarily be due to underregulating impulses. Research in self-regulation suggests that people have limited resources for engaging in self-regulation and taxing these resources has been shown to increase impulse buying (Vohs & Faber, 2002; Faber & Vohs, 2004). In contrast, compulsive buying is thought to result from misregulation, focusing on controlling mood state rather than buying impulses (Faber, 2003; Faber & Vohs, 2004). This leads to short-lived improvements in

affect, but eventually gives way to guilt, shame, and a worsening mood. This worsening mood leads to new pressure to regulate affect, setting a vicious cycle in motion.

The different forms compulsive buying takes and the differences between compulsive buying and impulse buying may help to develop meaningful typologies of compulsive buyers. These differences may be related to important aspects of OCD or impulse control disorder. At this point, to simply view compulsive buying as one or the other is too simplistic. Compulsive buying appears to be a symptom that can occur in the context of other comorbidities or in isolation. The most parsimonious course at this point appears to be to keep an open mind until much more is known about this little researched condition.

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Chapter 11

CONTRASTING TOURETTE'S SYNDROME AND TIC DISORDERS WITH OCD

Kieron O'Connor

THE OCD SPECTRUM

The notion of the OCD spectrum has been influential since Hollander (1993) proposed that the unifying factor for several impulsive and compulsive disorders was a difficulty to inhibit or delay involuntary repetitive movement. However, from the outset there were difficulties in specifying what exactly defined this continuum. Initially, the continuum ranged from risk avoidance to risk seeking, but in a later publication by Hollander and Benzaquen (1997), the spectrum was viewed as a continuum with overestimation of harm on the compulsive end and underestimation of harm on the impulsive end. The dimension was explicitly conceived within a biological framework of hyper-frontality versus hypo-frontality linked to increased–decreased serotonergic sensitivity. Subsequently, the risk end of the dimension has been labeled variously pleasure seeking, stimulation, and tension reduction. The dizzying array of psychiatric problems now subsumed under the OCD spectrum umbrella suggests that the explanatory power of the spectrum has been gained at the expense of predictive power. However, in order to evaluate the spectrum construct here, I consider it should predict that Tourette's syndrome (TS), chronic motor tic (CMT), and OCD can be classified in different degrees along the same neurobiological, cognitive, and behavioral dimensions. Furthermore, differences in symptomatology should be adequately accounted for by variations along these dimensions.

NEUROBIOLOGICAL EVIDENCE

Since a key element in the spectrum debate relates to biologically based hypotheses—particularly the dimensions of hyper- hypo-functionality of the frontal lobes, serotonergic sensitivity, and cortical inhibition, it seems essential to at least give a cursory glance at the neurobiological evidence regarding TS and OCD. The hypothesis that both disorders involve deficits in the basal ganglia and frontal cortex has

not been generally supported by brain imaging studies. In fact, where brain imaging studies do show differences in regional brain activity in OCD and TS, the picture is generally more complex and topographically diffuse than can be subsumed under a uniform frontal hypo- hyper-functionality dimension (eg, Busatto et al., 2000). Furthermore, the pattern of brain activity changes depending on symptom expression, state, and task demand (Phillips et al., 2000) and this suggests reciprocal interaction between brain and behavior and the possible role of compensatory mechanisms producing cortical changes in both TS and OCD (Hugo, van Heerden, Zungu-Dirwayi, & Stein, 1999; Peterson et al., 1999). Studies, for example, showing elevated glucose metabolism in the left frontal region in OCD are very specifically localized and differences are reversible with either pharmacological or behavioral therapy (Baxter et al., 1992). Furthermore, any link between metabolic differences and type or severity of OCD or TS symptoms remains inconclusive.

If we turn to electrophysiological and psychophysiological measures of activation, we find evidence that both OCD and TS may be highly activated peripherally and centrally, but in distinct rather than in continuous ways. In particular, OCD activation seems centered predominantly on the attentional system which leads to a misallocation of resources (Towey et al., 1994) and problems inhibiting normally ignored exogenous, endogenous stimuli particularly under threat (Enright & Beech, 1993). Conversely in TS and CMT, there is evidence of chronic activation mostly centered over the motor areas and recruiting larger portions of these areas than is normal during action (Biswall et al., 1998). TS seems to have a chronic higher behavioral and cortical activation which impedes practice effects, optimal task-arousal modulation, and interferes with brain-behavior relations during motor planning (O'Connor, Brisebois, Brault, Robillard, & Loiselle, 2003). One of the few studies to directly compare TS, OCD, and controls on cortical activity during sensory and motor stages of the same evoked potential waveform complex indicated that components characterizing OCD were related to attentional orienting stages, and in TS, to motor preparation and response expectancy stages (Weate, Newell, Bogner, Andrews, & Drake, 1993). The important implication here is that both OCD and TS may have attentional or distraction problems, but each for very distinct reasons. In OCD, attention may be too intense and over-detailed particularly under threat whilst in TS the distraction may be due to higher level of sensori-motor activation impeding focus. In TS, for example, attention can often be better sustained whilst the person is physically occupied.

If we look at neurochemical evidence, there is very little direct support for the serotonergic sensitivity dimension. Cath, Spinhoven, Landman, & van Kempen (2001a), for example, found no relationship between serotonin measures and questionnaire measures of impulsivity or compulsivity. Research to localize the role of serotonin receptor hypo-sensitivity has led to conclusions that it is not directly involved in the mediation of the symptomatology of OCD but may act as a trait marker (Khanna, John, & Lakshmi Reddy, 2001). However, OCD and TS do seem to differentially respond to serotonergic reuptake inhibitors (SSRI). Unquestionably, in multicenter placebo-controlled studies, the main SSRI medications have shown efficacy in treating OCD whilst this medication is generally ineffective in TS (Cohen, Leckman, & Shaywitz, 1992). Although case studies report remission of OCD symptoms using SSRI medications, successful response up to a significant decrease of 35% in OCD symptoms is reported in only 20–50% of cases (Vythilingum, Cartwright, & Hollander, 2000). Other research points to a more complex neurotransmitter network in OCD

involving dopamine (Harvey, Scheepers, Brand, & Stein, 2001) and the successful use of dopamine antagonists such as risperidol. In fact, a linear dose–response relationship has been reported between olanzapine and improvement in an OCD patient, treatment-resistant to SSRI (Romasubbu, Ravindran, & Lapierre, 2000).

The pharmacological picture in TS is equally confusing with practically every neurotransmitter system seemingly involved in at least one successful case study. Observations consistent with a hyperfunctional dopamine system come principally from the limited success of treatment protocols using dopamine antagonists. But clinically speaking, not all patients improve as a result of neuroleptic administration (Regeur, Pakkenberg, Fog, & Pakkenberg, 1986). The initial drug of choice for TS was a low dosage of haloperidol, but pimozide, is now preferred in terms of efficacy and side-effect profile and shows good clinical response in some studies, but is contested in others (Sallee, Nesbitt, Jackson, Sine, & Sethuraman, 1997). Other dopamine agents antagonistic to presynaptic D_2 receptors similar to pimozide, such as pergolide, have shown benefit in TS (Griesemer, 1997). However, some of these dopamine agents show "tardive Tourette's" (ie, paradoxical worsening of TS), secondary to neuroleptics. Atypical neuroleptics such as olanzapine have shown more favorable outcome and less side effects in comparison to the more typical neuroleptics such as pimozide (Robertson & Stern, 2000). However, dopamine agonists such as deprenyl and clonidine have also shown efficacy in TS and alpha-2 adrenergic agonists such as quanfacine have reported improvement (Chapell et al., 1995).

The genetic mode of inheritance in both TS/CMT and OCD has been increasingly recognized as a complex and likely to involve the contribution of several genes. At one time the pattern of inheritance within the same families was deemed consistent with transmission of an autosomal dominant genetic locus. However, such a claim seems tenable only if subsyndromal 'caseness' using best-estimate criteria is included, and even then approximately 35% of TS and 50% of OCD show no family pedigree (O'Connor et al., 2001). The means of assessing probands and first degree relatives has always raised controversy (eg, Shapiro & Shapiro, 1992) and recently Burd, Kerbeshian, and Klug (2001) reported a 5–12% rate of misclassification in a 12-year follow-up study of probands and extended family members. McMahon, Carter, Fredine, and Pauls (2003) also note the importance of using structured clinical assessments for accurate classification.

One recent attempt to deal with the issue of phenotypic heterogeneity has been to try to relate heritability more to discrete clusters of both OCD and TS symptoms usually derived from factor analysis. Therefore, for example, Leckman et al. (2003) found good sib pair concordance for two out of their six OCD sub-factors, namely checking compulsions and symmetry, whereas Alsobrook and Pauls (2002) reported significant within-family correlation for three out of four Tourette symptom sub-factors, namely aggressive behavior, tics, and compulsions. But as Leckman et al. (2003) note, the very specificity of these symptom correlations begs the question of whether the familial contribution could be genetic or environmental. Interestingly, studies of psychosocial aspects of tic/OCD onset have largely paralleled genetic studies in failing to find specific parenting or learning patterns particular to TS or OCD (other than as apply to a general anxiety disorder factor) (Turgeon, O'Connor, Marchand, & Freeston, 2002). But recent studies focusing on childhood experience, rather than formal parenting patterns, seems to produce better precursors at least of cognitive appraisals relevant to OCD (Careau, Freeston, O'Connor, & Todorov, 2003).

One issue particularly relevant here is the hypothesis that TS/CMT and OCD are alternative expressions of the same genes. Pauls (1992) has been a long-term advocate of this position even suggesting a sex linkage and that TS is the male and OCD the female expression of the same gene. Overall, there does seem convincing evidence that first degree relatives of people with OCD show increased prevalence of tic disorders. But this prevalence does not seem to vary between OCD with or without tics, and does not support a sex linkage (Grados et al., 2001). Conversely, TS/CMT and OCD are present significantly more in the children of TD parents and more so if both parents have TS/TD (McMahon et al., 2003). But the reverse does not apply and childhood TS/TD does not predict TS/TD in the parents.

Both genetic and psychosocial profiles of TS and OCD are equivocal at this time. Although Pauls (2001) notes that we stand on the threshold of identifying the genes important to TS, we are clearly not there yet. Certainly better controlled genetic research paradigms may reveal environmental, nongenetic as well as genetic factors important to TS and OCD expressions.

OCD, CMT, AND TS AS BEHAVIORAL DISORDERS

Although the focus in the spectrum debate on discrete neurobiological indices is informative, both tics and OCD are essentially behavioral disorders involving an array of cognitive, emotional, and situational factors. Also, from a behavioral perspective, isolated movement sequences can be considered responses to more distal influences.

In the case of all OCD subtypes, the compulsive action is a direct or indirect product of an obsession. The person doubts: whether the door is closed, the hands are dirty, the object is not well placed, the newspaper contains information; as a consequence, the person performs the compulsive action to "neutralize" the doubting or anxiety—respectively, checking, washing, ordering, hoarding. In the case of mental compulsion, the person may perform ritualized mental neutralizations (praying, repeating phrases) aimed at alleviating the anxiety from the evaluation of an initial intrusion. Indirect compulsions may arise when a person may develop routines to try (maladaptively) to cope or avoid anxiety/discomfort.

The compulsive action then is not a movement in itself, but a response to a prior obsessional stimulus. The compulsive action has a purpose, and anxiety is generated about the consequences if the compulsive activity is not performed. Furthermore, the severity of OCD may be defined more by the nature of the obsession than by the compulsion. In fact, the presence of OCD with overvalued ideation (ie, poor insight) arguably constitutes a separate treatment resistant category that may require special approaches (Abramowitz, 1997; Dunne, 2000).

Clearly, however, in tics this premeditation does not occur. People are often unaware of their tics, or if they are aware, the awareness is often patchy. The diagnostic and statistical manual of mental disorders (*DSM*-American Psychiatric Association, 1987) distinguishes transitory tics from TS and CMT. Tourette syndrome is a diagnostic category with multiple tics including vocal (phonic) tics occurring several times per day, and although clinician consensus tends to view CMT as a milder form of TS, diagnosis of TS is categorical not dimensional. Tics may be simple or complex. Simple tics include blinking, cheek twitches, head or knee jerks, and shoulder shrugs.

Tics are mainly confined to the upper body and the most common occur in the eye, head, shoulders, and face. Tics can also be vocal and include coughs, tongue clacking, sniffing, throat clearing, hiccing, barking, and growling.

Complex tics may also take the form of self-inflicted repetitive injurious actions such as head slapping, face scratching, tense-release hand gripping cycles, or dystonic posture. Complex vocal or, more precisely, phonic tics (Jankovic, 1997) take the form of repeated sounds, words or phrases or swear words, and in rare cases, swearing (coprolalia). Normal actions and words of the person may also be repeated, and copying others can itself evolve into a complex repetitive movement, either by motor mimicry (echopraxia) or by repeating others' words, phrases, or sounds (echolalia).

Tic onset seems linked with tension level rather than with thought. The person with a tic rather experiences a rising tension or sensation sometimes anatomically located and termed a "premonitory urge" which precedes or maybe accompanies the tic, although this sensation is not always present with every tic. People with tics report a feeling of tension release after ticcing. Tic-affected muscles seem tenser and less flexible than non tic-affected sites (O'Connor, Gareau, & Borgeat, 1995). It is not clear, however, if tics do reduce muscle tension, or whether the muscle tension produces the need to be stimulated or activated which the tic then reinforces. Tics, unlike OCD compulsions, can be ego-syntonic and people may identify with their tics as in the "click with your tic" advocacy movement.

OCD and tics could then lie on a continuum between tension reduction and anxiety reduction. However, the conceptual basis for such a dimension is unclear, since the two end points do not indicate a natural gradation. In any case, subjectively tics may reduce tension but chronically increase tension or activation in a similar way that smoking augments stress (Parrott, 1999) or OCD reinforces anxiety. Therefore, in fact, tics and compulsive actions may both serve the same function of maintaining an internal aversive state of arousal.

One of the key emotional distinctions between tics and OCD compulsions is related to the emotional experience at the time of doing the tic or OCD ritual. In CMT, onset and feedback from the tic is sensorically-based. Although impulsive disorders give rise to pleasure (Hoogduin, 1986; Shapiro & Shapiro, 1988), tics seem triggered by psychasthenic feelings of sensory incompleteness or insufficiency (Leckman et al., 1994/1995). Cath et al. (1992) see this "felt emotion" as crucial to diagnosis. Summerfeldt, Richter, Antony, and Swinson (1999) have suggested that sensory-based perfectionism may be at the root of feelings of incompleteness. Summerfeldt et al. (1999) distinguished between symmetry compulsions with and without superstitious obsessions.

A sensory-cognitive distinction between tic and OCD disorders is captured well in the distinct subtypes of "just rightness." The "just right" phenomenon is a label applied to compulsions such as arranging books, or performing symmetrical movements, which seem to lack an obsessional precursor other than the need for something to be "just right." It is equally present in OCD with and without tics, and according to Leckman et al. (1994/1995), it is a complex mix of high activation, perceptual sensitivity, doubting, and repetitive action. However, a careful psychological analysis is necessary to effect such a differential diagnosis because although there may not be observable consequences to not performing the "just right" ritual, there may be consequences in the sense of how the person feels about themselves if things are not "just right." Coles, Frost, Heimberg, and Rhéaume (2002) have further suggested that the same

actions may be performed on the basis of two distinct motivations, (1) something not being just right and (2) the feared consequences. However, the authors consider this distinction as two subtypes of OCD rather than a distinction between tics and OCD.

COGNITIVE FACTORS IN TS, CMT, AND OCD

In recent cognitive models of OCD, thoughts and appraisals play a key role in the genesis of the disorder. The content of the intrusive thought may decide the subtype of the obsession, but these initial intrusions become significant because the person appraises them as significant due to assumptions about: the need to control thoughts, over-importance of thoughts, perfectionism, intolerance of uncertainty, over-estimation of danger, and responsibility (Frost & Steketee, 2002).

There has been very little study of cognitions in tics, but people with tics do not appear to show the same cognitive structure as in OCD. Tics are not preceded by an intentional thought and are not in general related to anxiety or anxious thoughts. Scores on the Padua Inventory are uniformly very low in CMT except for ambivalent items concerning repetitive movements. People with Tourette's syndrome do not report concerns of responsibility, guilt about actions, need to control thoughts, which reflect standard appraisals in OCD, neither do they report doubt and low self-confidence, although they may have self-image concerns. Appraisals relate generally realistically to onset and consequences of the tic (people will look at me, I will feel uncomfortable). The types of coping strategies used to contain or conceal tics are distinct from equivalent OCD coping strategies (Wojcieszek & Lang, 1995).

Unlike individuals with OCD and CMT, those with TS/CMT do not necessarily score high overall on measures of perfectionism, trait anxiety, or obsessionality (O'Connor et al., 2001). There were some early reports of high scores in TS on the Leyton obsessional inventory (Robertson, 1989) yet many items on the symptom scale of this questionnaire concern routine, repetition, neatness, and order which could be confused with complex tics. But people with CMT and TS, and to a lesser extent habit disorders (trichotillomania, bruxism, onychophagia) do seem to score higher than controls on subscales of the Frost multidimensional perfectionism scale related to personal standards and organization (O'Connor, Gareau, & Borgeat, 1997; O'Connor et al., 2001). The cognitive aspect of this perfectionism manifests itself in beliefs about the importance of being efficient, doing as much as possible, and not wasting time or appearing to do so. Perfectionism in OCD more typically takes the form of doubts about actions, and concerns over mistakes. Conversely, subscales of personal organization and personal standards seem not to relate to OCD (eg, Frost, Rhéaume, & Novara, 2002; Rhéaume, Freeston, Dugas, Letarte, & Ladouceur, 1995).

Now at first sight, this lack of OCD-like cognition in TS may appear to support the spectrum model. However, the spectrum model predicts an impulsive-compulsive dimension and would therefore predict impulsive thoughts concerning short term reward, stimulation or risky behavior to precede tics. People with TS do appear to have problems inhibiting inappropriate and sometimes self-mutilating actions, but this may be more a consequence of over-investment in attempting to impede the action than impulsivity. In fact, there is very little evidence that people with TS or CMT show high traits of impulsivity. For example, they do not score higher on trait measures of impulsivity, extraversion, or other risk-taking behaviors (Cath et al., 2001a).

COGNITIVE-BEHAVIORAL CONTEXT OF TS, CMT, AND OCD

Further qualitative differences between TS and OCD are evident when we look at the wider behavioral context of both problems. O'Connor, Gareau, and Blowers (1994) monitored high-, medium-, and low-risk situations in patients with CMT. All participants identified situations when the tic occurred and when it did not occur. High-risk situations could be either high- or low-arousal situations. However, whatever the physical activity level, the accompanying thoughts and feelings most frequently concerned impatience and frustration and not performing as desired.

In a more recent study, O'Connor et al. (2003) examined behavioral factors affecting tic onset. In this study, 39 people aged 18–63 including a range of tics were compared with 37 habit disorders, and completed a form ranking low-risk and high-risk activities when the tic was least and most likely to occur. In addition, subjective appraisals distinguishing the two types of activities were elicited. The results indicated that different tics were associated with different types of activities. Socialization was a high-risk for blink tics, manual work for shoulder tics, and intellectual work for head tics. The high-risk activities for ticcing were more likely to be appraised as 'active' in tic disorder with states characterized as tense and unsatisfying, whereas habit disorders were most likely to be evaluated as boring and inactive physically. The link between tics and behavioral activation is clearer when examining the style of planning action generally in CMT and TS.

Both TS and CMT show a premeditated style of action involving a tendency to attempt too much at once, premature abandonment of tasks, unwillingness to relax, pace action appropriately, invest more effort than necessary, or be in advance of self (O'Connor et al., 1997; O'Connor et al., 2001). A style of action questionnaire (STOP) discriminated well between CMT, OCD, and controls. Two robust dimensions emerged from the factor analysis: over-activity and over-investment in preparation. Internal consistency and test–retest reliability of high loading items for the tic sample were good (O'Connor, Aardema, & Brisebois, 2001). The first dimension reflected continual activity, a difficulty to keep still or to do one thing at a time. Typically, the overactive person will have several tasks planned at the same period time (eg, going to the mall, returning a library book, visiting a friend, going to the bank, etc). The result is that the person is dissatisfied, frustrated, irritable, feels trapped, and judges him/herself badly. The second dimension, over-investment, relates to doing more than is necessary and expending more effort than necessary on an intellectual, emotional, and physical level. Even if the person is immobile during the tic, thoughts can create unnecessary frustration and an overinvestment in negative anticipation and preparation (O'Connor et al., 1994). Paradoxically, such anticipation and preparation can elicit the tic.

People with OCD do not have this behavioral activity profile. In fact, in our discriminant function analysis, the STOP profile distinguished between OCD and CMT better than between generalized anxiety disorder and CMT. Although there seems evidence of a continuum in this behavioral activity profile between tic and habit disorder, it is clear that the same parameters do not characterize OCD (Petter, Richter, & Sandor, 1998). The close association between TS (but not OCD) and hyperactivity has long been reported in children, although in adults the over-activity is accompanied by perfectionistic thoughts about personal organization. Perfectionistic beliefs may

also be similar in CMT and habit disorder, and the person with CMT may report rigid all-or-nothing types of beliefs such as: "Either I do everything at once or I'm lazy."

HEIGHTENED SENSORY AWARENESS IN CMT COMPARED TO OCD

The hypersensibility of TS/CMT has been noted by several authors in visual, auditory, and tactile modalities (eg, Bliss, 1980; Kane, 1994) and this may complement high motor activation. Subjectively, individuals with TS and tic disorders report increased sensory discomfort, tingling, or itches, sometimes resulting directly in scratching, rubbing, or moving (eg, Leckman & Cohen, 1999, p. 27). The premonitory urge prior to ticcing could itself be a result of such hypersensitivity, and there is some experimental evidence of sensory excitability and augmented sensory evoked potentials in TS and tic disorder (Johannes et al., 2001; van Woerkom, Roos, & Dijk, 1994). Enhanced self-attention and self-awareness may result from increased self-focus. Kane (1994) has suggested that urges represent a heightened attention to physical sensations. He suggests that a particularly heightened sensitivity of the person with tics to somatic sensation produces a sensory focus that provokes the tic. A fairly consistent finding with CMT is the sensitivity of such patients to the judgment of others (Leckman & Cohen, 1999; Petter et al., 1998). The person with CMT is over-concerned with self-image, expecting that others will pass judgment (including on appearance of the tic), and detect subtle deficits in reaction and style, leaving the person dissatisfied with him/herself (Petter et al., 1998). In a recent study, items of self-image distinguishing CMT patients from controls were mainly concerned with feelings of being ill, at ease with others, and dissatisfied with self-image (O'Connor et al., 2001). The concern with self-image, self-focus, and sensitivity does not seem merely a consequence of ticcing since the self-focus can be present even in situations at low risk for ticcing. The opposite side of the coin to over-preoccupation with self-image and self focus in TS is auto-mutilation directed at specific body parts (commonly the eyes and head). Robertson, Trimble, and Lees (1989) compared self-mutilation in TS with distorted body-image common in psychotic self-mutilation, but the act in TS seems driven by a sensory-based self-focus.

OVERLAP BETWEEN TS, CMT, AND OCD: COMPLEX TICS AND COMPULSIONS

A key factor supporting the link between OCD and TS is the apparent high comorbidity between OCD and tics. Estimates of adults with TS and OCD vary between 28% and 63%, and OCD with tics around 17%. Distinctions between the two disorders may then be more apparent in a population with both OCD and tics. Cath et al. (2001c), in a series of studies differentiating between symptoms in tic-related OCD and TS, noted that current instruments are probably not sensitive enough to discriminate complex tics from OCD compulsions. The authors developed their own interview schedule that discriminated repetitive action and thoughts from anxiety-related obsessional fears. Shapiro and Shapiro (1992, p. 157) noted this confusion and listed a series of complex movements which could count as tics. As Cath et al. (2001b) point out, both OCD and TS can involve the same repetitive behaviors depending on whether they

are defined as impulsions or compulsions (distinguished according to the absence or presence of anxiety and goal directedness). The only clear distinction between OCD and TS on the basis of type of movement was rubbing and touching repetitions and echo phenomena. The findings reported by Cath et al. (2001b, 2001c) also support distinctions between a tic-related symmetry factor versus a nontic-related OCD washing factor. In this and other studies, TS with OCD are considered the most severe group in terms of symptomatology, and this may be because complex motor tics are confused with compulsions. Complex motor or phonic tics may have a higher frequency than obsessional compulsions. But the distinction between the two is only likely to be evident when considering cognitive factors preceding tic onset.

Cath et al. (1992) have also introduced the notion of a "cognitive tic" as a means of clarifying some of the confusion between intrusive mental impulses and obsessional ruminations. The distinguishing factor between "cognitive tics" and "ruminations" according to Cath et al. (1992) is that the former are playful, often following simple urges with no rationale behind them, whereas ruminations are more complex. So, counting a sequence of numbers for no reason would be classified as a "cognitive tic," whereas a flash of a scene of potential catastrophe would constitute an obsessional intrusion. However, in practice, it is difficult to separate the two. Obsessions may come to resemble cognitive tics in their repetitive senselessness when they become overlearned habits devoid of original cognitive motivation. In addition, a patient may retrospectively ascribe an intentional motivation to a tic, and a purposeless movement may sometimes be given a sense by the person post hoc (Cath et al., 1992). A head movement may be interpreted by the person as meaning there was something to look at. A defining distinction between cognitive tics and obsessions is the presence of cognitive rituals accompanying obsessions. These are mental operations following the initial intrusion, such as wiping away or suppressing or substituting intrusive thoughts as a way of neutralizing their impact. Mental neutralization is equivalent to the overt neutralization of compulsive rituals.

Obsessions are usually coherent thoughts, doubts or images of aversive events, leading to mental or physical neutralization. Mental tics are often initially neutral, stimulating with an elementary playfulness. If mental tics lead to actions, the action is often in accordance with the mental tic (eg, thinking of song theme leads to singing the theme) not as with OCD neutralization where the intent is to act against the obsession.

In certain cases, the tics and compulsions although functionally independent can interact. For example, a person with superstitious rituals viewed their tics as lucky even though tic appearance was independent of obsessional intrusions. Tics and rituals may also work against each other. A young man who had long dressing rituals would become impatient by the repetitive sequence. The impatience would provoke his tics which interfered with the dressing routine.

Shapiro and Shapiro (1992) argued that it is precisely the confusion between complex tics and compulsions that results in erroneous rates of comorbidity between OCD and TS or tic disorders. Current criteria are centered on the presence or absence of anxiety and obsessions accompanying the same repetitive movements. Tourette syndrome rituals, according to George, Trimble, Ring, Sallee, and Robertson (1993), tend to be ego syntonic, impulsive, and directed to the self, whereas obsessional compulsions are more elaborate, ego dystonic, and world-directed actions like cleaning or checking. But the attempt to distinguish tics and rituals is further hampered by the sometimes limited insight and awareness of people with these problems. Frequently,

TABLE 11.1. Some cognitive-behavioral differences between compulsions and tics in OCD and Tourette's syndrome

Tics in Tourette's syndrome	Compulsions in OCD
Action not goal-directed and regulates 'feel'.	Action goal-directed and aims to neutralize obsessional doubt and anxiety.
High chronic level of peripheral and central motor activation (eg, muscle tension).	Normal arousal except under conditions of stress. Hyperfunctionality in cognitive/attentional systems.
Tics can substitute for each other.	Rituals rarely substitute for one another.
Onset linked to behavioral activity and tension.	Onset linked to intrusive thoughts.
Predominant emotion at tic onset is frustration/impatience.	Predominant emotion preceding compulsive activity is anxiety.
Perfectionism in personal organization and personal standards.	Perfectionism concerns doubts about actions, concern over mistakes.
Over-active style of planning action	Normal style of planning action.
Respond to awareness training and relaxation therapy.	Do not respond to relaxation and awareness training.

individuals with OCD are more focused on their emotions than on their thoughts. Conversely people with tics may rationalize their action and give the tic some explanation post hoc. These problems could compromise questionnaire approaches (eg, Coles et al., 2002) to clarifying the distinctions. Differential diagnosis between complex tics and compulsions may be improved by assessing the wider behavioral context to better separate repetitive actions aimed towards regulating state and those regulating doubt. Some sample distinctions are listed in Table 11.1.

COGNITIVE-BEHAVIORAL TREATMENT APPROACHES

The behavioral approach to treating tics is by habit reversal (Azrin & Peterson, 1990). This is a multicomponent treatment package whose main ingredients are awareness training, relaxation, and competing response training. Although habit reversal is listed in the Expert Consensus Treatment Guidelines for OCD (*Journal of Clinical Psychiatry* 1997: 58(Suppl. 4)) there are no reported studies of the use of competing response training in OCD. From a theoretical perspective, it is difficult to see how asking a person with OCD to contract their muscles in response to the compulsive urge would alleviate the compulsive urge. Self-awareness training is also a specific strategy useful for managing tic disorders which is generally not useful in OCD. People with OCD are already painfully aware of their problem and it is the appraisals they have attached to unwanted intrusive thoughts that need to be addressed in treatment. Systematic relaxation has been repeatedly shown to be ineffective in treatment of OCD. It can even produce adverse effects, and at best, any positive benefits are indirect (Steketee, 1993; de Silva & Rachman, 1998). In a recent study, relaxation was used on a placebo control condition and found to be ineffective (Greist et al., 2002). Conversely, in tic disorder, relaxation is a useful therapeutic strategy. In a recent study, applying behavioral treatment to CMT and habit disorders (O'Connor et al. 2001) 68% of participants found the relaxation component helpful. Other treatment models for

CMT and TS have emphasized the importance of reducing sensori-motor activation levels to prevent tic onset (O'Connor, 2002).

Exposure and response prevention procedures appear to be a common strategy in OCD and TS. In TS, exposure has been used with partial success. Exposure therapy, particularly to premonitory sensations, has been useful for some severe Tourette's patients (Hoogduin, Verdellen, & Cath, 1997). But here again there are distinctions since in OCD it is the response prevention element which seems most important (Rachman & Hodgson, 1980). In any case, this application of the general principle of habituation is no more specific to TS or OCD than as applied to craving in cigarette smoking or cue exposure in addiction.

There have been no studies exclusively applying cognitive therapy to tic disorders, but studies including a cognitive therapy component in TS focus on situational evaluations, anticipations and attributions of the ticcing, rather than as in OCD on appraisals (O'Connor et al., 2001). Finally, the presence of tics does not impede the treatment of OCD and vice versa (Himle, Fischer, Van Etten, Janeck, & Hanna, 2003) and behavioral treatment of the one disorder does not necessarily impact on the other.

CONCLUSION

The position here is that TS, CMTs, and habit disorders form a behavioral continuum since relevant behavioral, emotional, and cognitive dimensions vary quantitatively amongst these disorders, and all respond well to the same basic behavioral treatment protocol (Pelissier & O'Connor, 2004). On the other hand, OCD is a separate disorder and the OCD spectrum construct is unsupported. There is no evidence that TS and OCD differ in degrees from one another or that the two problems can mutate by degrees into each other. Indeed, OCD may eventually itself split into two or more separate disorders on the basis of intensity of obsessional beliefs and the role of the subsequent cognitive and emotional appraisals. Importantly, there are areas of research, such as genetic/environmental contributions to phenotypical variation as discussed previously, which could rightly influence future clinician consensus. However, whereas we may legitimately talk of an "anxiety spectrum," "delusional spectrum," and "Tourette's spectrum," talk of an OCD spectrum does not seen viable provided the empirical and clinical evidence. On the contrary, there is a need for a more refined diagnostic protocol that distinguishes between OCD and complex tics, and in particular, complex cognitive tics. Table 11.2 lists some types of cognitive tics

TABLE 11.2. Examples of similar cognitive tics and mental compulsions

Cognitive tics	Mental compulsions
Counting letters on a billboard as quickly as possible.	Counting letters on a billboard to always arrive at an even number.
Playing a scene over in the head for fun.	Playing a scene over to see what went wrong.
Looking at every object in the room to feel complete.	Staring at every object in order to see it is correct.
Repeating the same word or phrase in the head until it feels right.	Repeating the same word or phrase to be sure it is pronounced correctly.
Mentally replacing objects or jumping over objects to keep active.	Mentally replacing objects so that they are in a good order.

that could be confused with obsessions. Such a differential diagnosis cannot be made without in-depth cognitive and behavioral analysis. Moreover, because the treatments in each case are distinct, such differential diagnosis has major implications for treatment matching and therefore the well-being of the patient.

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Part II

ETIOLOGY

Chapter 12

NEUROPSYCHIATRIC MODELS OF OCD

David R. Rosenberg, Aileen Russell and Andrea Fougere

As recently as 15 years ago, obsessive-compulsive disorder (OCD) was considered a rare and untreatable illness. Now recognized as a severe, highly prevalent, and chronically disabling disorder affecting between 1% and 3% of the population worldwide (Rasmussen & Eisen, 1994), empirically supported cognitive-behavioral and pharmacological treatments are available for OCD (March & Leonard, 1998). The clinical phenomenology/nosology and empirical treatment for OCD have been well delineated across the life span in both children and adults, thereby making OCD a leading candidate for neurobiologic study. Specifically, OCD may be less vulnerable to ambiguities in expression across the lifetime (eg, in comparison to bipolar disorder and major depressive disorder). Investigations of OCD close to illness onset can then remain applicable to adult patients. Indeed, combining unique assessment/treatment and neuroimaging/genetic expertise at specific performance sites has already resulted in substantial progress in understanding the brain mechanisms that may be involved in treatment response (or lack, thereof) (see Rosenberg & MacMillan, 2002 for review). A combination of expertise in technological and clinical disciplines provides an ideal forum for investigating mechanisms of treatment response in OCD.

The International Conference on Surrogate Endpoints and Biomarkers at the National Institute of Health (NIH) concluded that surrogate endpoints are objective endpoints that reliably serve as early indicators for clinically meaningful endpoints. At a subsequent NIH Workshop, De Gruttola et al. (2001) argued that recent advances in biosciences and technology have increased our ability to understand, measure, and model biological mechanisms. This necessarily requires collaboration of quantitative and laboratory scientists for the translation and appropriate implementation of surrogate neurobiologic markers into clinical trials and treatment development. Hoagwood and Olin (2002) have developed a conceptual "blueprint" model for the translation of science into practice, which requires that linkages be made and supported across a range of scientific disciplines and between scientists and practitioners. They emphasize the importance of translational research to facilitate widespread dissemination by incorporating interdisciplinary perspectives from diverse areas including neuroscience, genetics, and treatment development, among others. In this chapter we detail

this exciting story and discuss how treatment can serve as a heuristically valuable probe into the neurobiology of OCD.

MODEL: NEUROCIRCUITRY OF OCD

One of the more striking findings over the past decade in adult and pediatric neuropsychiatry has been the repeated identification of abnormal information processing in cortico-striatal-thalamo-cortical circuitry (see Rosenberg & MacMillan, 2002 for review). Cortico-striatal-thalamo-cortical computational functions are a property of the circuit as a whole and are isolated from adjoining cortico-striatal-thalamo-cortical circuits so that the behavioral/symptomatic consequences of dysregulation in cortico-striatal-thalamo-cortical circuits depends upon lesion specific functional neuroanatomic/neurochemical factors. These factors, at least in theory, should be differentially modifiable by psychosocial or pharmacological treatment approaches working via similar or distinct mechanisms (Sallee & March, 2001). Consistent with the approach advocated in evidence based medicine (Geddes & Carney, 2001), the treatment of OCD can be conceptualized as analogous to the treatment of diabetes, recognizing that the brain necessarily requires psychosocial interventions of greater complexity. While medication and psychosocial treatment is framed as a probe into the underlying neurobiology of OCD, in the broadest sense, it constitutes a test of the outlined neurodevelopmental model and, by inference, embraces the science-based trend toward conceptualizing mental illness within a medical framework (Hyman, 2000).

NEUROANATOMICAL STRUCTURES INVOLVED IN OCD

Global Changes

While total brain volume and intracranial volume has not been found to differ between OCD patients and controls (eg, Rosenberg & MacMillan, 2002), increased cortical gray–white matter ratios have been reported in OCD patients (Breiter et al., 1994; Jenike et al., 1996). Such findings may reflect an aberration in prenatal programmed cell death and/or a postnatal delay or reduction in myelination (Jenike et al., 1996; MacMaster, Dick, Keshavan, & Rosenberg, 1999; Rosenberg et al., 1997a). In addition, increased third ventricular volumes and increased ventricular brain ratios have been observed in children and adolescents with OCD (Behar et al., 1984; Rosenberg et al., 1997b). Recent advances in neuroimaging methodology also allow for the precise measurement of specific regions of interest implicated in the pathogenesis of OCD.

Basal Ganglia

The basal ganglia may represent the primary site of pathology in OCD (Rauch, Whalen, Dougherty, & Jenike, 1998). Disturbance in the basal ganglia's filtering and suppressing of cortical input is believed to be involved in the emergence of OCD symptoms (Insel, 1992). Increased rates of OCD are observed in basal ganglia

disorders including postencephalitic parkinsonism, Huntington's disease, Tourette's syndrome, pediatric autoimmune neuropsychiatric disorders associated with group A beta-hemolytic streptococcal infections (PANDAS), progressive supranuclear palsy and neuroacanthocytosis (Cummings, 1993; Pitman, Green, Jenike, & Mesulam, 1987; Swedo, 1994; von Economo, 1931). Bilateral reduction in basal ganglia volume has been observed in OCD patients compared to controls (Luxenberg et al., 1988; Robinson et al., 1995; Rosenberg et al., 1997b), although contradictory reports exist (Aylward et al., 1996; Jenike et al., 1996; Stein et al., 1993).

Increased basal ganglia volumes in PANDAS patients with OCD have also been demonstrated (Giedd et al., 1995; Giedd, Rapoport, Garvey, Perlmutter, & Swedo; 2000), consistent with the proposed antibody-mediated inflammation of the basal ganglia in PANDAS (Swedo et al., 1998). Serial MRI scans have demonstrated a dramatic association among basal ganglia volume, severity of OCD, and effective treatment with plasmapharesis in a child with the PANDAS subtype of OCD (Giedd, Rapoport, Leonard, Richter, & Swedo, 1996). Higher antistreptoylsin O titers have also been shown to be associated with larger basal ganglia volumes in patients with OCD with chronic or recurrent streptococcal infections (Peterson et al., 2000). These results suggest that the direction of change in the basal ganglia (eg, increase or decrease) may be less important than the gradient of change and perturbation from normal in the pathogenesis of OCD.

In contrast to volumetric MRI studies, functional neuroimaging studies in OCD patients have more consistently identified functional and metabolic abnormalities in the basal ganglia associated with severity of illness and response to treatment (see Rosenberg & MacMillan, 2002 for review). Comparable reductions in caudate glucose metabolism have been observed before and after cognitive behavioral therapy (CBT) or pharmacotherapy with serotonin-reuptake inhibitors (SRIs) in OCD patients (Baxter et al., 1992; Schwartz, Stoessel, Baxter, Martin, Phelps, 1996). Disturbances in basal ganglia-frontal cortical interactions and feedback loops may play a critical role in the pathogenesis of OCD (Insel, 1992).

Prefrontal Cortex

Neurobiological models have consistently implicated the prefrontal cortex in the pathogenesis of OCD (Baxter, 1994; Baxter et al., 1992; Insel, 1992). Szeszko et al. (1999) reported bilateral reductions in orbital frontal volume in OCD patients compared to controls. Alterations in anterior cingulate-basal ganglia-thalamocortical circuitry may also play a particularly critical role in compulsive behaviors (Alexander & Crutcher, 1990; Cummings, 1993). A developmentally mediated anatomic neural network dysplasia involving reduced basal ganglia volume and increased anterior cingulate volume may disrupt ongoing purposeful behavior in OCD (Rosenberg & Keshavan, 1998). Increased anterior cingulate volume associated with reduced basal ganglia volume has been reported in two independent samples of treatment-naïve pediatric OCD patients compared to controls (Rosenberg & Keshavan, 1998; Szeszko et al., 2004a).

Using cortical parcellation methodology based on the sulcal anatomy, abnormalities in anterior cingulate volume were specific to the gray matter with no alterations between OCD patients and controls observed in white matter (Szeszko et al., 2004a). Increased anterior cingulate gray matter in OCD patients is consistent with functional

neuroimaging studies in OCD patients demonstrating increased anterior cingulate glucose metabolism and activation associated with OCD symptom severity and treatment response (Baxter et al., 1996). Consistent with animal studies demonstrating the involvement of the anterior cingulate cortex in reward expectancy (Shidara & Richmond, 2002), a recent functional magnetic resonance imaging study suggested that the anterior cingulate may be critically involved in abnormal conflict detection as part of an overactive action monitoring system in OCD (Ursu, Stenger, Shear, Jones, & Carter, 2003).

The aforementioned findings may have important treatment implications. Pathological correlations among the ventral prefrontal cortex, basal ganglia, and the thalamus have been observed in OCD patients before treatment, but neither in healthy controls nor in OCD patients after effective treatment (Baxter et al., 1996; Brody et al., 1998; Rauch et al., 2002; Saxena et al., 1999; Saxena, Brody, Schwartz, & Baxter, 1998; Schwartz et al., 1996; Swedo, Pietrini, Leonard, 1992; see Figure 12.1). Such

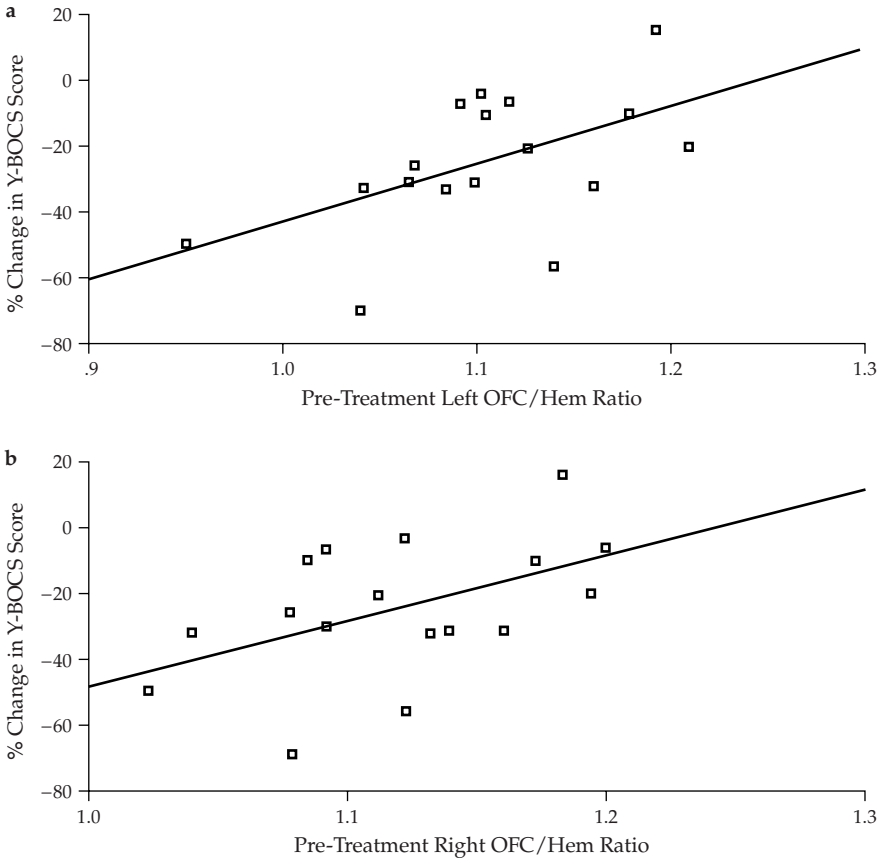

FIGURE 12.1. (*a*) Scatter plot of pretreatment glucose metabolic rate in left orbitofrontal cortex, normalized to ipsilateral hemisphere (left OFC/Hem), and change in Y-BOCS score after paroxetine treatment (Kendall's tau $= -.39$, $P = .01$); (*b*) Scatter plot of pretreatment glucose metabolic rate in right orbitofrontal cortex, normalized to ipsilateral hemisphere (right OFC/Hem), and change in Y-BOCS score after paroxetine treatment (Kendall's tau $= -.35$, $P = .02$).

investigation targets the fundamental question of whether or not specific patterns of pretreatment brain activity can indicate which treatment (eg, CBT or SRI) will be most effective for patients with OCD. The aforementioned literature suggests, for example, that decreased orbital frontal-hemispheric metabolic rates and increased activity in cingulate cortex predicted better response of OCD patients to SRI, while increased orbital frontal-hemispheric metabolic ratios pretreatment predicted enhanced response to CBT.

Thalamus

The thalamus is the final subcortical input to the frontal cortex, and when released from the inhibitory tonic influence of the striatum, it stimulates cortical output, thereby playing a key role in conscious information integration and perception (Baxter et al., 1996; Jones, 1997). The thalamus, particularly the dorsomedial nucleus of the thalamus, has been implicated in the pathogenesis of OCD. Lesions of interest can result in neuropsychological and neurobehavioral disturbances comparable to those observed in patients with OCD (Alexander, Crutcher, & DeLong, 1990; Cummings, 1993). Vascular and degenerative disorders of the thalamus often are indistinguishable from classically described "frontal lobe" syndromes (Cummings, 1993). Compulsive behaviors in animals can be provoked by alteration in thalamic function (Bergmann, Chaimovitz, Pasternak, & Ramu, 1974; Sasaki, Miyakawa, Sudo, & Yoshizaki, 1997). Thalamic stimulation has also been found to cause compulsive behaviors in human beings (Portenoy et al., 1986). Conversely, partial neurosurgery of the thalamus (eg, partial thalamotomy) has been reported to decrease OCD symptoms in treatment-refractory patients with OCD.

Using volumetric MRI, Gilbert et al. (2000) reported significantly increased thalamic volume in treatment-naïve pediatric patients with OCD compared to controls, which decreased significantly after monotherapy with the SRI paroxetine, to levels not significantly different from healthy controls. This "normalization" in thalamic volume was positively correlated with reduction in OCD symptom severity (Figure 12.2). Higher pretreatment thalamic volume in OCD patients predicted enhanced response to SRI, whereas lower pretreatment thalamic volumes predicted poorer response to SRI. To our knowledge, this is the only treatment data on volumetric measures in pediatric or adult patients with OCD. However, additional indirect support for these findings comes from two other independent reports. First, Kim et al. (2001) found increased thalamic gray matter in 25 medication free patients with OCD compared to controls. However, pretreatment volume was not explored. Second, a separate cohort of treated OCD patients on SRIs, (Jenike et al., 1996) found no significant change in thalamic volume between OCD patients and controls. Subsequent investigation also demonstrated no significant changes in thalamic volume in treatment-naïve pediatric OCD patients before and after 12 weeks of CBT (Rosenberg et al., 2000). Although further study is needed, these preliminary results suggest that decrease in thalamic volume may be specific to SRI pharmacotherapy, the result of a generalized treatment response, or spontaneous resolution in symptoms (Rosenberg et al., 2000).

Increased thalamic volume in OCD patients that decreased after SRI treatment may also be consistent with functional neuroimaging studies demonstrating increased metabolic activity in the thalamus that decreased after SRI treatment (Baxter et al., 1996). Grome and Harper (1986) have demonstrated that serotonin agonists reduce

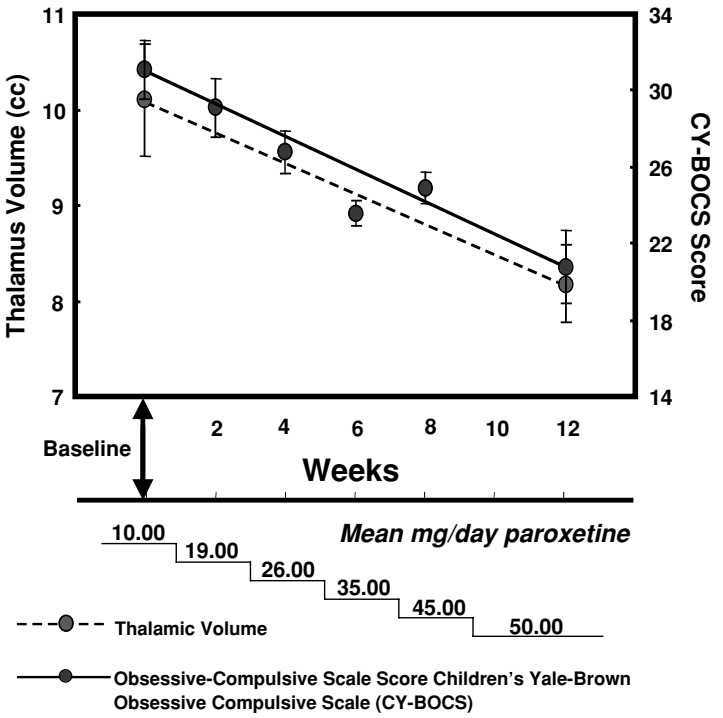

FIGURE 12.2. Decrease in thalamic volume associated with reduction in Obsessive-Compulsive Score of the Children's Yale-Brown Obsessive Compulsive Scales.

brain glucose metabolic rates in animals. As described in greater detail below, alterations in serotonin are believed to be critically involved in the pathogenesis of OCD (Insel, 1992). Serotonin has been shown to play a key neuromodulatory role in thalamocortical development and activity (Bennett-Clarke, Chiaia, & Rhoades, 1996; Bennett-Clarke, Lane, & Rhoades, 1995). Nonetheless, it is important to note that thalamic changes with SRI treatment may be an epiphenomena related to the underlying neuropathology and/or treatment intervention. Hemodynamically mediated medication treatment effects are also possible, although therapeutic doses of the SRI paroxetine do not appear to have significant hemodynamic or electrophysiological effects. Serotonin-reuptake inhibitors may also have dopamine blocking effects suggesting that alterations in the dopaminergic system may be related to the thalamic changes (Korsgaard, Gerlach, & Christensson, 1985).

Medial temporolimbic structures (eg, the amygdala and hippocampus) may play an important role in the neurobiology of OCD. Cybernetic models (Pitman, 1987) have proposed that the hippocampus plays an important role in compulsive behavior. The hippocampus maintains strong connections with basal ganglia regions that have been implicated in the pathophysiology of OCD (Robinson et al., 1995). Gray (1982) has proposed that the septohippocampal system functions to compare predicted events with actual events. When there is a mismatch of predicted versus actual events, the septohippocampal system redirects attention to stimuli associated with the mismatch and can take control of behavioral programs in order to monitor the environment

as needed. In this model, hippocampal dysfunction may be especially vulnerable to environmental novelty and more likely to inhibit ongoing, purposive behavior in favor of checking. It has also been proposed that hippocampal dysfunction in OCD results in failure to inhibit negatively reinforced stereotypic behavior (Pitman, 1987, Pitman et al., 1987). In this regard, it is interesting to note that Rauch et al. (1997) observed aberrant hippocampal activation in OCD patients during performance of an implicit memory task.

Amygdalocentric models of OCD have also been proposed in light of the amygdala's role in emotional appraisal and conditioned fear responses (Bechara et al., 1995; Davis, 1997; Ketter et al., 1996). The amygdala may be an important neuroanatomic substrate of the anxiety that maintains compulsive behaviors in OCD (Rauch et al., 1998). While Jenike et al. (1996) observed no significant differences in amygdala volume in adults with OCD as compared to controls, Szeszko et al. (1999) found bilateral reductions in both amygdala and orbital frontal volume. These findings are particularly intriguing given data that serotonin release in the amygdala is associated with emotional appraisal, fear modulation and conditioned anxiety (Kawahara, Yoshida, Yokoo, Nishi, & Tanaka, 1993; Sommer et al., 2001; Zangrossi, Viana, & Graeff, 1999). Specifically, Cheng, Wang, and Gean (1998) found serotonergic receptors to be involved in the inhibition of amygdala neurotransmission. Moderate to high densities of 3[H] paroxetine binding sites have been observed in the amygdala in quantitative autoradiographic studies (Chen, Clark, & Goldman, 1992; De Souza & Kuyatt, 1987), suggesting that the amygdala may be an ideal target for SRI treatment. Serotonin-reuptake inhibitors have also been shown to affect receptors in various amygdaloid nuclei (Costall, Kelly, Naylor, Onaivi, & Tyers, 1989; Gonzalez, Andrews, & Files, 1996).

NEUROCHEMICAL SYSTEMS INVOLVED IN OCD

Serotonin

Consistent evidence for the efficacy of SRIs in the treatment of OCD has resulted in their being the most widely used form of therapy for members of all age groups with this disorder (Cartwright & Hollander, 1998; Rosenberg, 2002). Findings that SRIs are more effective compared to nonserotonergic medication for OCD led to the "serotonin hypothesis of OCD." Serotonin transporter protein (5HTPR) capacity indexed in platelets by 3H-paroxetine is decreased in OCD patients compared to controls (Bastani, Arora, & Meltzer, 1991; Marazziti et al., 1997; Marazziti, Hollander, Lensi, Ravagli, & Cassano, 1992; Sallee, Richman, Beach, Sethuraman, & Nesbitt, 1996; Sallee, Stiller, Perel, & Rancurello, 1986; Weizman et al., 1986). Increased levels of 5-hydroxy-indoleacetic acid (5HIAA), a primary metabolite of serotonin, have also been observed in OCD patients when compared to nonpatients (Insel, Mueller, Alterman, Linnoila, & Murphy, 1985; Thoren, Asberg, Cronholm, Jornestedt, & Traskman, 1980). Further evidence for a serotonergic role in OCD is provided by robust correlations between a decrease in OCD symptom severity and an SRI-induced reduction in cerebrospinal fluid (CSF) levels of 5HIAA (Thoren et al., 1980) in platelet serotonin concentrations (Flament et al., 1985). Higher 5HIAA pretreatment levels were associated with

increased severity of OCD and predictive of an enhanced response to the SRI clomipramine (Swedo et al., 1992). Conversely, although Asberg, Thoren, and Bertilsson (1982) also found higher pretreatment 5HIAA levels to predict better response to clomipramine, they reported OCD symptom severity to correlate with decreased rather than increased pretreatment CSF 5HIAA levels. Some reports have found no differences in blood and CSF serotonin measures in OCD patients (Black, Kelly, Myers, & Noyes, 1990; Insel et al., 1985; Kim, Dysken, Pandey, Davis, 1991; Pandey, Kim, Davis, & Pandey, 1993). An individual's weight, height, diet, season of the year, activity, and menses can influence blood and CSF serotonin measures limiting their reliability (Insel & Winslow, 1992). It is important to note that these measures are limited in that they provide only a peripheral index of brain serotonin neurotransmission.

Hollander, Prohovnik, and Stein (1995) conducted a functional neuroimaging study in concert with pharmacological challenge using the mixed serotonin agonist-antagonist, meta-chlorophenylpiperazine. A metabolite of trazadone, *meta*-chlorophenylpiperazine acts at the postsynaptic 5-HT2c receptor and is thought to exacerbate OCD symptoms (Hollander et al., 1992, 1995; Pettibone & Williams, 1984; Pigott et al., 1991, 1993; Seibyl et al., 1991), although there are contradictory reports (Goodman et al., 1995). Hollander et al. (1995) found *meta*-chlorophenylpiperazine symptom provocation to be associated with a global increase in cortical blood flow in OCD patients while a subsequent investigation failed to confirm this finding and, in contrast, reported reduced global blood flow (Ho Pian, Westenberg, Den Boer, de Bruin, & van Rijk, 1998). The refinement and validation of more specific measures of serotonin in the brain may potentially clarify the role of serotonin dysregulation in OCD.

N-Acetyl-aspartate

The neuronal marker, *N*-acetyl-aspartate (NAA) is the second most abundant amino acid in the central nervous system (Birken & Oldendorf, 1989) and can be measured by MRI using a technique called proton magnetic resonance spectroscopy (^{1}H MRS). *N*-acetyl-aspartate is localized to mature neurons and not found in CSF, blood, or mature glial cells. Reduction in NAA can reflect neuronal dysfunction, decreased neuronal viability or neuronal loss (Tsai & Coyle, 1995). ^{1}H MRS permits a powerful, noninvasive, in vivo brain "biopsy" of compounds such as NAA and can reliably detect mean changes of 3% in NAA concentrations between two scans. Reduced NAA levels, suggesting reduced neuronal viability, have been observed in OCD patients compared to controls in the caudate nucleus (Bartha et al., 1998), anterior cingulate cortex (Ebert et al., 1997), and in the medial (but not lateral) thalamus (Fitzgerald, Moore, Paulson, Stewart, & Rosenberg, 2000). No significant differences in NAA were observed between OCD patients and controls in regions not implicated in the pathogenesis of OCD including the lateral thalamus, parietal white matter, and the occipital cortex (Ebert et al., 1997; Ohara et al., 1999; Rosenberg et al., 2000). The localized functional neurochemical alterations in NAA are intriguing since NAA changes have been shown to be reversible with treatment depending upon the extent of neuronal loss or dysfunction (Birken & Oldendorf, 1989; Maier, Ron, Barker, & Tofts, 1995).

Reduced levels of NAA, which are suggestive of neuronal dysfunction or loss in ventral prefrontal-striatal-thalamic circuitry in OCD patients, may result from

excitotoxicity caused by increased activity of afferent glutamate neurons (Salt & Eaton, 1996). Increased metabolic activity in ventral prefrontal cortex, the basal ganglia and thalamus (Baxter et al., 1996) are consistent with increased glutamate afferent input, which could be toxic to neurons in these regions with the resultant reduced NAA levels (Bartha et al., 1998).

Glutamate

Rosenberg and Keshavan (1998) proposed that abnormalities in glutamate–serotonin interactions may underlie OCD. Indirect support for this hypothesis comes from an animal model, in which transgenic mice with increased glutamate output to the striatum exhibit a phenotype resembling OCD and comorbid Tourette's syndrome (McGrath, Campbell, Parks, & Burton, 2000; Nordstrom & Burton, 2002). In a recently published genome scan of early onset OCD, suggestive linkage was found in a region of chromosome 9p, which contains the neuronal glutamate transporter gene SLC1A1 (Hanna et al., 2002). In a family based association study conducted by Veenstra-Vanderweele et al. (2001) no evidence of biased transmission was observed. Most recently, Arnold et al. (2004) found a positive association of the glutamate receptor gene, ionotropic *N*-methyl-D-aspartate 2B (GRIN2B), with OCD suggesting that polymorphisms in GRIN2B may be associated with susceptibility to OCD. The gene for the subunit of *N*-methyl-D-aspartate B encoded by the GRIN2B gene on chromosome 12 is preferentially expressed in regions of metabolic abnormality in OCD including the striatum in a developmentally specific fashion (Cull-Candy, Brickley, & Farrant, 2001).

Intact neuronal and glial cell density and function are required for normal metabolism of glutamate (Gallo & Ghiani, 2000; Magistretti, Pellerin, Rothmann, & Shulman, 1999). Glutamate plays a key neuromodulatory role in ventral prefrontal-striatal-thalamic circuitry (Kalivas, Duffy, & Barrow, 1989; Taber & Fibiger, 1993). The caudate nucleus receives an especially dense glutamate innervation from frontal cortex with stimulatory 5-HT2a receptors on GABAergic neurons inhibiting the projection of glutamate from prefrontal cortex to the basal ganglia and thalamus (Fonnum, Storm-Mathisen, & Divac, 1981; Koller, Zeczek, & Coyle, 1984). Glutamate afferents from frontal cortex account for the majority of axon terminals in the striatum (Parent & Hazrati, 1995; Salt & Eaton, 1996) so that ablation of the frontal cortex leads to a reduction in striatal glutamate. L-Glutamate diethylester, an antagonist of glutamate receptors, inhibits excitatory responses to cortical stimulations in the striatum (Spencer, 1976). Serotonergic neurons influence glutamate output, while glutamate potently inhibits serotonin release in the caudate nucleus (Becquet, Faudon, & Hery, 1990).

Rosenberg et al. (2000) conducted an in vivo ^{1}H MRS study in treatment-naïve pediatric OCD patients before and after monodrug therapy with the SRI paroxetine compared to age and sex-matched healthy pediatric controls. Caudate glutamatergic (Glx) concentrations were significantly elevated in psychotropic-naïve OCD patients compared to controls. In contrast, no significant differences in occipital cortex Glx were observed in OCD patients as compared to controls. After 12 weeks of SRI treatment, caudate, but not occipital, Glx decreased to levels not significantly different from controls and the reduction in caudate Glx was associated with a decrease in OCD symptom severity. In pediatric OCD, reductions in caudate Glx following

SRI treatment have been shown to persist after medication discontinuation if symptoms remain in remission (Bolton, Moore, MacMillan, Stewart, & Rosenberg, 2001). In contrast, no significant changes in caudate Glx were observed in psychotropic-naïve pediatric patients with OCD before and after 12 weeks of CBT (Benazon, Moore, & Rosenberg, in press). Therefore, reduced caudate Glx may be specific to SRI treatment rather than the result of a more generalized treatment response or spontaneous resolution of OCD symptoms. Since Glx activity has been shown to parallel brain glucose metabolism (Sibson et al., 1997), increased caudate Glx in OCD before treatment, that then decreases after SRI therapy, is consistent with prior findings of increased caudate glucose metabolism in OCD patients before treatment that decreased significantly after SRI therapy (Baxter et al., 1996). El Mansari, Bouchard, and Blier (1995) observed that SRI treatment increases serotonin release by desensitizing terminal serotonin autoreceptors in ventral prefrontal cortex suggesting that SRI treatment could alter serotonergic neurotransmission surrounding cell bodies in the prefrontal cortex which would alter projection of frontostriatal glutamate, thereby resulting in measurable changes in caudate glutamate concentrations (Rosenberg et al., 2000).

While preliminary, these results suggest that neuroimaging measurements could facilitate genetic investigations of OCD that may be more homogeneous than clinical phenotypes (Rosenberg & Hanna, 2000). These brain imaging profiles could be measured as either categorical or quantitative traits and suggest that neuroimaging could prove useful for better delineating the genetic heterogeneity of OCD or in describing potential differences between familial and sporadic OCD. Investigators at Wayne State University and the Centre for Addiction and Mental Health in Toronto are currently collaborating on a combined genetics-imaging study of a large sample of pediatric OCD patients before and after treatment. Although it is still not clear whether advances in neuroimaging and/or genetic studies will lead to advances in other areas, if a particular candidate gene were to be consistently associated with OCD, it may become more feasible to integrate and translate these findings into treatment development trials. Neuroimaging can potentially quantify the expression of susceptibility genes in OCD while monitoring the functional neuroanatomic and neurochemical effects of pharmacotherapeutic and psychosocial interventions for patients suffering from OCD. While costs of combining neuroimaging and genomic scans may be prohibitive, such approaches may lead to better assessments and treatments for the disorder.

It should be noted that an ongoing controlled multicenter trial of the antiglutamatergic agent riluzole (Rilutek), an agent that is FDA-approved for the treatment of amyotrophic lateral sclerosis in treatment-refractory OCD patients has recently been initiated, based in part, on the aforementioned glutamatergic alterations in OCD. A recently published case study reported the effectiveness and safety of riluzole in a patient previously refractory to all other treatments for OCD (Coric et al., 2003). Our laboratory is currently conducting ^{1}H MRS studies measuring brain glutamatergic concentrations in treatment-refractory OCD patients before and after treatment with riluzole.

Choline

Compounds that contain choline (Cho) can also be measured by ^{1}H MRS. The Cho signal measured by ^{1}H MRS is made-up of several important constituents in membrane synthesis and breakdown including phosphocholine;

glycerophosphocholine; and smaller concentrations of choline, acetylcholine, and CDP-choline (Barker et al., 1994). Smith et al. (in press) recently conducted a ^{1}H MRS investigation comparing 27 nondepressed, psychotropic-naïve pediatric OCD patients to age and sex-matched psychotropic-naïve pediatric patients with major depressive disorder and a group of healthy pediatric controls. Results revealed significantly increased left and right medial, but not lateral, thalamic Cho concentrations in OCD patients compared to both healthy controls and patients with major depressive disorder. No significant differences in medial thalamic Cho levels were observed between major depressive disorder patients and controls. Thus, localized functional neurochemical marker alterations in medial thalamic Cho differentiated OCD patients from both healthy controls and a psychiatric comparison group, suggesting that Cho may be an important biomarker specific to OCD.

The initial finding described above is encouraging, and suggest that additional research identifying sensitive and specific biomarkers in OCD could more narrowly define the phenotype by reducing heterogeneity in genetic studies and permitting earlier detection and treatment. Ideally, a biomarker such as Cho would have high sensitivity and specificity for OCD in affected patients (Bartha et al., 1998; Ebert et al., 1997; Rosenberg et al., 2000). Nevertheless, caution is also warranted particularly since Cho abnormalities have been shown to be reversible for many neuropsychiatric disorders (Bhatara et al., 1998; Moore et al., 2000b; Renshaw et al., 1997).

Increased thalamic Cho, as measured by ^{1}H MRS, may also be consistent with prior volumetric MRI studies in OCD that have reported increased thalamic volume in patients with OCD (Gilbert et al., 2000; Kim et al., 2001). Since the Cho signal measured by ^{1}H MRS arises principally from glycerophosphocholine and phosphocholine metabolites of phosphotidylcholine (Barker et al., 1994), a metabolite which plays a key role in intracellular signal transduction (Blusztajn & Wurtman, 1983; Exton, 1990, 1994), altered signal transduction may result from increased medial thalamic Cho, and therefore may be involved in the pathogenesis of OCD (Rosenberg et al., 2001).

Increased medial thalamic Cho in pediatric patients with OCD may also be consistent with prior suggestions of developmental alterations in myelinization in OCD (MacMaster et al., 1999; Rosenberg et al., 1997b; Rosenberg & Keshavan, 1998), since abnormalities in Cho have been reported in patients with multiple sclerosis in regions of acute demyelination (Ross & Michaelis 1994; Vion-Dury, Meyeroff, Cozzone, & Weiner, 1994). Since myelinization continues through the peak period of onset of OCD (Yakovlev & LeCours, 1982), developmental alterations in mylenization could lead to increased thalamic volume and thalamic Cho concentrations in OCD patients.

CONCLUSIONS

Current research findings illustrate the promise of integrating neurobiological studies designed to elucidate biomarkers of illness and treatment response. Collaboration across disciplines and synergy among study teams provides a model for multidisciplinary clinical and research practice. There are now unprecedented opportunities to combine expertise through use of state-of-the-art neuroimaging and genotyping methods to allow for the development, evaluation, and dissemination of new biomarkers, which may help to improve the assessment and treatment of OCD. Collaboration across the fields of psychiatry, psychology, biophysics, genetics,

among others, will increase the credibility and scientific rigor of this research and increase the acceptability of results across the various disciplines. Emphasis on the integration and translation of technologic advances in MRI and genetics as they relate to neurodiagnostic assessment and treatment development for OCD, is warranted. Such an approach will begin to lay the groundwork to address the most clinically relevant question of "which treatments to offer which patient with OCD with which set of subgrouping characteristics using relevant biomarkers (eg, SRI or CBT for patients with specific neuroanatomic/neurochemical patterns)?" The field of medicine is replete with examples where enhanced understanding of the biologic mechanisms underlying a specific disease has resulted in improved assessment and treatment of the condition.

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Chapter 13

COGNITIVE-BEHAVIORAL MODELS OF OCD

Roz Shafran

It has been argued that the best way to develop effective psychological interventions is to understand the development and maintenance of the disorder in question, and then to devise treatments that reverse the maintaining mechanisms (Clark, 1997). Such an approach has proven successful in a range of psychological disorders including panic disorder (eg, Clark, 1986), bulimia nervosa (Wilson & Fairburn, 2002), and OCD (Abramowitz, 1997). This chapter first describes the purpose of cognitive-behavioral models of OCD and then goes on to discuss five cognitive-behavioral models that have been proposed to account for OCD symptomatology. Each of these models is presented in detail, and the empirical evidence evaluating them is reviewed. The existing research incorporates a range of methods including questionnaire studies and controlled experimental laboratory research. It is concluded that the data are largely consistent with the cognitive-behavioral approaches, but that important questions remain to be addressed. Implications of these models for the conceptualization and treatment of OCD are also discussed.

THE PURPOSE OF COGNITIVE-BEHAVIORAL MODELS

Gelder (1997) considered models of psychological disorders to be theoretical schemes for ordering information in a broad and comprehensive way. Such models have two main purposes: First, they provide a means of understanding the development or maintenance of the essential phenomenological aspects of a disorder. In OCD, the cognitive aspects that require explanation include (but are not limited to) (*a*) the occurrence of persistent, repetitive intrusive thoughts, images, or impulses, (*b*) the experience of these thoughts as repugnant and unwanted, and (*c*) the attempts to ignore or suppress the thoughts. The primary behavioral symptoms in OCD that require explanation are compulsive rituals (eg, handwashing, checking), neutralizing (overt and covert), and avoidance. Related aspects to be explained include why the themes of the obsessions are consistent (ie, aggressive, blasphemous, sexual) and why it is that although 90% of people experience intrusive thoughts of a similar content, only

2–3% develop clinically significant OCD symptoms (Krochmalik & Menzies, 2003; Rachman & de Silva, 1978).

The second purpose of cognitive-behavioral models is to inform treatment. Although there is often an overlap between the factors that lead to the *development* of a disorder and those that *maintain* the disorder, it is important to draw the distinction between development and maintenance since it is the *maintenance* mechanisms that must be reversed if treatment is to be effective. Understanding the development of a disorder could give information as to the processes that might reverse the development of the disorder, how to prevent the disorder from occurring, and how to prevent relapse after successful treatment. However, the most useful information regarding intervention is obtained by understanding the mechanisms that maintain the disorder since reversing these maintenance processes will result in a weakening of *existing* symptoms.

There is a clear association between the two functions of a cognitive-behavioral model. Understanding the phenomenology of a disorder is necessary for the development of effective treatment interventions, and treatment interventions based on a clearly specified model can indirectly help inform our understanding of the disorder and lead to revisions of the theoretical model (see Salkovskis, 2002 for a detailed discussion of this topic).

THE BEHAVIORAL MODEL OF OCD: A PRECURSOR TO CONTEMPORARY APPROACHES

It is easy to overlook the important contribution of purely behavioral approaches to the understanding and treatment of OCD. The behavioral theory of OCD was based on learning theory; particularly the two-factor model of fear and avoidance presented by Mowrer (1939, 1960). This model proposed that normal intrusive thoughts, images, or impulses become associated with anxiety via classical conditioning so that when an intrusive thought occurs, anxiety increases. The person then learns, via operant conditioning, to reduce obsessional anxiety by escaping or avoiding stimuli that evoke obsessional thoughts. Thus, compulsive behavior is performed to escape from obsessional anxiety and is negatively reinforced by the reduction in anxiety that it engenders. Moreover, the obsessional anxiety is never extinguished.

In a series of experiments with dogs, Solomon and Wynne (1954) demonstrated that escape and avoidance responses to classically conditioned stimuli were highly resistant to extinction and continued long after pairing of conditioned stimuli with aversive consequences had stopped. Moreover, the escape and avoidance responses developed into stereotyped behaviors that were analogous to compulsive behaviors observed among patients with OCD.

There are multiple strengths of the behavioral model. First, it has empirical support as demonstrated in a series of now classic experiments in which exposure to obsessional stimuli resulted in increased anxiety, and performance of compulsive behavior decreased this anxiety (see Rachman & Hodgson, 1980). Second, the behavioral approach is based on the assumption that learning processes involved in the maintenance of OCD are normal and that there is nothing pathological about the occurrence of unwanted intrusive thoughts per se. The finding that 90% of people experience

unwanted intrusions of a similar content (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984) supports this view. Third, an effective treatment for OCD, exposure and response prevention (ERP), is derived directly from this model and the notion that OCD patients have developed avoidance and escape habits that prevent the natural extinction of obsessional anxiety. Exposure and response prevention involves (*a*) exposure to stimuli that evoke obsessional distress and (*b*) assistance with resisting urges to avoid or escape using compulsive behaviors. This treatment is highly efficacious with an estimated 75% of treatment completers improving significantly and remaining improved at follow-up (eg, Franklin & Foa, 2002).

It should, however, be noted that limitations of ERP include that many patients refuse or prematurely discontinue this treatment because of the prospect of confronting obsessional fears (Stanley & Turner, 1995), and that efficacy is likely to be significantly less for patients who present with obsessions without overt compulsions (Rachman, 1997).

In addition to the aforementioned limitations of behavior therapy, behavioral theory has been criticized for its failure to differentiate between the theoretical conceptualization of the range of anxiety disorders (Salkovskis, 1998). That is, the theory does not explain some of the clinical phenomena that are peculiar to OCD, such as the observation that the presence of a therapist decreases obsessional anxiety and compulsive checking (Rachman, 1976). These reasons, coupled with the observation that obsessions are cognitive phenomena, led to the need for consideration of cognitive components in which the crucial element was the meaning placed on the occurrence and content of intrusive thoughts (Salkovskis, 1985, 1989, 1999).

COGNITIVE-BEHAVIORAL MODELS OF OCD

Five cognitive-behavioral models are presented below along with supporting empirical evidence. Two issues are worth highlighting. First, although they diverge somewhat in emphasis, the various models are more similar than they are different. In particular, the fundamental premise of each model is that obsessional problems occur as a result of the appraisal of otherwise normal intrusive thoughts, images, and impulses as highly significant or threatening. In addition, all of the theories view such appraisals as the key cognitive process that leads to an escalation in the frequency and intensity of obsessive intrusive thoughts (Clark, Purdon, & Wang, 2003). The second point concerns the quality and nature of the evidence used to evaluate the different theories. Data from self-report questionnaire studies allow for the evaluation of hypotheses regarding associations but tend to be cross-sectional and therefore do not address the direction of causality. Experimental manipulations can inform about the direction of causality and therefore provide an excellent test of theory. However, experimental research conducted within a laboratory can be criticized for its artificiality and lacking in ecological validity. Successful treatment response, however, does not provide a basis upon which to evaluate the validity of an etiological theory since "the effectiveness of a particular treatment tells us nothing about the mechanisms involved in a particular disorder" (Salkovskis, 2002, p. 5). To illustrate, consider that the effectiveness of aspirin in alleviating headaches does not mean that a deficit of aspirin *caused* the headache. Nevertheless, if a treatment is *ineffective* yet changes a key hypothesized maintaining mechanism, this is an indication that the theory is incorrect.

Incorporating measures of hypothesized maintaining mechanisms will also allow treatment to better inform the theory from which it was derived (Salkovskis, 2002).

Salkovskis' Cognitive-Behavioral Theory of OCD

In 1985, Salkovskis proposed a cognitive-behavioral analysis of OCD in which appraisals of intrusive thoughts, particularly responsibility appraisals, were suggested to lead to compulsive behavior. This formulation proposes that people with obsessional problems appraise normal intrusive thoughts, images, and impulses as an indication that (*a*) harm to themselves or others is a particularly serious risk and (*b*) they may be responsible for such harm (or its prevention) (Salkovskis & McGuire, 2003). It is the specific interpretation of intrusive cognitions in terms of responsibility for harm to oneself or other people that is believed to link such intrusions with associated discomfort and urges to perform neutralizing behavior such as compulsive rituals, to reduce this discomfort.

This model begins with the understanding that intrusive thoughts are normally occurring phenomena. Indeed a series of studies has found that up to 90% of the population report unwanted, intrusive thoughts, ideas, images, and impulses that run contrary to their belief system and are similar in content to the obsessional thoughts described by patients with OCD (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984). Examples include unwanted urges to jump in front of trains or busses, ideas about germs or the possibility of illness, impulses to attack others sexually or nonsexually, and the idea that things are not "just right" or perfect (for a lengthy list, see Rachman & de Silva, 1978). Most individuals pay little attention to such thoughts and properly disregard them as normal and harmless. However, people who have an enduring tendency to appraise such intrusions as threatening, or indicating personal *responsibility* for harm (or for the prevention of harm) will experience the pattern of discomfort and compulsive behavior that characterizes OCD. The Obsessive Compulsive Cognitions Working Group (OCCWG) has defined "inflated responsibility" as:

> The belief that one is especially powerful in producing and preventing personally important negative outcomes. These outcomes are perceived as essential to prevent. They may be actual, that is, having consequences in the real world, and/or at a moral level. Such beliefs may pertain to responsibility for doing something to prevent or undo harm, and responsibility for errors of omission and commission (OCCWG, 1997). Examples include "If I don't act when I foresee danger, I am to blame for any bad consequences." (Frost & Steketee, 2002, p. 7).

A number of interrelated consequences are proposed to arise from the interpretation of normal intrusive thoughts as indicating personal responsibility for harm, including:

1. Increased discomfort.
2. Increased attention to the intrusions and external triggers of the intrusions.
3. Increased accessibility to the original intrusion and other related ideas.
4. Behaviors such as compulsions, neutralizing, avoidance, reassurance-seeking, and thought suppression which, in this account, are attempts to reduce or escape responsibility.

Each of these effects subsequently prevents the natural extinction of anxiety, prevents disconfirmation of the person's appraisal of the intrusion, and increases preoccupation with the intrusions (and therefore also their occurrence and frequency) (Salkovskis & McGuire, 2003). OCD patients are seen as trying too hard to exert control over their own cognitive function, over the occurrence of thoughts, over their memory, and over the details of how they perform everyday actions and so on. The increased frequency of intrusions is therefore largely due to the behaviors (overt and covert) that are motivated by faulty appraisals of intrusions. According to this account, successful completion of neutralizing reduces perceived responsibility, alleviates distress, and is almost inevitably accompanied by the absence of the feared consequence and the failure to disconfirm faulty appraisals. Hence the association among intrusion, appraisal, and neutralizing is strengthened. For example, a patient experienced the intrusive thought "I might harm my baby" and appraised it as indicating that she might become responsible for such harm. She repeatedly checked her baby for signs of abuse and found none (ie, her feared consequence had not occurred). The checking therefore reduced her perceived responsibility for harm, reduced her distress, and increased the likelihood she would check the baby when she had these intrusive thoughts again (Salkovskis & Freeston, 2001). The appraisal of responsibility arising from the occurrence and the content of intrusions can be at least partially independent according to this account, although they are often linked (Salkovskis, Richards, & Forrester, 2000).

Salkovskis' model has been criticized for being silent on motivational components specific to the disorder, not able to explain why people are upset by their appraisals, failing to account of the repetitive quality of obsessional symptoms, and disregarding emotional concerns as factors in etiology and maintenance (Jakes, 1996; O'Kearney, 1998). In response to these criticisms, Salkovskis and Freeston (2001) have specified that the theory's cornerstone is that the appraisal of intrusions motivates avoidance, compulsions, and neutralizing. Furthermore, cognitive theories are predicated on the notion that anxious patients are motivated by the same things that motivate us all; many aspects of obsessional behavior reflect a deliberate strategy of trying too hard to ensure one is not responsible for harm. Rather than actually having a general failure of mental control, memory, or decision making, patients with OCD are thought to be so highly *concerned* about these areas that they try too hard to control them, often resorting to counterproductive means, as described above.

The Origins of Inflated Responsibility

Salkovskis' cognitive-behavioral theory proposes that people are predisposed towards making particular appraisals of intrusive cognitions because of assumptions that are learned from past experiences. Such assumptions are thought to interact with critical incidents to produce negative interpretations of certain thoughts. Salkovskis and colleagues (Salkovskis, Shafran, Rachman, & Freeston, 1999) speculated that five particular types of learning experiences could contribute to the development of distorted beliefs about intrusive cognitions, including:

1. An early developed and broad sense of responsibility that is deliberately or implicitly encouraged or promoted during childhood (eg, being the eldest of many children).

2. Rigid and extreme codes of conduct and duty (eg, the religious belief "sin by thought, sin by deed").
3. Childhood experiences where sensitivity to ideas of responsibility develops as a result of never being confronted by it (eg, because of highly overprotective parents).
4. An incident in which one's actions or inaction actually contributed in a significant way to a serious misfortune (eg, a doctor making an error on a prescription).
5. An incident in which it appeared that one's thoughts and/or actions or inaction contributed to a serious misfortune (eg, wishing someone was dead and finding out that they died later that day).

Factors proposed to interact with these experiences include criticism and blame, situational increases in responsibility (eg, birth of a child/having an infant; Abramowitz, Schwartz, & Moore, 2003a), and the types of coincidental events illustrated in number 5 above. These experiences predispose one to make negative appraisals when a normal, intrusive, unwanted thought occurs. For example, having a broad sense of responsibility that was promoted during childhood and being blamed when events turn out badly may result in the assumption that *any* influence over outcome is equal to being fully responsible for that outcome (Salkovskis & McGuire, 2003). In such circumstances, if an intrusive thought such as "that piece of glass could harm someone" occurs, a person with the assumption "influence equals responsibility" may appraise the intrusion as "I'm responsible for making sure that the glass is removed and that nobody could accidentally injure themselves." Removal of the glass and repeated checking that the area is safe may then result. The occurrence of obsessional thoughts interacts with assumptions such as "influence equals responsibility" so that situations in which there is a possibility of harm by failing to act (omission) are seemingly transformed into situations where the person has actively chosen to allow harm to take place (commission) (eg, Wroe & Salkovskis, 2000; Wroe, Salkovskis, & Richards, 2000).

Empirical Evidence for the Model

A number of testable predictions arise from Salkovskis' cognitive-behavioral model, including that:

1. People with OCD have an inflated sense of responsibility compared to people with other anxiety disorders and nonclinical controls.
2. People with OCD assume that having any influence over negative outcomes equals being responsible for that outcome.
3. Increasing the sense of responsibility will increase discomfort and neutralizing behavior; conversely, decreasing responsibility will decrease discomfort and neutralizing.
4. Neutralizing will increase the frequency of intrusions and discomfort, and will prevent the disconfirmation of patients' fears.
5. Inflated perceptions of responsibility will increase vigilance for threat with the possible result of attention deficits in other areas.
6. Low mood will increase the persistence of intrusive thoughts.

Questionnaire Studies. Self-report questionnaires have been developed to test the prediction that people with OCD have an inflated sense of responsibility compared to people with other anxiety disorders and nonpatients. An early study demonstrated that patients with OCD do have higher responsibility for outcomes related to their thoughts than do matched nonclinical controls (eg, Freeston, Ladouceur, Gagnon, & Thibodeau, 1993). Other studies provided subsequent support for this initial finding. In the largest self-report study focusing exclusively on this question, 76 patients with OCD, 46 with other anxiety disorders and 138 nonclinical controls completed a "responsibility interpretations questionnaire" (Salkovskis et al., 2000). Those with OCD interpreted their intrusive thoughts in terms of responsibility to a significantly greater degree than did those with other anxiety disorders, who, in turn, had higher scores than nonclinical controls. The relationship between OCD symptoms and responsibility interpretations was significant even when the level of anxiety and depression was accounted for. A similar pattern of results was found in a large study by the OCCWG (2003) in which responsibility beliefs and appraisals made by 248 patients with OCD were significantly higher than those made by 105 non-obsessional anxious controls and over 300 nonclinical controls. In summary, the results of self-report questionnaire studies are consistent with the prediction that people with OCD interpret intrusive thoughts in terms of responsibility for harm (or its prevention), and that this phenomenon is specific to this patient group.

A second prediction of Salkovskis's model is that people with OCD assume that any influence over the outcome is equivalent to being *responsible* for that outcome. As a result, it is proposed that patients do not distinguish between being responsible for harm that they have actively caused (commission) and harm that they may have caused inadvertently by failing to act (omission). A self-report instrument designed to test this prediction was developed by Wroe and Salkovskis (2000) and administered to 42 patients with OCD, 25 patients with other anxiety disorders, and 53 nonclinical controls. The authors found that the occurrence of intrusive thoughts in specific situations increased the individual's level of distress and sense of involvement in the situation regardless of whether the person had OCD. However, unlike people in the other groups, those with OCD did not distinguish between errors of omission and errors of commission. Thus, when considering situations about which they are concerned, people with OCD seem to be more sensitive to omission than people without OCD. Most likely, this sensitivity helps to influence the decision whether or not to act to prevent harm.

In a subsequent study (Forrester, Wilson, & Salkovskis 2002), 22 obsessional and 30 nonclinical participants were provided with details of ambiguous situations and either a negative or neutral intrusive thought relating to this situation. Situations including an intrusive thought about harm were associated with more intense behavioral and emotional responses than situations with a neutral intrusion. Participants were also asked to rate their reactions to ambiguous situations in which possible negative consequences of a failure to act were either included or not. The authors concluded that the occurrence of intrusions likely transforms a situation that *might* involve an omission into a situation *requiring* an active choice. These findings are consistent with view that people with obsessional problems may be unduly sensitive to some situations because they are particularly likely to foresee harm in the form of intrusions, and that such foresight elevates anxiety (Forrester et al., 2002).

Laboratory Experiments. Studies involving the experimental manipulation of responsibility have been used to test the third prediction that increasing levels of responsibility will increase discomfort (anxiety) and neutralizing behavior; and that decreasing responsibility will decrease discomfort and neutralizing. In a study conducted by Ladouceur et al. (1995), nonclinical participants were randomly allocated to a "high" or "low" responsibility condition, asked to sort colored pills, and observed during performance of this task. Participants in the high responsibility condition were told that the sorting task was for a project concerning the development of medication for a widespread virus and therefore of high importance. Those in the low responsibility condition were told that the task was inconsequential since the researchers were only interested in the perception of colors. Consistent with the cognitive-behavioral model, participants in the high responsibility condition reported greater preoccupation with errors and anxiety during the sorting task, and hesitated and checked more than participants did in the low responsibility condition.

Lopatka and Rachman (1995) also tested whether perceived responsibility directly affected perceived risk, anxiety, and urges to compulsively check among 30 people with OCD. In their experiment, the level of responsibility was manipulated by having participants sign contracts attesting to their taking either full responsibility or no responsibility for the outcome of a variety of situations (eg, locking the door and walking away from it). Results indicated that under conditions of high responsibility, urges to perform compulsive behaviors (eg, checking), subjective levels of anxiety, and estimates of the probability of threat were all increased relative to the low responsibility condition. Feelings of control over the threat did not differ between the high and low responsibility conditions. Similar findings were obtained when responsibility was manipulated by the presence/absence of a trusted other person (Shafran, 1997).

The fourth prediction outlined above is that neutralizing behaviors increase both the frequency of intrusions and their associated discomfort, as well as prevent discomfirmation of patients' fears. In a study testing these hypotheses in a nonclinical sample, Salkovskis, Westbrook, Davis, Jeavons, and Gledhill (1997) gave instructions to 13 people to neutralize intrusions, and 15 people to distract themselves, but not neutralize. Those who had neutralized in the first phase of the study subsequently experienced more discomfort, stronger urges to neutralize and stronger urges to distract themselves than did those who had not neutralized previously. There was also evidence that engaging in neutralizing responses during the first phase made it difficult to stop neutralizing during the second phase. A recent study with OCD patients replicated these findings, which are consistent with predictions of the cognitive-behavioral model (Salkovskis, Thorpe, Wahl, Wroe & Forrester, in press).

The cognitive-behavioral model proposes that because of their inflated perceptions of responsibility, people with OCD have an increased vigilance for a broad range of potentially threatening stimuli. Such increased vigilance for threat may mean that there is also a diminished ability to inhibit other environmental stimuli such as irrelevant intrusive thoughts (Pleva & Wade, 2001). The hypothesis that inflated responsibility is related to deficits in attention (prediction 5 listed above) was investigated in an analog study in which a correlation was found between responsibility and visual selective attention deficits beyond those which could be attributed to the symptoms of OCD. Responsibility was a stronger predictor of obsessive-compulsive symptoms than was attention, and it was the only significant predictor of membership in the high

versus low obsessional symptom group. Specifically, the odds ratio from this model associated with responsibility indicated an almost 10-fold increase in the risk for being in the high obsessive-compulsive group for every standard deviation increase in responsibility (Pleva & Wade, 2001).

The sixth prediction stated above, that low mood will increase the persistence of intrusive thoughts, has been observed in a case-series analysis of 150 patients in which mood disorders were selectively associated with a worsening of obsessions (Ricciardi & McNally, 1995). A similar finding was also reported in an analog sample of undergraduate students who had a tendency to worry (Startup & Davey, 2003). In this study both responsibility and mood were successfully manipulated and the outcome variable was the number of catastrophizing steps taken by an individual in a catastrophizing interview. Catastrophizing is the tendency of individuals to apply a "what if . . . ?" questioning style to potential problematic features of their life and, in worriers, this usually results in the consideration of worse and worse outcomes. Although catastrophizing steps are not the same as intrusive thoughts, this study can provide insight into the relationship between responsibility, mood, and cognitive processes. Under conditions of high responsibility, those with negative mood had more catastrophizing steps than did those with neutral or positive mood. However, the relationship is likely to be complex since under conditions of low responsibility, those with positive mood had more catastrophizing steps than did those in neutral/negative mood states. The authors concluded that their findings suggest "a relatively complex relationship between responsibility and mood, where there are conditions in which high responsibility does not generate greater persistence than low responsibility" (Startup & Davey, 2003, p. 502). Since this was an analog study that assesses "catastrophisizing steps" rather than obsessional intrusions, strong conclusions regarding the cognitive-behavioral model require further evidence. Nevertheless, such an interaction between inflated responsibility and negative mood is not inconsistent with Salkovksis's cognitive-behavioral account and, furthermore, resonates with clinical experience. The authors have examined the perseveration of checking thoughts in a study yet to be published (Davey, Startup, Zara, & MacDonald, in press).

Rachman's Cognitive Theory of Obsessions

One motivation for developing a cognitive account of OCD was that behavioral theories did not seem to explain the occurrence of obsessions without compulsions. Moreover, the treatment of patients with this presentation of OCD within a behavioral framework met with relatively limited success (see Rachman, 1983). Drawing on the work of Salkovskis (1985) and Clark (1986), Rachman (1997) proposed a cognitive theory of obsessions hypothesizing that "obsessions are caused by catastrophic misinterpretations of the significance of one's thoughts (images, impulses)" (Rachman, 1997, p. 793). This leads to the prediction that obsessions will persist as long as these misinterpretations continue, and they will diminish when the misinterpretations are weakened. The misinterpretations are not limited to responsibility appraisals, but can include any interpretation that the intrusive thought is personally significant, revealing, threatening, or even catastrophic. As Rachman (1997) put it, such an interpretation has the effect of "transforming a commonplace nuisance into a torment" (Rachman, 1997, p. 794). The person usually interprets the intrusive thought in a personally significant way and as implying that the person is "bad, mad, or dangerous."

Examples include a devoutly religious patient who had obscene images about the Virgin Mary whenever she tried to pray. She interpreted such images as meaning that she was "a vicious, lying hypocrite and that her religious beliefs and feelings were a sham." In another example, a man whose wife had just given birth to their first child (a son) had unwanted thoughts of molesting the infant. He interpreted such thoughts as meaning that he was a pervert and "clearly unfit to be a parent." Such interpretations are thought to give rise to anxiety and dysphoria, with the consequence being intense resistance to the obsessions, attempts to suppress them, neutralization, and avoidance behavior. It is speculated that the dysphoria resulting from the appraisal of the intrusion as significant perpetuates a vicious cycle of obsessions, personal significance, dysphoria, obsessions, and so on.

Evidence in support of Rachman's (1997) theory include that cognitions can cause anxiety (eg, Clark, 1986), that patients report that their obsessions are meaningful (eg, Freeston et al., 1993), and the presence of cognitive biases such as thought–action fusion (TAF; Shafran, Thordarson, & Rachman, 1996). Thought–action fusion is usually considered to have two related forms. The first, Likelihood TAF, refers to the belief that having an intrusive thought increases the likelihood that a specific adverse event will occur (eg, "if I think about someone else falling ill, it makes it more likely that they will become ill"). The second component of TAF is labeled "Moral TAF" and refers to the belief that having an unacceptable intrusive thought is almost the moral equivalent of carrying out that particular act. For example the belief "if I think about swearing in Church, this is almost as bad as actually swearing in Church." Such beliefs will make one vulnerable to the catastrophic misinterpretation of intrusive thoughts. Other vulnerability factors include elevated moral standards, depression, and anxiety.

The cognitive theory was subsequently elaborated in an attempt to explain the frequency of obsessions, their persistence, the internal and external provocations of obsessions, and the nature of the content of obsessions (Rachman, 1998). Specifically, Rachman (1998) suggested that:

1. When a catastrophic misinterpretation of the significance of the intrusion is made, this increases the range and seriousness of potentially threatening stimuli and a wide range of stimuli may be converted from neutrality into threat. For example, sharp objects become potential weapons. Increasing the range of threats increases the opportunities for the provocation of obsessions. Internal sensations such as anxiety can also be converted into potential threat.
2. Avoidance or covert neutralization provides temporary relief from obsessional distress, yet the significance of the obsessions remains unaltered. Neutralization is viewed as attempts to "put matters right" and results in (*a*) relief, (*b*) the belief that the act of neutralizing prevented the feared event, and (*c*) a failure to disconfirm the significance of the intrusion. It is predicted that the significance attached to an obsession will remain unchanged (or increase) after repeated neutralization and the significance will decrease after repeated instances in which the obsession is not followed by neutralizing.
3. The very frequency of the intrusive unwanted thoughts can be catastrophically misinterpreted as evidence of their significance.
4. Attempts at thought suppression as a result of an inflated increase in the significance of an unwanted intrusive thought can paradoxically produce an increase in the frequency of the obsession.

Empirical Evidence

Given that Rachman's cognitive theory of obsessions is heavily influenced by Salkovskis's cognitive-behavioral account described above, there are few competing hypotheses that could differentiate the two models. The difference between them appears to be that Salkovskis' original model emphasizes appraisals of intrusive thoughts in terms of responsibility, whereas Rachman's cognitive theory of obsessions is broader and emphasizes a range of interpretations, some of which (such as TAF) are closely linked with responsibility appraisals. Hence, empirical support for Salkovskis's account (described above) are also consistent with the cognitive theory of obsessions. Other empirical studies are described below and can also be considered to be relevant to both Rachman's and Salkovskis's account.

Questionnaire Studies. Questionnaire studies concerning TAF are relevant to the evaluation of the cognitive theory of obsessions although they cannot provide a direct evaluation of the theory. The majority of such studies find a moderate association between obsessional symptoms and TAF, particularly Likelihood TAF (eg, Rassin, Diepstraten, Merckelbach, & Muris, 2001a; Rassin, Merckelbach, Muris, & Schmidt, 2001b; Shafran et al., 1996). However, some researchers have recently reported that TAF is not specific to patients with OCD, and instead may be characteristic of anxiety disorders in general (eg, Abramowitz, Whiteside, Lynam, & Kalsy, 2003c; OCCWG, 2003; Shafran & Rachman, in press).

Laboratory Experiments. Experimental investigations have been conducted examining the role that TAF plays in the etiology and maintenance of OCD. In an ingenious study, an experimental group of 19 high school students were wired up to electrical equipment and told that the equipment would monitor their thoughts (Rassin, Mercklebach, Muris, & Spaan, 1999). Specifically, they were told that if they thought of an "apple" it would result in a mild electric shock being administered to another person. Participants were also informed that they could prevent this electric shock by pressing a button immediately after an "apple" thought occurred. A control group of 26 students were simply told that the equipment would read their thoughts (eg, the word "apple"). Study participants indicated that they perceived the experimental manipulation as credible and the experimental group experienced more discomfort, more intrusive thoughts about apples, and more resistance to the word "apple" than did the control group. There was also an association between the number of reported "apple" thoughts and the frequency of button presses. These findings provide support for the hypothesis that believing one's thoughts can have real-world detrimental consequences (ie, TAF) can transform normal intrusive thoughts into anxiety-evoking, persistent, obsession-like intrusions.

In another experiment, student participants were asked to write the sentence "I hope that ____ is in a car accident" and then insert the name of a loved one (Rachman, Shafran, Mitchell, Trant, & Teachman, 1996). The researchers hypothesized that misinterpreting the significance of one's thoughts on a moral level, or based on the belief that such thoughts inflate the likelihood of harm, will elicit the urge to neutralize which will maintain obsessional anxiety. Results indicated that among students prone to TAF, activating TAF-related beliefs by writing the aforementioned sentence indeed elicited anxiety and the urge to neutralize (eg, destroy the piece of paper,

writing something positive), thus supporting the cognitive theory. Similar findings in students not prone to TAF have been reported (van den Hout, Kindt, Weiland, & Peters, 2002; van den Hout, van Pol, & Peters, 2001), although the role of neutralizing in these studies has been controversial (Shafran & Rachman, in press).

Purdon and Clark's Cognitive Theory Emphasizing the Importance of Thought Control

Purdon and Clark (1999) have developed a model in which (*a*) faulty beliefs about the importance of controlling one's thoughts and (*b*) negative misinterpretations of the consequences of failure to control unwanted intrusive thoughts are considered critical to the pathogenesis of obsessional problems. Examples of faulty beliefs include "I must control every thought that enters my mind, especially negative ones," "losing control of thoughts is as bad as losing control over behavior," "I would be a better person if I could control unwanted thoughts," and "control over thoughts is an important part of self-control" (Purdon & Clark, 2002, p. 31). It is proposed that such beliefs result in (*a*) heightened vigilance for the occurrence of the very intrusive thoughts to be controlled and (*b*) active resistance to such thoughts, for example, by attempting to suppress them.

Notably, the importance of controlling one's negative thoughts is addressed in both Salkovskis' and Rachman's models. For example, Rachman (1998) suggested that "an inflated increase in the significance attached to an unwanted intrusive thought, such as an obsession, will lead to more vigorous and intense attempts to suppress such thoughts" (p. 393). Salkovskis (1999) also delineates the role of attempted thought suppression in the maintenance of obsessions. Like Clark and Purdon (1993), these accounts suggest that attempts to suppress unwanted or "unacceptable" thoughts (resulting from appraisals of such thoughts in terms of responsibility/personal significance) represent one mechanism by which obsessions increase in frequency. This may occur because attempts to suppress thoughts are usually unsuccessful and may paradoxically increase the frequency of the target thought. This model of the development of obsessions is based on the work of Wegner and colleagues (Wegner, Schneider, Carter, & White, 1987) who found that deliberate suppression of a neutral ("white bears") thought was associated with an *increase* in its frequency during and after thought suppression.

The failure in thought control that results from the paradoxical effects of thought suppression, and for other reasons such as a decline in mood state resulting from initial failed attempts at thought control, are thought to result in escalating attempts to regain control. Such attempts can reinforce other beliefs about the thought (eg, "this thought is revealing about my true nature") and further exacerbate low mood (Clark & Purdon, 1993; Purdon & Clark, 1999). Moreover, failed attempts to control unwanted thoughts may evoke more catastrophic beliefs about the responsibility and personal significance concerning such thoughts. For example, "if I try and still can't control this terrible thought, it must mean the thought is *really* important and I must do something about it." Purdon (1999) proposed that patients with OCD are more likely to engage in thought suppression attempts than are individuals with other anxiety disorders because of the unique ego-dystonic quality of obsessions. That is, obsessional stimuli elicit stronger resistance and urges to suppress or control

as compared to more ego-syntonic cognitive phenomena such as worries, which are thought to evoke less resistance (Purdon, 1999).

Empirical Evidence

Questionnaire Studies. Self-report questionnaire research has shown that patients with OCD do attempt to control their intrusive obsessional thoughts. Clark and Purdon (1995) developed a measure called the Meta-Cognitive Beliefs Questionnaire, which assessed a range of beliefs about intrusive thoughts (including responsibility) which patients with OCD may possess. Indeed, patients with OCD scored more highly on this measure than did nonclinical individuals. Moreover, the subscale measuring control over thoughts was the only subscale that predicted the severity of obsessional symptoms among this nonclinical study sample.

Two studies have used the Thought Control Questionnaire (TCQ; Wells & Davies, 1994) to examine strategies that patients with OCD use to deal with their obsessional thoughts. The TCQ measures the tendency to use five categories of strategies for controlling intrusive cognitive stimuli, including: (*a*) distraction (eg, "I do something that I enjoy"), (*b*) social control (eg, "I talk to a friend about the thought"), (*c*) reappraisal (eg, "I challenge the thought's validity"), (*d*) punishment (eg, "I get angry at myself for having the thought"), and (*e*) worry (eg, "I focus on different negative thoughts"). Wells and Davies (1994) found that worry and punishment strategies were especially maladaptive and related to measures of psychopathology in their large student sample. In the first TCQ study with OCD patients, Amir, Cashman, and Foa (1997) found that patients with OCD used worry, punishment, social, and reappraisal strategies more often than did non-anxious control participants. Moreover, the use of worry and punishment predicted more severe OCD symptoms. A second study by Abramowitz, Whiteside, Kalsy, and Tolin (2003b) replicated and extended the earlier findings by demonstrating that even after controlling for general anxiety and depression, patients with OCD used maladaptive thought control strategies more often than did nonclinical individuals and those with other anxiety disorders. Abramowitz et al. (2003a, 2003b, 2003c) concluded that the use of thought control strategies such as worry and punishment contributes to the maintenance of OCD symptoms because such strategies preserve mistaken interpretations of intrusive thoughts and evoke increased attempts to suppress the thoughts.

Also consistent with the cognitive-behavioral model, the perceived need to control thoughts was a significant and unique predictor of immediate and subsequent efforts to suppress intrusive thoughts, even after controlling for other types of thought appraisals (Purdon, 2001). Interviews conducted by Freeston, Ladouceur, and colleagues (eg, Freeston & Ladouceur, 1997) confirmed that people with OCD engage in strategies to control their thoughts including compulsions, distractions, and self-reassurance, and these strategies appeared specific to patients with OCD.

Beliefs about thought control, as assessed by measures, developed by the OCCWG correlate with measures of OCD symptoms and distinguish patients from anxious and normal controls (OCCWG, 2001, 2003). Wells and Papageorgiou (1998) found that beliefs about uncontrollability and danger predict obsessional thoughts, although Emmelkamp and Aardema (1999) did not replicate this finding. In an undergraduate sample, beliefs about the control of intrusive thoughts and perceived negative

consequences due to uncontrolled mental intrusions had a unique and specific relationship with obsessions (Clark et al., 2003).

Barrett and Healy (2003) recently examined OCD-related cognitions in children with the disorder. Ratings of responsibility appraisals, estimates of probability, symptom severity, TAF, self-doubt, and cognitive control were compared in 28 children with OCD, a clinical control group of 17 anxious children, and a nonclinic control group of 14 children. Consistent with all the cognitive-behavioral theories, children in the OCD group gave significantly higher ratings of responsibility, severity, TAF, and less cognitive control in comparison to nonclinic children. However, OCD children were only differentiated from anxious children on ratings of cognitive control. The authors concluded that this study provided preliminary evidence that cognitive-behavioral accounts of OCD can be extended to children. Moreover, these findings support Clark and Purdon's emphasis on cognitive control as an important feature in the maintenance of OCD.

In questionnaire studies measuring thought suppression, the tendency to suppress was associated with OCD symptoms severity, but not severity of other anxiety disorders (Rassin et al., 2001). Although consistent with Purdon and Clark's (2002) theoretical model, the exact mechanism by which thought suppression may exacerbate psychopathology has yet not been clarified (Rassin & Diepstraten, 2003). Similarly, Smari and Holsteinsson (2001) found that thought suppression, along with responsibility appraisals, mediated the relationship between intrusive thoughts and obsessive-compulsive symptoms in a large study of 211 nonclinical individuals. Although self-report questionnaire studies may shed some light on the relationship between thought suppression and OCD symptoms, a laboratory paradigm that addresses causal hypotheses has been developed; the results of these experiments are described below.

Laboratory Experiments. Overall, the results of studies on whether attempted thought suppression produces paradoxical effects (as described above) are equivocal. A meta-analytic review (Abramowitz, Tolin, & Street, 2001) found little evidence that target thoughts increase while a person is trying to suppress them. However, there was some support for a "rebound effect"; that is, at some point *after* attempting to suppress target thoughts, people experience more intrusions of those thoughts compared to individuals who did not attempt to suppress them. Because of the obvious relevance, researchers interested in OCD have conducted a number of studies to examine whether thought suppression can cause obsessions. In one of the earliest studies, Salkovskis and Campbell (1994) had nonclinical participants identify naturally occurring intrusive thoughts. Half of the participants were then instructed to attempt to suppress these thoughts for 5 min, during which the occurrence and characteristics of the target thought were assessed. Assessment continued during a subsequent non-suppression period. Results indicated that attempts to suppress can result in increased cognitive intrusions.

In the first experimental study of thought suppression using patients with OCD, Janeck and Calamari (1999) found limited evidence for enhanced paradoxical effects of thought suppression, but there were significant methodological limitations of that study. More recently, Tolin, Abramowitz, Przeworski, and Foa (2002) reported two studies on thought suppression in OCD. In the first investigation, 15 patients with OCD, 14 nonclinical controls, and 16 anxious controls were asked to suppress thoughts of a "white bear." Patients in the OCD group, but not the other two groups, showed

an increase in "white bear" thoughts during suppression attempts and none of the groups evidenced a subsequent rebound effect. Tolin et al.'s (2002) second experiment was designed to overcome potential reporting biases and used a lexical decision paradigm that measured the priming strength of a target word under thought suppression conditions. Patients with OCD showed a decreased lexical decision latency for their suppressed thought, thus showing the paradoxical effect of thought suppression. Although the findings were interpreted as suggesting that deficits in cognitive inhibitory processes may underlie the intrusive repetitive nature of clinical obsessions, they are also consistent with the cognitive behavioral accounts described above.

A Cognitive-Behavioral Model Emphasizing Danger Expectancies

Carr (1974) was the first to propose that an inflation in estimates of the likelihood and consequences of danger were at the heart of OCD, and McFall and Wollersheim (1979) later suggested that it was the appraisal of threat that was critical understanding OCD. According to "threat-based" models, optimal treatment must involve procedures that maximize the opportunity to decrease excessive danger beliefs. Menzies and colleagues (eg, Jones & Menzies, 1997, 1998a, 1998b) have investigated this model empirically and have attempted to integrate it with Salkovskis's cognitive-behavioral approach by suggesting that the mechanism by which responsibility influences OCD is by impacting estimates of severity of negative outcome (Menzies, Harris, Cumming, & Einstein, 2000). Such "danger-based" accounts can be criticized for failing to distinguish between OCD and the other anxiety disorders which are also characterized by elevated estimates of danger (Salkovskis, 1996). Specifically, Salkovskis (1998) suggested that if the appraisal solely concerns harm or danger without an element of responsibility, then the effect is more likely to be general anxiety or depression. Nevertheless, the "threat-based" model of OCD has been investigated in individuals with contamination obsessions and compulsive washing rituals in a series of studies by Jones and Menzies (1997, 1998a, 1998b).

Empirical Evidence

Questionnaire Studies. In one study, Jones and Menzies (1997) asked 27 patients with OCD to provide ratings of danger expectancy, responsibility, perfectionism, anticipated anxiety, and self-efficacy before and during a task involving exposure to various stimuli such as potting soil, animal hair, food scraps, and raw meat. Correlational analyses examined the associations between the self-report ratings and anxiety, urge to wash hands, time spent in task, and duration of washing after the experiment was complete. Strong relationships between expectations of danger and these dependent measures were found, even when mediating variables were held constant. Jones and Menzies (1997) concluded from this study that danger expectancies are the most likely mediator of washing-related behavior in OCD (Jones & Menzies, 1997).

Laboratory Experiments. In an experimental study of 18 undergraduate students who had displayed washing/contamination concerns, Jones and Menzies (1998a) manipulated the perceived level of danger during a task involving exposure to a mixture of contamination-related stimuli such as animal hair, food scraps, potting soil, and raw meat. Participants in the high-danger condition were informed that there was a

possibility of developing an illness from bacteria in this mixture, whereas those in the low-danger condition were reassured that there was no risk of illness. All participants were asked to immerse their hands for 5 min in the bin containing mixture. Consistent with the danger expectancy theory, participants in the high-danger condition showed greater avoidance and spent longer washing their hands after completing the task compared to participants in the low-danger condition.

The hypothesis that responsibility influences OCD by impacting on estimates of the severity of possible negative outcomes has also been examined. Menzies et al. (2000) assigned students to complete a questionnaire with scenarios in which they either had a high degree of responsibility for a negative outcome, or in which they had a low level of responsibility for the outcome. Participants also rated the perceived likelihood and severity of the negative outcome. There were significant differences between groups on ratings of the severity, but not the likelihood of outcome. Specifically, participants given scenarios in which they had a high degree of personal responsibility for the outcome rated the severity of negative outcome as greater than did those who were not personally responsible for the outcome. Menzies et al. (2000) concluded that increasing perceptions of responsibility increases the cost associated with a negative outcome.

A Cognitive Theory of Compulsive Checking

Rachman (2002) has recently proposed a more detailed cognitive analysis of compulsive checking. According to this account, compulsive checking occurs when people who believe that they have a special responsibility for preventing harm (mostly to others) are unsure that the perceived threat has been reduced or removed. The intensity and duration of checking is therefore determined by the following variables (termed *multipliers*): increased responsibility, increased probability of harm, and an increase in the anticipated seriousness of the potential harm. The recurrence (ie, compulsive nature) of checking is proposed to result from a paradoxical increase in responsibility, an increase in perceived probability of harm, reduced confidence in one's own memory, and the absence of a certain end to the threat. Specifically, this theory views compulsive checking as attempts to achieve absolute certainty regarding the *absence* or *unlikelihood* of harm (ie, safety). However, Rachman (2002) proposed that attempts to check for safety produce adverse affects that turn the checking behavior into a self-perpetuating mechanism as follows:

1. An unsuccessful search for certainty that probability of harm has been reduced or removed.
2. Repeated checking tarnishes memory of checking which makes achievement of certainty even less likely.
3. The perceived probability of harm (and possibly the seriousness of harm) is elevated when the person feels responsible.
4. Responsibility increases after they have checked for safety.

IMPLICATIONS FOR TREATMENT

It follows from the cognitive-behavioral models described above that OCD symptoms can be successfully reduced if patients (*a*) change the way they interpret their

intrusive cognitions as threatening and (*b*) cease the performance of neutralizing behaviors (including deliberate mental acts) that serve to maintain the faulty interpretations. Thus, cognitive-behavior therapy (CBT) assumes the task of helping patients normalize their experiences leading to a better understanding of the nature of their problem as one involving thinking and deciding to act, as opposed to the risks and uncertainty they fear (Salkovskis et al., 2000). Patients with compulsive checking symptoms are helped to view their problem not as one in which they might be responsible for causing harm, but instead as a problem in how they attach undue significance to, and deal with, intrusive thoughts about harm. Similarly, those who wash and clean compulsively are helped to see that their is a problem with why they *fear* contamination and illness, rather than that they *may be at risk* of becoming ill.

Salkovskis et al. (2000) outlined five components of CBT, including:

1. Collaborative development of a comprehensive and individualized explanation of the cognitive-behavioral factors underlying the patient's OCD symptoms as well as a new, nonthreatening explanation.
2. Procedures to help patients recognize, challenge, and modify dysfunctional appraisals of their intrusive thoughts.
3. Discussion techniques for challenging faulty appraisals and the flawed basic assumptions on which such appraisals are based.
4. Experiments, such as ERP exercises, to test the validity of dysfunctional appraisals against the new, nonthreatening explanation of OCD.
5. Identification and modification of underlying general assumptions that give rise to catastrophic or responsibility misinterpretations of intrusive thoughts. This helps the patient to view their problem as the result of trying to hard to control otherwise innocuous cognitive intrusions, rather than perceiving the intrusive thoughts as a sign that they are out of control.

Empirical Support for CBT

Behavior therapy using procedures derived from the classical behavioral model of OCD (ie, ERP) has undergone extensive evaluation in numerous treatment centers around the world. The substantial beneficial effects of this treatment package are among the most consistent findings in the mental health treatment outcome literature to date (Franklin & Foa, 2002). Nevertheless, because ERP entails confronting fear-evoking situations and abstaining from safety-seeking compulsive behavior, a somewhat sizeable proportion of patients refuse to begin this treatment or discontinue therapy prematurely.

Recent research has examined the effects of treatment derived from the cognitive-behavioral models described above. Most of the newer treatment packages incorporate ERP procedures to some extent, yet ERP is practiced within the context of modifying cognitions as opposed to extinguishing classically conditioned anxiety. A controlled study on the treatment of OCD patients presenting with obsessions without overt compulsions ($N = 29$) was conducted by Freeston and colleagues (Freeston et al., 1997). Fifteen of the patients received cognitive therapy designed to change the misinterpretation of intrusive thoughts and the remainder formed a wait-list control group. Patients receiving cognitive therapy improved substantially more than did those in the wait-list group; and this improvement was clinically significant. Moreover,

treatment gains were maintained at 6 month follow-up. These results are consistent with the prediction that changing the personal significance of obsessional thoughts decreases covert neutralization, the frequency and intensity of intrusive thoughts, and ultimately eliminates obsessions (Rachman, 2003).

Whether catastrophic misinterpretations of intrusive thoughts are best modified with cognitive therapy procedures or ERP is not clear since a study by McLean et al. (2001) found that these treatments were equally effective in producing changes in the cognitive biases presumed to underlie OCD. Findings from other randomized controlled trials (eg, Rassin et al., 2001a) are also consistent with the view that behavioral treatments may effect cognitive change (Rassin et al., 2001a, 2001b).

In a small randomized controlled trial of 21 OCD patients with primarily contamination and washing symptoms, Jones and Menzies (1998b) found that compared to a waiting list, treatment procedures focused on danger expectancies led to reductions in OCD symptoms. The intervention was solely directed at decreasing danger-related expectancies concerning contamination and did not include exposure, response prevention, or behavioral experiments. Instead, components of this treatment package (entitled Danger Ideation Reduction Therapy, or "DIRT") included attentional focusing, filmed interviews, corrective information, cognitive restructuring, expert testimony, microbiological experiments, and a probability of catastrophe assessment task.

Taken together, the results of treatment studies evaluating interventions derived from cognitive-behavioral models of OCD are encouraging. In a meta-analysis including only randomized controlled trials of ERP and CBT, Abramowitz, Franklin, and Foa (2002) found an effect size of 1.50 across eight studies of ERP, 1.19 across two studies of cognitive-therapy, and 0.99 across three studies in which ERP and cognitive techniques were combined.

KEY COGNITIVE BIASES VERSUS DISEASE PROCESSES

A significant element of the behavioral and cognitive-behavioral models described above is that they provide a clearly articulated, theoretically sound, and empirically supported account for the symptoms of OCD that is derived from normal learning principles (ie, conditioning) and normal (yet biased) cognitive processes. Moreover, treatment interventions derived from these models that attempt to modify these psychological processes work well in reducing OCD symptoms. This idea represents a contrast with biological and genetic models that attempt to explain OCD in terms of structural and/or functional deficits or abnormalities located in neurotransmitter systems or in the brain itself. It is not difficult to understand the appeal of such disease models when it comes to OCD given that the most observable aspects of this disorder are the seemingly uncontrollable and bizarre thinking and behavior. Nevertheless, results from research on serotonin dysfunction in OCD are largely equivocal and thus not convincing (eg, Gross, Sasson, Chopra, & Zohar, 1998). The most consistent finding in the biological literature to date is that OCD symptoms respond preferentially to serotonin reuptake inhibitor (SRI) medication as opposed to medications with other mechanisms of action. However, from an epistemological standpoint, successful treatment outcome data can not provide a basis for evaluating the validity of etiological theories since they do not provide

information about the mechanisms involved in the development of a particular disorder (Salkovskis, 2002).

This is not to say that biology is not, at some level, involved in OCD—indeed all mental and behavioral processes have biological substrates. However, the assumption that neurobiological processes function normally, as in the cognitive-behavioral models, is very different from the supposition of a neurobiological *disease*. Neuroimaging studies collectively indicate that brain regions such as the caudate nucleus and orbital gyrus are in some sense involved in OCD (Whiteside, Port, & Abramowitz, 2003). Nevertheless, the designs of existing studies do not permit one to conclude that (*a*) differences between patients and controls represent *abnormalities* in functioning or (*b*) the differences are related to the *cause* of OCD. In order to infer causation, controlled experimental studies would need to be conducted in which brain activity in certain areas of is manipulated, resulting in the development of OCD symptoms. In the absence of such experimental manipulations in the neurobiological literature, statements regarding OCD and neuroimaging findings should be limited to the conclusions allowed by correlational data. Specifically, there are three possible explanations for the current findings: (1) alterations in functioning in certain brain regions cause OCD; (2) OCD causes alterations in functioning as observed in certain brain regions; or most likely, (3) a third variable causes both phenomena.

Interestingly, studies using the symptom provocation paradigm, an experimental paradigm in which imaging procedures are employed while patients' obsessional fears are evoked, find that when symptoms are provoked, radiotracer uptake is altered in the brain regions of interest (eg, Zohar et al., 1989). Although these studies are often cited as supporting the theory that OCD is caused by a brain dysfunction (eg, Saxena, Boto, & Brody, 2001), when considered carefully, they actually provide compelling experimental evidence for the alternative proposition: that OCD symptoms cause alterations in radiotracer uptake. Moreover, similar results have been found in other anxiety disorders such as social phobia; thus, the appropriate conclusion from provocation studies is that differences between patients and nonpatients are a product of the presence of anxiety in general, rather than OCD in particular (Cohen et al., 1996).

Cognitive-behavioral models, on the other hand, provide a more parsimonious (and therefore preferable) explanation of the development and maintenance of OCD that does not appeal to as yet unsubstantiated claims of disease states. It is assumed that obsessional problems develop from normal cognitive intrusions that are misinterpreted in biased ways (eg, TAF) as threatening or otherwise unacceptable. This leads, understandably, to high levels of anxiety and a number of natural sequelae. First, anxiety normally produces preoccupation with the perceived threat. Thus, people with OCD become engrossed with their intrusive obsessional thoughts and with attempts to control such thoughts which they feel are unacceptable. Failure to gain control leads to negative mood states and more negative thinking. Second, anxiety motivates efforts to reduce distress via escape or avoidance, thus patients resort to compulsive and neutralizing behavior (including mental behavior) to accomplish this. Unfortunately, because such behavior results in the short-term reduction of distress, it is reinforced and develops considerable strength. In the long-term, compulsions and neutralizing are likely to have the effect of preventing the otherwise normal reduction in anxiety. Salkovskis (1996) has likened this "normalizing" cognitive-behavioral approach to that developed to explain the persistence of panic disorder (eg, Clark, 1986).

CONCLUSIONS

In conclusion, this chapter has described a range of cognitive-behavioral approaches to obsessional compulsive complaints. There is a great deal of empirical support for them but critical questions remain. First, even if these models help understand the phenomenology of OCD and indicate that such cognitive constructs should be addressed, what are the best methods of tackling them? If ERP produces as much cognitive change as CBT, should the latter intervention be advocated since it is arguably harder to implement? Or should CBT be given only to people that refuse ERP? Second, what exactly is the nature of the relationship among these (and other) cognitive constructs? Are some of these constructs more critical than others? Finally, how can we link the cognitive-behavioral theories and treatments to progress in neuroscience? A harmonious relationship between the different approaches is desirable so that they may inform each other rather than be placed in opposition. Such a relationship would facilitate the goal of achieving a comprehensive understanding of the nature of OCD so that we can develop increasingly effective and palatable interventions for our patients.

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Reply to Shafran:

BIOLOGICAL AND COGNITIVE MODELS OF OCD: SEEKING SIMILARITIES AND ACHIEVING PROGRESS TOGETHER

David R. Rosenberg, Aileen Russell, and Andrea Fougere

In her chapter, Dr Shafran outlines a series of closely related cognitive-behavioral models that account quite well for the symptoms of OCD. These coherent formulations have been subjected to empirical scrutiny using a variety of research designs, and the data from these studies is largely consistent with theory. Dr Shafran also eloquently describes how cognitive-behavioral therapy (CBT), a highly effective treatment for many OCD patients, is logically and empirically derived from the cognitive-behavioral models she presents. The main objective of CBT is the normalization, rather than the abolition, of intrusive obsessional thoughts. Moreover, therapy works by modifying beliefs that lead to the misinterpretation of obsessional stimuli as dangerous or that lead to a heightened sense of responsibility.

At first glance it might seem that there are substantial differences between neurobiological and cognitive-behavioral models of OCD. For example, whereas neurobiological models call attention to neuroanatomical and neurochemical dysfunctions, cognitive-behavioral models accentuate the role of normal (albeit biased) thinking and learning (conditioning) processes. However, we believe that these two theories differ primarily in their emphasis, and that important points of convergence between them should not be overlooked. For example, there are a number of explanations for the observed neuroanatomical alterations in OCD, including that they are epiphenomena and reflect the presence of OCD symptoms (ie, are epiphenomena), or that they denote a compensatory response to illness. Moreover, changes in neurobiological variables have been observed following successful CBT, suggesting that the modification of cognition and behavior can alter neurobioloigic functioning. Thus, the neurobiological model is not necessarily inconsistent with cognitive-behavioral accounts of the development of OCD symptoms.

At present very little is known about the association of neurobiological and cognitive markers including moderator (present at baseline) and mediator (changing with treatment) variables and their interaction with the pathophysiology and

disease-specific treatment in OCD. Application of sophisticated neuroimaging techniques along with comprehensive clinical, cognitive, and neurobehavioral characterization of patients with OCD may lead to an enhanced understanding of the pathogenesis of the illness and potentially to improved diagnosis and treatment. Studying patients closer to illness onset unconfounded by treatment effects is important, as are longitudinal studies of patients before and after treatment, to tease apart illness-related brain changes, "scar effects," compensatory effects of illness, and medication/therapy effects.

Cognitive and neuroimaging models, while on the surface different, actually converge in joined models that connect disorder, cognitive profile, clinical neuroscience, treatment components, and treatment development. Used together, the two approaches have the potential to increase our knowledge of the developmental basis of OCD and help us understand which patterns (brain, cognitive, psychosocial) might best define a particular subytpe of OCD and its response to a particular treatment (eg, medication, cognitive-behavioral therapy, or their combination).

In this way, there are parallels with the case of understanding epilepsy, which prior to precise neurodiagnostic assessment with the electroencephalogram was considered more of a psychiatric than a neurologic condition. The advent of the electroencephalogram and more precise neurodiagnostic assessment not only helped facilitate diagnosis of specific type of epilepsy, but based on the assessment could help better determine treatment. The electroencephalogram does not *replace* effective clinical and cognitive assessment, but each complement each other in terms of better defining the condition. Given the heterogeneity of OCD, there ultimately may be several models or subtypes that best explain particular phenotypic expressions of illness. Thus, disparate but converging models for elucidation of the underlying pathogenesis and mechanisms of response to treatment are critical.

Reply to Rosenberg et al.:

BIOLOGICAL VERSUS PSYCHOLOGICAL APPROACHES TO OCD: WAR OR PEACE?

Roz Shafran and Anne Speckens

Rosenberg and colleagues provide an excellent review of different brain areas and neurotransmitters that might be implicated in the development of OCD. They hope that the neuroanatomical and neurochemical abnormalities that were found in patients with OCD will assist in more narrowly defining the phenotype of OCD and help identify which patients might benefit from treatment with selective SRIs and cognitive behavioral therapy (CBT). Specifically, the chapter opens by suggesting that combining unique assessment/treatment and neuroimaging/genetic expertise at specific performance sites has already resulted in substantial progress in understanding the brain mechanisms that may be involved in treatment response (or lack thereof). Their chapter concludes by restating that in their view, the most clinically relevant question is to determine which treatments are best for which patients. Their view is that this important clinical question is best answered by examining relevant biomarkers.

We certainly agree that the question of "what works for whom" is important, but whether a neuropsychiatric approach is going to be the most helpful way to tackle it is debatable. To help address this question, we focus on four main issues raised by the chapter. First, why are the findings in neuropsychiatric research so inconsistent? Second, has the neuropsychiatric research really helped us understand factors that *cause* OCD or has it rather provided information about neurological *correlates* of the disorder? Third, what sort of information is likely to yield the prognostic information that is so clinically valuable? Fourth, as research becomes increasingly neurological, is there a danger of underemphasizing the clinical phenomena that need to be addressed? We go on to examine differences between the psychological and biological approaches to OCD and explore whether the two accounts can be integrated.

INCONSISTENCIES

The neuropsychiatric literature appears to be full of inconsistencies. Bilateral *reduction* in basal ganglia volume has been observed in OCD patients compared to

controls in some studies (eg, Rosenberg et al., 1997), yet others have found *increased* basal ganglia volumes in patients with OCD (Giedd, Rapoport, Garvey, Perlmutter, & Swedo, 2000). OCD symptom severity has been associated with both higher (Swedo et al., 1992) and lower pretreatment CSF 5-HIAA levels (Asberg, Thoren, & Bertilsson, 1982), and there are also reports of no differences in blood and CSF serotonin measures in OCD patients (eg, Pandey, Kim, Davis, & Pandey, 1993). mCPP symptom provocation has been reported to exacerbate OCD symptoms and to be associated with a global increase in cortical blood flow (Hollander, Prohovnik, & Stein, 1995), but the opposite has also been reported (Ho Pian, Westenberg, Den Boer, de Bruin, & van Rijk, 1998). How is one to make sense of this?

The inconsistent findings of neuropsychiatric research might be partly due to huge variability in study designs. Study populations are different in terms of diagnosis (OCD, anxiety disorders in general, depression, schizophrenia), age (children/adolescents, adults), and treatment history. Neuroimaging studies have involved structural or functional MRI, positron emission tomography, or proton magnetic resonance spectroscopy. Some of these use symptom provocation paradigms or specific tasks, others do not. Neurochemical studies examine different substances (serotonin transporter protein, 5-HIAA), in different media (blood, cerebrospinal fluid) or use pharmacological challenge tests. In studies looking at changes before and after treatment, interventions might have involved different psychopharmacological agents and different psychological treatments. In addition, it is not always clear how treatment outcome has been assessed: which symptoms improve exactly? And last but not least, the number of patients included in the studies is often very small.

It is clear that before we can make any progress in identifying biomarkers of different subtypes of OCD or different outcomes with SRI or psychological treatment, we need more specific and consistent study populations, methodology, interventions, and larger samples.

CAUSE VERSUS CORRELATE

Rosenberg and colleagues describe a large number of studies. Some of these compare neuroanatomical and neurochemical findings in people with and without OCD (eg, Szeszko et al., 1999), some correlate them with symptom severity (eg, Rauch et al., 1994), and some examine pre- and post-treatment changes in such markers (Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996). Rosenberg et al. interpret such findings as providing information about the etiology of OCD (eg, that disturbances in basal ganglia–frontal cortical interactions and feedback loops may play a critical role in the pathogenesis of OCD). However, the results from these studies are consistent with the explanation that changes in such markers are a *consequence* of the symptoms or change in symptoms, but are not necessarily a *cause* of the symptoms. For example, it is not surprising that there is a range of biological differences between people with OCD and healthy controls; but this cannot be assumed to mean that such a difference is the *cause* of OCD or that it demonstrates *abnormal* brain functioning. To establish a causal role one would have to induce such biological changes and observe an increase in OCD symptoms. To our knowledge this has not yet been done.

Unfortunately for the neuropsychiatric model, research suggests that differences between OCD patients and healthy individuals probably represent biologically

functional (ie, normal) responses to excessive anxiety. For example, Mataix-Cols et al. (2003) demonstrated that the provocation of different types of obsessive-compulsive symptoms in healthy controls activated similar brain areas as in patients with OCD. Similarly, the finding that comparable reductions in caudate glucose metabolism have been observed following successful CBT or pharmacotherapy with SRIs in OCD patients (Schwartz et al., 1996) suggests that no matter how symptom change is achieved, such change is reflected at a neurological level. These are open questions but the possibility that such biological changes are epiphenomena must be considered in light of the available correlational data.

PREDICTORS OF OUTCOME

Rosenberg et al. suggest two different biomarkers to target the question of whether specific patterns of pretreatment brain activity can indicate which treatment (eg, CBT or SRIs) will be most effective for patients. One of the examples given is that decreased orbital frontal-hemispheric metabolic rates and increased activity in cingulate cortex predicted better response of OCD patients to SRIs, while increased orbital frontal-hemispheric metabolic ratios pre-treatment predicted enhanced response to CBT (Brody et al., 1998). The other example they provide is that caudate glutamatergic concentration decreased with SRI treatment, whereas no significant changes in caudate glutamate were observed before and after CBT (Benazon, Moore, & Rosenberg, 2003). Although the examples provided give an indication of some prognostic significance of these biological markers, correlations or differences in means do not necessarily mean that these markers have a high positive or negative predictive value with regard to treatment outcome. The identification of patient characteristics that usefully and consistently predict the outcome of different types of treatment is a long-sought but somewhat elusive goal in psychiatric research and requires multiple, large-scale randomized controlled studies with standardized treatment protocols (Agras et al., 2000). It might be more useful to concentrate such efforts on patient characteristics that are readily available (such as age of onset, duration of disorder, type of symptoms, psychiatric comorbidity), rather than results of neuroanatomical or neurochemical investigations.

CLINICAL PHENOMENA

Finally, Rosenberg et al. describe studies of animal models of OCD and compulsive behavior (eg, Cummings, 1993) and integrate this work with research on humans. Lesions that result in neuropsychological and neurobehavioral disturbances presumed to be comparable to patients with OCD, and research using mice which apparently exhibit a phenotype resembling OCD and comorbid Tourette's syndrome (eg, Nordstrom & Burton, 2002) is specifically discussed.

While animal studies have their place in psychiatric research, the hallmarks of OCD are (*a*) unwanted intrusive thoughts that are repugnant to the individual and highly resisted; and (*b*) compulsive behaviors that are motivated by a desire to reduce anxiety or danger (American Psychiatric Association, 1994). It is easy to see how any repetitive behaviors can become labeled as "compulsive," but by focusing merely on

repetitiveness, there is a significant danger of failing to appreciate the complexity of the behavior observed in people with OCD. Critically, a nosology that fails to differentiate between a range of repetitive behaviors with a variety of motivations may negatively impact on the development of effective treatments. For example, patients with OCD and Tourettes Syndrome appear to be less responsive to SRIs than are patients with OCD alone (eg, McDougle et al., 1994), and such findings have led to the view that tic-related and non-tic related OCD may be different subtypes of the disorder (see Riddle, 1998). It is our view that grouping a variety of behaviors together under a generic category of "compulsions" without careful consideration of the clinical features (eg, whether the behavior is performed in response to obsessions) is not likely to advance either our understanding or treatment of OCD.

Rosenberg et al.'s suggestion that neuroimaging measurements could provide endophenotypes for genetic investigations of OCD that may be more homogeneous than clinical phenotypes raises comparable concerns. The further one moves away from the clinical phenomena that need to be addressed, the less likely that the approach will result in clinically useful advances. In our view, neuropsychiatric research could benefit from looking at the phenomenology of OCD in as much detail as possible. For example, there is evidence that different symptom dimensions in OCD might be associated with activation of different brain areas (Mataix-Cols et al., in press). There is also a wealth of information about psychological or neuropsychological factors that might be relevant in the development of OCD, waiting to be incorporated in neuropsychiatric studies. In this way, we agree with Rosenberg et al. about the importance of making linkages across a range of disciplines and between scientists and practitioners (Hoagwood & Olin, 2002).

COGNITIVE-BEHAVIORAL VERSUS NEUROPSYCHIATRIC MODELS

In their chapter, Rosenberg and colleagues echo Hyman's (2002) suggestion that medication and psychosocial treatment can be used to test neurodevelopmental models and, by inference, embrace the science-based trend towards conceptualizing mental illness within a medical framework. Aside from the logical difficulties with using treatment response data to inform theoretical models (see Chapter 13 and Salkovskis, 2002 for a discussion of this issue), the implication of this argument is that a medical framework—a disease-based approach—is both scientific and optimal. We argue that the cognitive-behavioral model, which is empirically grounded yet does *not* place OCD within a disease-based framework, offers advantages over diseased-based medical models. The cognitive-behavioral model begins with the finding that unwanted intrusive thoughts are normal occurrences (Rachman & de Silva, 1978), and it is the appraisal of such thoughts that distinguishes people with OCD from those without. Thus, the fundamental difference between the two approaches is that one is disease-based and the other is not. Since the nonlesion based model (ie, the cognitive-behavioral model) can account for the phenomenology of OCD without resorting to a "diseased brain," it is more parsimonious and therefore preferable to neuropsychiatric (diseased-based) models. There is also the important point that none of the neuropsychiatric models has yet to lead to treatment advances, rather than vice-versa. On the other hand, the behavioral model of OCD resulted directly in the development of a

highly effective therapeutic program for OCD, namely ERP (Rachman & Hodgson, 1980).

Can the biological and psychological approaches be integrated despite such a fundamental difference in their foundations? Is "peace" preferable to "war?" Arguably, the answer to this question is "yes" on both theoretical and practical grounds. Theoretically, it is clear that OCD is a multifactorial disorder and that genetic, biological, and psychological factors all interact and contribute to the disorder. Genetic and biological factors likely make some individuals vulnerable to developing OCD. And although neuropsychiatric findings do not always provide evidence of the etiology of OCD, they could improve our understanding of what happens in the brain when people have obsessive-compulsive symptoms, or what happens when their symptoms change with treatment. This knowledge might be used to improve therapeutic interventions, whether pharmacological or psychological. Practically, it is possible that if biological mediators of OCD are addressed by whatever mechanism (eg, medication), then this might enable patients to engage in CBT and to benefit from it, whereas the intensity of the symptoms might have prevented patients from embarking on any psychosocial intervention. Ideally, large-scale randomized controlled trials similar to those conducted with patients with panic disorder (Barlow, Gorman, Shear, & Woods, 2000) must be conducted to determine the implications of combining such interventions.

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Part III

TREATMENT

Chapter 14

FORMAL COGNITIVE THERAPY: A NEW TREATMENT FOR OCD

Jeanne Fama and Sabine Wilhelm

DEVELOPMENT OF THE COGNITIVE MODEL OF OCD

The occurrence of persistent, intrusive, anxiety-provoking thoughts (obsessions) is one of the defining features of obsessive-compulsive disorder (OCD) (American Psychiatric Association, 1994). Research has also shown that most healthy people occasionally experience transient intrusive thoughts (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984). Unlike those with OCD, however, healthy individuals can dismiss such thoughts and do not experience them as particularly distressing. Such observations led researchers to hypothesize that it is not the experience of intrusive thoughts per se that is pathological; but rather the way in which patients interpret such thoughts that leads to the pathological anxiety, depression, and guilt that is characteristic of OCD (eg, Freeston, Rhéaume, & Ladouceur, 1996; Rachman, 1993, 1997; Salkovskis, 1985, 1989; Wilhelm, 2000).

On the basis of this conceptualization, theorists drew from Beck's (1976) cognitive theory to develop models delineating the mechanisms through which intrusive thoughts could lead to obsessional anxiety and ritualizing. For example, Salkovskis (1985) suggested that distorted beliefs about personal responsibility for preventing harm was the primary mechanism through which intrusive thoughts generate anxiety in OCD patients. Accordingly, an intrusive thought about one's home burning down, which would be simply dismissed by someone without OCD, activates beliefs about perceived irresponsibility and, hence, great anxiety in someone with OCD who feels an inordinate sense of responsibility for preventing feared catastrophic outcomes. This would then lead to compulsive behavior (eg, returning to home to check the stove). Salkovskis (1985) further suggested that cognitive therapy (CT) techniques to modify dysfunctional beliefs about responsibility might augment behavioral therapy (BT) by exposure and response prevention (ERP), the most empirically supported psychotherapy for OCD. In particular, he proposed that cognitive techniques would be especially helpful in cases where OCD was accompanied by severe depression or overvalued ideation, and when fears could not be easily incorporated into exposure practice.

Drawing on a growing body of clinical and empirical literature on cognitive distortions in OCD, the Obsessive Compulsive Cognitions Working Group (OCCWG, 1997) outlined three cognitive levels of analysis in need of study in OCD: (1) intrusions, that is, unwanted thoughts, impulses, or images; (2) appraisals, that is, meaning given to a specific event such as an intrusion; and (3) beliefs, that is, relatively enduring pan-situational assumptions often held by OCD patients. Moreover, based on empirical research and expert consensus, the OCCWG outlined six domains of beliefs thought to be relevant in the development and maintenance of OCD. A brief description of each domain follows.

Inflated Sense of Personal Responsibility

As noted above, patients with OCD often mistakenly regard themselves as responsible for preventing harm they associate with intrusive thoughts. Inflated responsibility takes two forms: (*a*) responsibility for errors of *commission* (eg, excessive responsibility for having an intrusive aggressive thought they fear will lead to committing an aggressive act) and (*b*) responsibility for errors of *omission* (eg, responsibility for not taking every step possible to ensure the well-being of others, even those for whom they are clearly not responsible; eg, Salkovskis, Richards, & Forrester, 1995).

Overimportance of Thoughts

People with OCD often believe that the mere presence of a thought implies its importance. Additionally, they may report distorted beliefs about the fusion of thoughts with actions (ie, "thought action fusion" [TAF]; Rachman, 1993). Thought action fusion may occur in at least two forms. Moral TAF refers to the belief that thinking about negative events (eg, molesting a child) is almost as unacceptable as carrying out such behavior. Likelihood TAF refers to the mistaken idea that having thoughts about a negative event increases the likelihood that the event will occur. Hence, intrusions can cause great anxiety in OCD patients who mistakenly believe that such thoughts reveal something negative about their inner nature (eg, that they are dangerous individuals likely to harm others).

Need to Control Thoughts

Dysfunctional beliefs about the overimportance of thoughts are accompanied by maladaptive beliefs about one's thought processes (ie, metacognition). Given the significance OCD patients attribute to intrusive thoughts, they are often engaged in a struggle to control their thoughts. Attempts to control one's thinking are based on the mistaken belief that people can and should exert complete control over their thought processes. Although it is nearly impossible to prevent the occurrence of intrusive thoughts (Wegner, 1989), OCD patients do not realize this, and therefore experience great anxiety and guilt when they are unable to gain such control.

Overestimation of Threat

Many researchers (eg, Carr, 1974; Foa & Kozak, 1986; OCCWG, 1997; Salkovskis, 1985) have recognized how OCD patients mistakenly overestimate the risk of negative

outcomes. Additionally, individuals with OCD often believe that the consequences associated with feared negative events will be much graver than is probable. Unlike those without OCD, OCD patients often presume a situation is dangerous until otherwise proven safe (Foa & Kozak, 1986). Hence, patients' mistaken beliefs about pending threat often lead to great anxiety and rituals performed to reduce the perceived threat.

Intolerance of Uncertainty

Distorted beliefs about uncertainty contribute to the anxiety and indecisiveness characteristic of OCD (eg, Guidano & Liotti, 1983; Kozak, Foa, & McCarthy, 1988). Sookman, Pinard, and Beck (2001) suggest that patients' distorted beliefs about the need for certainty are related to their perceived inability to cope with ambiguity, newness, and unpredictable change. Because it is usually impossible to achieve an absolute guarantee of safety, the mistaken belief that complete certainty must be achieved to prevent harm can lead to virtually unremitting anxiety in OCD patients.

Perfectionism

Several writers (eg, Frost & Steketee, 1997; McFall & Wollersheim, 1979; Rasmussen & Eisen, 1989) have underscored the potential etiological significance of perfectionism in OCD. The mistaken belief that one can and should attain perfection can lead to excessive anxiety about the inability to think or act "perfectly" and, likely, to rituals aimed at achieving perfect states (eg, re-reading in order to understanding something perfectly, repetitive cleaning to achieve perfect cleanliness). Not surprisingly, researchers have demonstrated associations between perfectionism and OCD checking (Gershuny & Sher, 1995) and washing (Tallis, 1996) symptoms.

TECHNIQUES USED IN COGNITIVE THERAPY FOR OCD

Empirical research on OCD-specific beliefs has led to refinement of cognitive theories of OCD. This work has been paralleled by the development of CT protocols incorporating techniques to help patients challenge and modify the dysfunctional beliefs that maintain OCD symptoms (eg, Freeston et al., 1996; Freeston & Ladouceur, 1997; van Oppen & Arntz, 1994; Wilhelm, 2000, 2001, 2003; Wilhelm & Steketee, 2002). Some examples of CT techniques found especially effective are briefly described below. General techniques are listed first, followed by OCD-belief domain-specific techniques.

General Techniques

Explaining the Cognitive Model

Therapists explain that psychological distress (eg, anxiety, guilt) does not result from intrusive thoughts or certain situations per se, but rather from how the individual appraises and responds to such stimuli (Beck, 1976). Patients learn how dysfunctional appraisals and beliefs lead to negative emotional responses, and to behavioral patterns, (eg, compulsions, avoidance) intended to neutralize anxiety or guilt. Finally,

patients learn about the role of rituals and avoidance in the maintenance of OCD (eg, Salkovskis, 1985).

Socratic Questioning

Using Socratic dialogue, therapists assist patients to systematically examine the logic that underlies their mistaken beliefs. Therapists offer logical corollaries to patients' flawed logic, play devil's advocate, and ask questions such as: "How useful is that thought right now?"; "What is the evidence that supports that thought?"; "Is there any evidence that refutes that thought?"

Downward Arrow Technique

Therapists repetitively (but gently) query patients about the meaning they ascribe to their own intrusive thoughts (eg, "What does it mean that you had that thought/image?"), increasing patients' awareness of their distorted appraisals of unwanted thoughts. Further questioning (eg, "And what would that mean?") helps OCD patients identify dysfunctional beliefs associated with intrusions and appraisals (eg, Wilhelm, 2001, 2003).

Identification of Cognitive Errors

Patients are shown a list of common cognitive errors (eg, *catastrophizing;* always expecting the worst outcome), asked to identify which errors they engage in when interpreting stimuli and situations, and helped to generate alternative interpretations.

Mindfulness Skills and Use of Metaphor

Patients are asked to view stimuli and situations from alternative, less threatening perspectives. For example, instead of viewing intrusions as threatening thoughts that need to be controlled, they can be conceptualized as thoughts that patients can let float through their minds naturally, like clouds passing by or like leaves floating down a river.

The techniques listed below are described in detail in multiple CT protocols (eg, Freeston et al., 1997; Freeston & Ladouceur, 1997; Steketee, 1999; van Oppen et al., 1995; van Oppen & Arntz, 1994; Wilhelm, 2000, 2001, 2003; Wilhelm & Steketee, 2002). They are grouped according to belief domains in which they are frequently used but can often be used in other beliefs domains.

OCD Belief Domain-Specific Techniques

Techniques for Reducing Exaggerated Estimates of Responsibility

Courtroom Role Play. Patients and therapist engage in a role play in which patients act as prosecuting attorneys and therapists act as judges or defense attorneys. Patients present arguments in an attempt to prove their own guilt for causing a feared consequence. Therapists present arguments demonstrating that the patients' "evidence" (eg, they *feel* guilty—without any evidence of wrongdoing) would never hold up in court. This helps illustrate to patients how they overestimate their personal responsibility for the feared consequences.

Pie Technique. Patients identify a feared consequence (eg, "I unknowingly drop a coin and a child picks it up, puts in his mouth, and chokes to death on it") and give an initial (gut level) estimate of the percent responsibility that would be attributable to them if this consequence were to occur. Patients then generate a list of the parties (other than themselves) that would have some responsibility for the feared consequence (eg, the child, the parent supervising the child, the child's medical team, etc). They then draw a pie chart, each slice of which represents one of the responsible parties identified. Next, patients label all parties' slices according to their percent responsibility (eg, the parent—50% responsibility, the child's medical team—25%, etc) and label their own slice last. By the exercise's end, it is generally clear to patients that the majority of the responsibility for the feared event would not be their own.

Techniques for Reducing the Overimportance of Thoughts

Psychoeducation to Normalize Intrusions. To help patients view their experiences with unpleasant intrusive thoughts as normal and universal, therapists can give patients a list of intrusive thoughts reported by individuals who do *not* have OCD (eg, Wilhelm & Steketee, 2002). This enables patients to see how their own intrusive thoughts (eg, thoughts of harming loved ones) are similar in content to those reported by others without OCD. Patients often report feeling relieved when they realize that intrusions are experienced universally and not an indication of depravity or "craziness." Furthermore, the realization that most people experience these intrusions *without* associated anxiety can underscore the importance of therapeutically addressing *beliefs and interpretations about intrusions* rather than trying to eliminate the intrusions per se.

Cognitive Continuum. This technique assists patients with discriminating between the presence of an intrusion and the commission of a negative act. As such, it can be especially effective in modifying Moral TAF. Using a visual analog scale from 0 (most moral person ever) to 100 (most immoral person ever), patients rate how immoral they are for having intrusive thoughts. Next, patients rate the morality level of other individuals who have committed acts of varying degrees of immorality (eg, a serial rapist, abusive parents). Then, patients re-rate themselves and reevaluate how immoral they are for simply experiencing intrusive thoughts.

Behavioral Experiments. Patients conduct "experiments" to evaluate the accuracy of their predictions regarding obsessional fears. In contrast to ERP exercises, which are conducted for extended time periods and intended to increase patients' anxiety in order that they may experience habituation to feared stimuli, behavioral experiments in CT are usually brief, and used only to test patients' maladaptive predictions against other, more rational predictions. For example, a patient who believes that thinking about a negative event will lead to committing the corresponding action might be given a fragile object to hold and instructed to think about purposely breaking the object. Next, the therapist would review with the patient whether the original hypothesis (eg, my thoughts will make me break the object) was supported (eg, "Is the object still intact?" "Was there any attempt to break it?"). Ultimately, patients come to realize that merely thinking about something does not lead to the occurrence of the corresponding event.

Techniques for Reducing the Need to Control Thoughts

Thought Suppression Test. Adapted from Wegner's (1989) work demonstrating that attempts to suppress thoughts paradoxically lead to an *increase* in their frequency, this technique demonstrates that people are unable to control their thoughts and, moreover, that paradoxical effects result when people attempt to suppress target thoughts. Patients are instructed to think of an animal (eg, a giraffe) as frequently as they can, and record the number of times they experience a thought about the animal within a given time frame (eg, 1 min). Next, they are instructed *not* to think about the animal for 1 min, but to record the number of times the thought intrusively comes to mind. In most cases, patients report a greater number of animal thoughts while trying to suppress, compared to when trying to generate such thoughts. This demonstrates how attempting to suppress distressing obsessional thoughts (an oft-utilized strategy for OCD patients) is counterproductive.

Techniques for Reducing Overestimates of Danger

Calculating Probability Estimates. First, patients are asked to provide an estimate of the probability that a feared consequence (eg, leaving a lamp on and burning the house down) will occur. Next, patients make a list of the chain of individual events that would need to occur in order for their feared consequence to occur (eg, lamp overheats, lamp shade comes in contact with lightbulb, etc). Then, patients estimate the probability of the occurrence of *each* individual event listed in the chain. Then, using a calculator, patients calculate the cumulative probability of the ultimate, feared consequence (ie, the house burning down) by multiplying the probabilities of each individual event in the chain. Finally, patients compare their previously estimated "gut level" probability with the mathematically derived cumulative probability (which is usually a great deal lower), demonstrating how dramatically they overestimate the likelihood of danger.

Techniques for Reducing the Intolerance of Uncertainty

The "Advantages and Disadvantages" Technique. Once patients identify the various distorted beliefs that underlie their fears of intrusive thoughts, they make a list of the advantages and the disadvantages of maintaining what they now recognize as distorted beliefs. Table 14.1 provides an example of such a list (based on Freeston

TABLE 14.1. An example of advantages and disadvantages of trying to achieve perfect certainty

Advantages	Disadvantages
Knowing/being certain about a few things	Anxiety/self-doubt/frustration when I am not certain Preoccupation and distraction that can impair attention and memory Loss of interest or pleasure in reading things or participating in hobbies for fear that one will not be certain of them afterwards Family/friends becoming frustrated with my repetitive checking and reassurance-seeking

et al., 1996). This exercise can help point out how the advantages of being certain about a few things are usually far outweighed by the disadvantages of maintaining distorted, uncertainty-related beliefs that perpetuate significant anxiety and impairment.

Techniques for Reducing Perfectionistic Beliefs

Perspective Taking. Patients are asked whether they expect others (eg, their children, friends, etc) to be perfect, and whether they have contempt for those who make minor mistakes (eg, in a letter or a homework assignment). This is usually followed by a discussion about why patients believe it is OK for others, but not themselves, to make such mistakes.

Advantages and Disadvantages. As described above, this technique may also be effective in modifying beliefs about the importance of being perfect. By listing the advantages and disadvantages of trying to attain perfection, patients often come to realize that the drawbacks of their perfectionism (eg, loss of time, procrastination, loss of enjoyment, academic or occupational impairment) far outweigh the benefits of trying to make things perfect.

Summary

The above techniques are derived from a comprehensive cognitive model of OCD based on over two decades of clinical observations, conceptual analysis, and empirical research. This model suggests that when intrusive thoughts are followed by distorted appraisals and beliefs (which usually fall in one of the domains identified by the OCCWG), anxiety and compensatory compulsive and avoidance behaviors characteristic of OCD will ensue. Treatment strategies based on this model (ie, CT) have evolved to target the cognitive mechanisms hypothesized to generate and maintain OCD symptoms. Although empirical study on the effectiveness of these strategies in its early stages, research suggests that CT is an effective and pragmatic treatment for many OCD patients. We next turn to a review of the CT outcome literature.

THE EFFECTIVENESS OF COGNITIVE THERAPY

The first controlled study of CT for OCD patients was conducted by Emmelkamp, Visser, and Hoekstra (1988), who compared 10 sessions of CT to 10 sessions of ERP. Cognitive therapy was based on Ellis' (1962) Rational Emotive Therapy. Socratic questioning was used to challenge irrational perfectionism-, threat-, and certainty-related thoughts. Results revealed that CT and ERP were equally effective in reducing OCD symptoms and anxiety/discomfort, although the CT group had more treatment "failures," (ie, less than 30% improvement), than did the ERP group. CT, but not ERP, was associated with pre-post treatment decreases in depressive symptoms. Trend level results showed that CT, but not ERP, was associated with decreased irrational beliefs.

This study was the first to show that CT decreases obsessive-compulsive and depressive symptoms in OCD patients. It was also the first to document a potential

association between effective CT and a decrease in irrational beliefs. However, the inferences that can be drawn from between group comparisons may be limited given that some CT patients engaged in exposure exercises, despite not being instructed to do so. Additionally, ERP patients conducted self-controlled exposure instead of the potentially more effective therapist-guided exposure. Moreover, small sample sizes (9 per group), the absence of precise descriptions of the CT techniques, and the use of outcome measures not designed to measure OCD-specific irrational beliefs limit inferences about whether OCD-specific CT techniques decreased dysfunctional OCD-specific beliefs per se.

Emmelkamp and Beens (1991) replicated Emmelkamp et al.'s (1988) results in a subsequent study with 21 patients who were randomly assigned to either ERP or CT. All patients underwent a 4-week waiting period before receiving six sessions of either ERP or CT. Following this, patients underwent a second 4-week waiting period, after which ERP patients received six more ERP sessions and CT patients received six sessions of CT combined with ERP. Researchers conducted assessments at multiple time points, including post-treatment one (six sessions), post-wait period one, post-treatment two (12 sessions), and post-wait period two.

Post-treatment one assessments revealed that both CT and ERP were equally effective in reducing OCD symptoms, anxiety/discomfort associated with OCD symptoms, and depressive symptoms. However, after wait period two, CT patients had improved on a measure of irrational beliefs more than did ERP patients. Post-treatment two results suggested that combined CT and ERP was not more effective than ERP alone or CT alone in reducing OCD symptoms. These findings again demonstrated the effectiveness of CT, however, limitations similar to those in the Emmelkamp et al. (1988) study left unanswered questions about whether CT was effective in modifying OCD-specific beliefs or for patients with specific symptom presentations.

Cottraux et al. (2001) addressed the question of whether CT modifies certain OCD-specific beliefs in a study comparing CT to ERP. Cognitive therapy consisted of 30, 1-h sessions, occurring 1–2 times per week. Therapists elicited intrusive and automatic thoughts and used Socratic dialogue to discuss distorted beliefs regarding responsibility and danger and the importance of intrusive thoughts. Although, CT patients were not instructed to conduct *prolonged* ERP exercises, treatment did involve behavioral experiments used to test beliefs. Exposure and response prevention consisted of 2-h sessions conducted twice weekly for the first 4 weeks, followed by 12, 40-min sessions conducted every other week. Sixty-five nondepressed OCD outpatients entered the study and 30 in each condition completed treatment.

Cottraux et al.'s (2001) results further demonstrated the efficacy of CT. At post-treatment, CT and ERP were equally effective in reducing OCD symptom severity: 23 CT patients and 21 ERP patients met the 25% Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) improvement criterion. Furthermore, treatment gains were maintained in both groups: at 1 year follow-up, 19 of 25 CT patients and 20 of 23 ERP patients still met improvement criterion. Follow-up effect sizes calculated from Y-BOCS scores were larger for ERP than for CT, possibly suggesting a relative long-term superiority of ERP. However, because 26% of patients were lost to follow-up, long-term results may be somewhat misleading and the maintenance of CT treatment gains remains in need of research. Effect size estimates also suggested a relative superiority of CT to ERP in disturbance attributable to obsessive thoughts, depression, general quality

of life, and misinterpretations. Similarly, follow-up effect sizes suggested a relative superiority of CT versus ERP on measures of depression and distorted beliefs.

The first study to examine the effectiveness of "pure" CT versus "pure" ERP was conducted by van Oppen et al. (1995) who examined: (1) the effects of six sessions of CT *without behavioral experiments* versus six sessions of self-controlled in vivo ERP *without discussions of expectations regarding catastrophic future outcomes*, and (2) the effects of CT augmented by an additional 10 sessions that included behavioral experiments versus ERP augmented by discussion of expectations regarding catastrophic future outcomes. Unlike the previously reviewed studies, van Oppen et al., administered CT based on the theories of both Beck (1976) and Salkovskis (1985) and included OCD-specific CT techniques targeting two of the six domains described by the OCCWG: responsibility (eg, pie technique) and overestimation of danger (eg, calculating probability estimates).

Results indicated significant improvement in OCD symptoms from both CT and ERP after six sessions and after 16 sessions. After six sessions, brief "pure" CT was as effective as brief "pure" ERP. After session 16, CT patients improved significantly more than did ERP patients on measures of OCD, suggesting again that CT with behavioral experiments was at least as effective, and perhaps more so, than self controlled ERP. Results also showed that CT patients were relatively more improved than were ERP patients on measures of anxiety/discomfort associated with OCD symptoms and depression, suggesting (as in previous studies) that CT may have broader clinical effects than does ERP alone. Trend level results showed that CT patients improved more than did ERP patients on measures of irrational beliefs suggesting that CT's therapeutic effects may be related to its ability to modify distorted beliefs. However, because researchers quantified belief change with a general irrational beliefs measure, questions remain about the relationship between OCD-specific CT techniques and changes in OCD-specific beliefs.

Freeston et al. (1997) incorporated a wide range of contemporary OCD belief domain-specific CT techniques in their study on the efficacy of combining cognitive and behavioral techniques (CBT) for OCD patients with severe obsessions but no overt compulsions. CBT strategies included psychoeduction about the cognitive model of OCD, exposure to distressing thoughts via loop-tapes, response prevention, CT techniques, and a relapse prevention module. Contemporary OCD-specific CT techniques were matched with patients' idiosyncratic beliefs and targeted faulty thoughts regarding the overimportance of intrusive thoughts, perfectionism, intolerance of uncertainty, need to control thoughts, inflated responsibility, and overestimation of threat.

Results suggested that, relative to a wait-list condition, CBT was highly effective and resulted in significant improvement in OCD symptoms, general anxiety, and depression. Moreover, at 6-month follow-up, 53% of treated patients had maintained their improvement. Thus, Freeston et al.'s (1997) study demonstrated that CBT incorporating CT techniques targeted toward OCD-specific belief domains was highly effective for OCD patients without overt compulsions, a group generally thought to be highly treatment refractory (Greist, 1990). Nevertheless, because of the combined nature of the treatment, it is impossible to discern the unique contributions of CT and ERP techniques. However, the authors noted at least one potential contribution of CT: of the 22% of the patients who prematurely discontinued CBT, most did so during

the ERP phase. In the authors' opinion, treatment acceptance and adherence may have been enhanced by CT techniques. Hence, CT may be a successful augmentation strategy for patients who might otherwise drop out from ERP.

Research examining the effects of CT techniques aimed at modifying OCD-specific cognitive distortions is just now beginning. Following up on the results reviewed above, our group conducted an open trial of CT with 15 OCD patients with a wide range of OCD symptom presentations and domain specific-cognitive distortions (Wilhelm et al., in press). Care was taken to target and measure distorted beliefs in each of the six belief domains identified by the OCCWG. General and OCD-specific CT techniques were chosen on a patient by patient basis to address patients' idiosyncratic cognitive distortions. Brief behavioral experiments were allowed to test distorted beliefs and belief alternatives. Patients underwent 14, weekly, 50–60 min sessions. Outcome measures included pre-post treatment assessments of OCD severity, depression, and OCD domain-specific beliefs as quantified by the OCCWG's Obsessive Beliefs Questionnaire (Steketee, Fama, & Golan, 2003). Results showed significant improvements in OCD and depressive symptoms, again demonstrating CT's effectiveness in reducing symptoms of OCD and depression. Additionally, results showed significant decreases in all six of the OCD domain-specific beliefs.

In summary, research on the effectiveness of treatments incorporating the CT strategies outlined above suggests that these techniques are effective in treating OCD. Cognitive therapy with varying degrees of behavioral experiments is an effective OCD treatment in it's own right, one which may be as least as effective as ERP. Furthermore, research suggests that CT may have broader therapeutic effects than ERP. Specifically, CT may better increase quality of life, and more effectively decrease depressive symptoms, anxiety/discomfort, and distorted beliefs. Still, these tentative conclusions are based on few studies, and further research on the efficacy and mechanisms underlying change in CT is needed as is research addressing the efficacy of CT conducted in group formats.

RESEARCH ON GROUP COGNITIVE THERAPY

Two studies have examined the use of CT techniques in group therapy settings. Cordioli et al. (2003) conducted a randomized, controlled trial comparing group CBT against a waitlist control condition. Cognitive and behavioral techniques consisted of 12, 2-h, weekly sessions involving psychoeducation, ERP and CT techniques, and relapse prevention. Results indicated that group CBT outperformed waitlist control: at post treatment, 69.9% of the treatment group ($n = 23$) versus only 4.2% of the control group ($n = 24$) were improved, as defined by a Y-BOCS reduction of at least 35%. At 3-month follow-up, treatment gains were not only maintained, but enhanced, with 73.9% of patients showing a Y-BOCS reduction of at least 35%. However, despite significant improvement, 50% of patients remained symptomatic as post-treatment, most of whom had demonstrated poor compliance due to fear of exposure exercises.

In the second study, McLean et al. (2001) examined the relative effects of group CT and group ERP in reducing OCD symptoms and in changing distorted beliefs. Both treatments were evaluated against a waitlist control condition (in which patients later

received active treatment). Groups of 6–8 patients received 12, 2.5-h group sessions. Group CT consisted of psychoeducation about the cognitive model and therapist-assisted challenging of OCD domain-specific beliefs. Behavioral experiments were used to test beliefs. Group ERP included psychoeducation and hierarchically imposed exposure exercises. Additionally, ERP patients were instructed to account for their improvement with a behavioral (habituation) explanation.

Results showed that both CT and ERP were more effective than waitlist, and that ERP was slightly more effective than CT in reducing OCD symptoms at post-treatment and significantly more effective than CT at 3-month follow-up. This remained true even after the researchers controlled for the higher drop out rate in the ERP group. Although, the authors report that treatment responders showed a significant reduction in depressive symptoms, no differential effects of treatment were reported. Analysis of treatment-related cognitive change showed that distorted responsibility beliefs decreased equally in the CT and ERP conditions. However, no significant changes were observed in the other belief domains that were targeted.

These results suggest a relative superiority of group ERP over group CT. The authors hypothesized that the relative inefficacy of CT, in contrast to previous studies on individual CT, may be attributable to the group treatment modality. In the authors' opinion, the modeling and social pressure that occurs in group settings may have increased compliance and, hence, the results of ERP. Conversely, owing to the relatively idiosyncratic nature of distorted beliefs present in a heterogeneous group of OCD patients, it is possible that the group modality may be less amenable to the modification of beliefs and, hence, detract from the efficacy of CT. The plausibility of this explanation is supported by the study results: only one of seven belief domains measured showed any pre-post change.

COGNITIVE VERSUS BEHAVIORAL THERAPY FOR OCD: WHAT WORKS BEST FOR WHOM?

The efficacy of ERP for OCD has been well documented (eg, Abramowitz, 1997) and there is now a growing body of research reporting encouraging results of CT for OCD. However, there is no indication of categorical superiority of one treatment over the other for all OCD patients. Hence, the question arises: are there patients for whom one treatment may be preferable? We address this and related issues below.

Feasibility of Administration

Treatment Acceptance and Compliance

Exposure and response prevention is an effective treatment for OCD: response rates range from of 63% (see Stanley & Turner, 1995) to 90% (Riggs & Foa, 1993). However, variability in response rates is likely due in part to differences in inclusion/exclusion criteria, the type and intensity of treatment strategies employed, and the type of statistical analyses run (eg, intent-to-treat versus treatment completers). Despite its effectiveness, at least 10% of ERP completers fail to respond, and another 20% relapse (Riggs & Foa, 1993). Moreover, because of the demanding nature of this therapy, many patients refuse to undertake it. Refusal rates are generally estimated at

30% (Emmelkamp & Foa, 1983; Kozak, Liebowitz, & Foa, 2000). In addition, among those who do undertake ERP, many show poor compliance, which is a predictor of poor response (de Araujo, Ito, & Marks, 1996). Moreover, many patients eventually drop out of treatment. Drop out rates are variable and likely depend on many factors; some estimate ERP drop out rates as high as 40% (Kozak et al., 2000).

On the other hand, few patients refuse CT (Wilhelm, Steketee, Fama, & Golan, 2003). Differential compliance is in need of empirical attention but, as noted, some researchers (eg, Freeston et al., 1997) suggest CT techniques may *increase* compliance with treatments that include anxiety-provoking exposure exercises. Lastly, although some research has documented dropout rates as high as 20% in CT (eg, van Oppen et al., 1995), other estimates are much lower (McLean et al., 2001; Wilhelm et al., 2003; Wilhelm et al., in press). Indeed, in our open trial of CT, only one of 15 patients discontinued treatment; and this was due to relocation out of the area. Although research is needed on the relative acceptance, compliance and completion of CT versus ERP, it is likely that CT's nonreliance on prolonged exposure makes it less threatening and more acceptable to many patients.

Availability

The demanding nature of ERP makes it is an arduous treatment not only for patients, but for therapists as well. Many ERP protocols require multiple sessions per week and treatment exercises that must be completed outside the therapist's office (eg, in public rest rooms or in patient's homes; Foa, Franklin, & Kozak, 1998). Exposure and response prevention gains are most likely to occur when sessions are at least 90 min in duration (Foa et al., 1998); and ERP related symptom reduction has been correlated with increased patient–therapist interaction time (see Abramowitz, 1997). Unfortunately, therapists in most clinical settings are often unable (or unwilling) to schedule long and frequent sessions outside of their offices, making the provision of strong ERP somewhat impractical. Additionally, owing to the demands of ERP, many students do not receive adequate training in ERP procedures, whereas most students do receive training in CT techniques. A survey of training practices in clinical psychology showed that only 59% of clinical psychology programs provided didactic information on ERP for OCD, and even fewer (48%) provided supervised experience in its implementation. On the other hand, 90% of programs provided didactic information on Beckian CT for depression, and 80% provided supervised experience in its implementation. Similarly, whereas 22% of adult-focused internship programs provided supervised ERP experience, 59% provided supervised CT experience (Crits-Christoph, Frank, Chambless, Brody, & Karp, 1995.) The relatively lower number of ERP trained therapists likely detracts from its implementation given that CT trained therapists are more available in real world settings.

However, the positive results for group ERP may mitigate some of the above concerns in settings where such treatment is available. For example, if the group format increases compliance in patients via modeling and social pressure, then the above concerns regarding treatment acceptance, compliance, and completion may be less relevant for treatment delivered in group settings. Similarly, given the therapist to patient ratio in group treatment, the relative unavailability of therapists trained in the provision of ERP may be less problematic in settings where group treatment is an option.

OCD Symptom Subtypes

Although OCD is a highly heterogeneous condition, most studies do not examine how patients with different symptom presentations respond to treatment. Furthermore, as reviewed by Ball, Baer, and Otto (1996), most research on ERP has excluded patients with symptoms such as exactness, hoarding, counting, and slowness, focusing instead on those with primary checking and washing compulsions. Some of these studies have shown that patients with checking rituals respond relatively less well to ERP than do those with primarily washing compulsions (eg, Kozak et al., 2000; Steketee & Shapiro, 1995). Perhaps in accordance with this, trend level results in one study reviewed above (van Oppen et al., 1995) suggest the relative superiority of CT over ERP for patients with primary checking symptoms. However, given that many treatment studies have employed sample sizes that are too small to allow for meaningful comparisons among symptom subtypes, no consistent trends have emerged. Nevertheless, modifying cognitive distortions about certainty, responsibility, and threat is likely beneficial for patients who engage in repetitive checking. Clearly, additional research is needed before strong claims can be made about the relative superiority of CT over ERP for specific OCD symptom subtypes.

Patients with primary hoarding symptoms have been relatively underrepresented in many ERP treatment studies (Ball et al., 1996). This is of interest as some research suggests that hoarding, in comparison with other presentations of OCD, is associated with both higher drop out rates and poor response to ERP (Abramowitz, Franklin, Schwartz, & Furr, 2003; Black et al., 1998; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002). Controlled trials of the effectiveness of specific CT techniques with hoarders are needed. However, single case studies and pilot studies suggest that patients with hoarding symptoms may respond to multifaceted treatments combining psychoeducation, decision-making and organizational skills training, ERP, and cognitive restructuring techniques (Frost & Steketee, 1998; Hartl & Frost, 1999; Steketee, Frost, Wincze, Green, & Douglas, 2000). Hence, it seems prudent to recommend the incorporation of CT techniques into treatment protocols for patients with hoarding symptoms.

Cognitive therapy's focus on modifying distorted cognitions may indicate it as a treatment for OCD patients whose symptoms are primarily mental (eg, patients with pure obsessions or those who engage in mental ritualizing). However, research on this topic is also in its infancy. If future studies support this claim, CT would provide a welcome alternative to ERP, which may be of reduced effectiveness for OCD patients who do not evidence overt compulsions (eg, Christensen, Hadzi-Pavlovic, Andrews & Mattick, 1987). Although there are single case study reports of successful ERP in patients with primary obsessions and mental rituals (eg, Abramowitz, 2001, 2002), an early meta-analysis by Christensen et al. (1987) suggested primary obsessive thoughts predicted poor outcome with ERP. Subsequent studies have also suggested that sexual/religious obsessions predict poor outcome with ERP (Mataix-Cols et al., 2002). Although there is no empirical research addressing the relative efficacy of CT over ERP in patients with primary mental symptoms and sexual/religious intrusions, CT techniques that target distorted beliefs about the significance of intrusive thoughts may prove particularly useful with this population. Indeed, we included patients with aggressive, blasphemous, and sexual obsessions in our CT studies and have obtained good results (Wilhelm et al. 2003; Wilhelm et al., in press).

Patients with Comorbid Conditions

The review of CT studies in this chapter suggests that a reduction in depressive symptoms is more likely to accompany successful CT than behavioral therapy (eg, Cottraux et al., 2001; Emmelkamp et al., 1988; van Oppen et al., 1995). Indeed, a review of the treatment predictors literature suggests that severe or persistent depression may predict poor response to ERP, although depression has only inconsistently predicted poor response (Steketee & Shapiro, 1995). Still, based on the few studies reviewed here, CT does appear to be relatively more likely than ERP to ameliorate depressive symptoms, as well as other generalized psychological symptoms, such as OCD-related anxiety/discomfort and poor quality of life. Given the potentially broader therapeutic effects of CT in comparison with ERP, CT techniques may be well suited for patients for whom OCD is accompanied by multiple problems in need of therapeutic attention. Given the high comorbidity of OCD with depression and other anxiety disorders, and the poor quality of life reported by many OCD sufferers, research on this matter is of high importance.

One relatively consistent finding in the literature is that OCD patients with comorbid personality disorders, especially schizotypal personality disorder, are less likely to respond to ERP compared to patients without personality disorders (eg, Steketee, & Shapiro, 1995). Since those with personality disorders may be resistant to other types of psychotherapy, it is especially important to determine whether they would respond to CT. Indeed, recently documented success of CT strategies for schizophrenia (eg, Rector & Beck, 2002; Rector, Seeman, & Segal 2003), suggests that CT can be effective in treating patients with low insight as manifest in strongly held distorted, even delusional, beliefs.

Maintenance of Treatment Gains

Few CT studies have incorporated long-term follow-up assessments. Of these, some suggest that improvement with CT is maintained as well as that following ERP (Emmelkamp et al., 1988, Emmelkamp & Beens, 1991); yet other studies suggest the opposite (Cottraux, 2001; McLean et al., 2001). The long-term effectiveness of ERP is fairly well established (Foa & Kozak, 1996). However, methodologically sound, controlled trials designed to measure long-term effectiveness of CT are very much needed.

CONCLUSIONS

In his 1997 meta-analytic review of the OCD treatment literature, Abramowitz concluded that ERP and CT are both effective when compared to other psychosocial treatments for OCD (eg, relaxation therapy). Effect size analysis showed CT to be non-significantly more effective than ERP. However, given that Abramowitz's review only included studies published through 1995 and meeting strict methodological criteria, he was able to identify just four studies comparing CT to ERP. Three of these studies are reviewed here; the fourth was excluded because it included thought-stopping techniques, which we do not consider a cognitive intervention. As reviewed here,

research published subsequent to Abramowitz's review supports the conclusion that individual CT is an effective treatment for OCD and related symptoms. However, as Abramowitz's review highlights, there is a need for methodologically rigorous research on the relative strengths of CT versus ERP. Research that employs standardized protocols and standardized outcome measures is lacking. Pre-post treatment "improvement" is quantified in various ways, making comparisons among even those studies that report effect sizes exceptionally difficult. Such difficulty prevents adequate comparisons of CT versus ERP, and also hinders one's ability to compare the effectiveness of different versions of CT.

Despite some procedural and theoretical overlap, ERP and CT are presumed to reduce OCD symptoms via different mechanisms. The purported mechanism in CT is the direct modification of distorted beliefs; whereas the primary mechanism in ERP is habituation (eg, Kozak et al., 2000): when patients engage in prolonged exposure to feared stimuli *without performing compulsions* they eventually habituate to the once-feared stimuli. However, the extent to which CT and ERP actually work via these alleged mechanisms is unclear. This is due partly to procedural overlap and partly to the absence of studies employing research methods to adequately assess mechanisms of change. Studies that have included measures of distorted beliefs suggest that CT is relatively more effective than ERP in reducing distorted beliefs; but ERP has been demonstrated to reduce such beliefs nonetheless (eg, McLean et al., 2001). Conversely, several studies have shown that CT reduces anxiety/discomfort associated with OCD symptoms (Emmelkamp et al., 1988; Emmelkamp & Beens, 1991; van Oppen et al., 1995).

Pragmatically, research examining differential effects of "pure" CT and "pure" ERP is difficult, if not impossible, because of the degree of overlap in CT and ERP procedures. Cognitive therapy studies include various types of behavioral experiments. The theoretical rationale behind such experiments may differ from that of exposure, but nevertheless what patients actually do and learn in exposures and behavioral experiments might be quite similar. Moreover, many behavioral therapy protocols include psychoeducation on the rationale of behavioral therapy and ERP, and at least one study (McLean et al., 2001) encouraged patients to evaluate their successes in ERP by using their knowledge about the rationale for this treatment. The extent to which these procedures contribute to the modification of distorted beliefs is unknown. Furthermore, even studies that employ "pure" CT (ie, without behavioral experiments) (eg, van Oppen et al., 1995) cannot stop CT patients from exposing themselves to anxiety-provoking stimuli and preventing rituals on their own. Indeed, alleviation of symptoms, including compulsions, is the goal of any treatment for OCD. As importantly, neither can researchers prevent patients receiving ERP from restructuring cognitions on their own. In fact, as noted by Abramowitz (1997), some ERP experts believe the ultimate goal of prolonged and repeated exposure is the modification of threat appraisals via the modification of fear structures (Foa & Kozak, 1986; Kozak et al., 2000).

Clinically speaking, disentangling "pure" CT from "pure" ERP is not as important as is research that will provide insight into the mechanisms through which OCD patients can be treated most effectively. Other important questions remaining to be addressed include whether some therapeutic mechanisms, or combinations thereof, are better suited for some OCD patients over others; such as those with particular

cognitive distortions, specific symptom subtypes, comorbid conditions, and those whose options are constrained by managed care companies or other practical issues. As treatment research becomes more sophisticated, treatment studies will likely begin to employ measures that more closely examine mechanisms of change.

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Chapter 15

TREATMENT FOR OCD: UNLEASHING THE POWER OF EXPOSURE

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Obsessive-compulsive disorder (OCD) is estimated to occur in about 2.5% of the population (Karno, Golding, Sorensen, & Burnam, 1988). Before the emergence of exposure-based treatment, OCD was largely considered to be chronic. In its severe forms, it was associated with substantial long-term disability. The advent of exposure therapy (Meyer, 1966) was a therapeutic breakthrough that revolutionized psychotherapy for OCD and afforded relief to many individuals for whom no satisfactory therapeutic options were previously available. Despite numerous published treatment studies that have documented the efficacy of exposure treatment for OCD, it is not generally available to consumers, and alternative treatments, most notably pharmacotherapy by serotonergic compounds, and cognitive therapy (CT), have garnered substantial interest among clinicians, consumers, and researchers. How good is exposure treatment and where does it fall short? Satisfactory answers to these questions would point to the extent to which alternative or supplementary treatments are needed. This chapter will explore these issues.

EXPOSURE THERAPY FOR OCD

Exposure-based therapy, traditionally called exposure and response (ritual) prevention (EX/RP), entails exposure to situations that provoke obsessive fear. This exposure can be in the form of repeated imaginal or actual exposure to feared low-risk situations. Sometimes, the exposure procedure includes imaginal confrontation with feared disastrous sequelae of confronting the low-risk situations. For example, an individual who fears contamination from touching doorknobs and bathroom spigots would practice touching doorknobs and bathroom spigots, and might also practice imagining contracting infectious diseases from the contacts.

Another important component of EX/RP for OCD is abstaining from rituals. Rituals are typically performed in response to obsessive intrusions and are intended either to reduce the likelihood of harm from the feared situation, or just to reduce obsessive distress. Exposure and response (ritual) prevention treatment requires that the individual abstain from rituals in response to obsessive urges to ritualize. Such

urges can arise spontaneously, in response to encounters with various situations in vivo, or in response to systematic confrontation with feared situations. During exposure therapy, repeated confrontation with feared situations provokes obsessions and associated distress, which is usually described as fear or anxiety. The procedure requires that the individual continue the confrontation until the distress decreases spontaneously, without attempting to reduce the distress by withdrawing from the situation or by performing compulsive rituals.

The session structure of EX/RP can vary widely. A format that has been found very successful for the majority of consumers is a few hours of assessment and treatment planning, followed by 15 daily sessions, lasting 90–120 min each, spaced over about 3 weeks. Generally, the exposure sessions are supervised by a therapist, and self-exposure practice is prescribed as homework between the therapist-supervised sessions. Sessions might be divided between imaginal and in vivo exposure practice, depending upon the details of the obsessions and upon the pragmatics of confronting actual feared stimulus situations. Not all courses of EX/RP follow the intensive format described above, however, instances of fewer sessions, shorter sessions, and more widely spaced sessions are evident in the research literature and in clinical practice.

Exposure and response (ritual) prevention treatment procedures routinely begin with assessment of obsessions, rituals, and avoidance and of the anticipated harmful consequences of encountering feared situations without ritualizing. Before the exposure exercises are begun, however, the therapist describes the procedures thoroughly to the patient and explains why they are expected to be helpful. This rationale is an important component of the therapy because it helps to motivate the patient to tolerate the distress that typically attends the exposure exercises. A good rationale will include not only a learning-oriented explanation in terms that are readily understandable to the patient, but also information about how the exposure is commonly experienced, including the provocation and diminution of distress during prolonged exposure. The information gleaned from the assessment is used to plan, collaboratively with the patient, the specific exposure exercises that will be pursued.

In addition to explaining and planning a schedule of exposures, the preparatory stage of EX/RP must also introduce the patient to the "response prevention" component of the procedure. Response prevention refers to the prevention of compulsive rituals. The term "response prevention" can be confusing. Although, in principle, one could actively prevent the patient from doing certain kinds of rituals, for example, by disconnecting the water supply in a sink in the patient's room, such direct interference is impractical for many kinds of rituals, and even when practical, cannot be implemented without full time monitoring of the patient. Instead of the therapist's preventing rituals, the therapist must persuade patients to prevent themselves from performing rituals. In effect, the response prevention component of EX/RP is really abstinence from rituals, rather than prevention by an external agent. Self-monitoring of rituals is often prescribed to help support abstinence.

The exposure exercises typically begin with moderately distressing confrontations, and escalate over several days to the most distressing situations. Beginning with moderately difficult tasks probably increases the likelihood that the patient will tolerate the distress and complete the exposure exercise successfully. Early successes can increase confidence in the treatment and help motivate the patient to persist during the most distressing exposure tasks. At the end of each treatment session, the therapist instructs the patient to extend the practice for several hours what was done

in the session, but without the therapist, and in different environmental and interpersonal contexts. The most distressing exposure exercises are not left to the end of the treatment, but rather, are done about a third of the way through the schedule of exposure tasks. This schedule is designed to afford ample time for repeated therapist-guided exposure to the most difficult situations, and for enough self-exposure practice outside of the sessions to permit satisfactory generalization of the treatment effects.

During the later sessions of EX/RP, the therapist reviews with the patient the principles guiding the exposure effects, and emphases the importance of the patient's applying those principles to maintain and enhance treatment gains. Relapse prevention is presented as an active and continuing effort that involves the patient's recognizing occasional obsessive intrusions, and prompt self-exposure and abstinence from rituals.

Exposure and response (ritual) prevention is a powerful technique for reducing symptoms of OCD. Foa and Kozak (1996) reviewed 13 controlled and uncontrolled trials of EX/RP that were conducted between 1975 and 1992, and which involved 330 patients. The average proportion of improved patients, weighted by the number of patients in each study, was 83%. From 40% to 97% of patients were found improved, depending on the study. The more recent estimates of success rates with exposure treatment seem to be at least 75% (Abel, 1993; Abramowitz, 1997; Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000; Kozak, Leibowitz, & Foa, 2000; Stanley & Turner, 1995). It is clear that EX/RP has yielded very favorable responder rates in many studies of OCD: high responder rate is a very robust finding in this outcome literature.

How much symptom reduction occurs in responders is less clear from the treatment outcome literature, but it is clear that EX/RP reduces symptoms substantially. Definitions of "responding" vary across studies, and studies typically include several categories of responding (eg, minimally improved, much improved, and very much improved). A review of 17 controlled studies of EX/RP by Abramowitz, Franklin, and Foa (2002) revealed an average reduction in OCD symptoms of 48%. Because the low symptom reductions of nonresponders contribute to an average of this type, it probably underestimates the amount of symptom reduction that can be expected of responders. Preliminary results of an NIMH sponsored collaborative study (Kozak et al., 2000) indicated that mean symptom reduction for completers of EX/RP was 59% posttreatment and 57% at 3-month follow-up. As might be expected, these improvement rates for treatment completers are better than the overall average of 48% reported in the Abramowitz review. Notably, not all treatment completers are treatment responders. Because good response to EX/RP is the modal outcome, the average symptom reduction among treatment responders is probably somewhat higher than the 59% found for treatment completers, and this is a very palpable and important degree of improvement.

Although EX/RP is a very powerful procedure for OCD, it has limitations that might not be immediately evident from the spate of positive findings available in the outcome literature. Many individuals who are offered EX/RP decline to accept it, and these treatment refusers can be "invisible" if they do not enter a trial, and thus, do not appear in either the "intent-to-treat" or "completer" samples. According to one estimate, 20%–30% of patients either refuse to begin EX/RP or drop out prior to completion, and the responder rate decreases from 63% to 55% when dropouts and treatment refusers are included in responder rate estimates (Stanley & Turner, 1995).

Kozak (1999) noted that in an OCD specialty clinic in Philadelphia, about 25% of those diagnosed with OCD declined to participate in an EX/RP program. Multiplying an estimate of 75% responders among those who accept treatment by the estimate of 75% treatment accepters yields an adjusted responder rate estimate of 56%. This converges with the estimate offered by Stanley and Turner (1995). Thus, although EX/RP is very powerful for those who receive it, its power is importantly limited by treatment refusal.

As noted above, EX/RP yields substantial average symptom reductions that have been estimated from 48% to 59%. This implies that, on average, 41% to 52% of pretreatment symptom severity remains after successful treatment. Because these percentages include the residual symptoms of nonresponders, they do not resolve the question of the modal symptom reduction for responders. Some studies have reported average posttreatment Yale-Brown Obsessive-Compulsive Scale (YBOCS) scores in the low-to-mid teens (eg, van Noppen, Steketee, McCorkle, & Pato, 1997; van Oppen et al., 1995), but average posttreatment YBOCS scores below 10 were achieved in the NIMH multi center OCD trial (Kozak et al., 2000) that emphasized the kind of massed prolonged exposure sessions and rigorous abstinence from ritualizing that seems to produce optimal treatment response. It appears that the best reported average posttreatment YBOCS scores are in the high single digits, which indicate mild, but undesirable OCD symptoms. There is a range of residual symptoms among treatment responders, but the findings noted above indicate that despite substantial average symptom reductions, most patients have at least some residual symptoms after successful EX/RP, which they must monitor and keep in check with self-exposure practice after the formal course of EX/RP is completed. For some treatment responders, the residual symptoms are important enough that further treatment is sought.

In light of the range of estimates of responder rates and percentage symptom reductions with EX/RP for OCD, it can be difficult to conclude what to expect from this treatment. Although it is tempting to agglomerate the findings of available outcome studies to yield some overall outcome estimates, this approach might be deceiving. The EX/RP procedures used in various studies are themselves variable, on dimensions such as duration of exposure, number of exposure sessions, therapist supervision of exposure, and extent of abstinence from rituals. Those studies that use massed exposure sessions that provoke the obsessional distress and long enough to permit it to decrease within sessions, and rigorous ritual abstinence requirements seem to yield the best outcomes (Foa, Steketee, Grayson, Turner, & Latimer, 1984; Kozak, Foa, & Steketee, 1988; Rabavilas, Boulougouris, & Stefanis, 1976; Rachman, deSilva, & Roper, 1976). Thus, the outcome of treatment expectation should be greater or lesser, depending on particular qualities of the EX/RP procedures that are practiced. If an individual receives a suboptimal EX/RP regimen, then lower expectations for outcome, as suggested by the average data for large groups of treatment studies, are probably in order. On the other hand, if an individual receives an optimized EX/RP treatment, then it would be reasonable to expect an outcome suggested by the findings of studies that employed the most rigorous EX/RP procedures. If this analysis is correct, it offers a way to make sense of the somewhat different outcome estimates yielded by different treatment trials: done optimally, EX/RP for OCD is very powerful, but its power can be compromised when the best available procedures are degraded.

Unfortunately, the average health care consumer cannot reasonably expect to realize the high responder rates and large symptom reductions that have been

obtained with intensive treatment programs involving long-duration massed sessions, supervised by therapists with expertise in EX/RP for OCD. Such therapists are not commonly available. Practical limitations often dictate a suboptimal EX/RP regimen: infrequent (ie, weekly) sessions that at best permit brief or no therapist-guided exposure periods, conducted by a generalist practitioner with basic knowledge but not substantial expertise in EX/RP for OCD. It is not clear how the efficacy of EX/RP conducted within the pragmatic constraints of routine clinical practice would reflect the outcome findings for intensive treatment performed by experts. It would not be surprising if the modal outcome in routine practice is more modest than the best outcomes obtained with optimal procedures applied by specialists.

MECHANISMS OF ACTION FOR EXPOSURE THERAPY

Obsessive intrusions seem to involve the experience of emotion associated with a perception of threat, although an actual threat is not present. It is as if an obsession constitutes a threat related memory or image that includes instantiation of a distressing emotional response. Typically, a fearful idea that occurs in the absence of a dangerous situation will fade spontaneously when no harm ensues in the situation. Rachman (1980) noted that the fading of such emotional responses is important, and seems to be a kind of emotional processing, the impairment of which undergirds certain kinds of psychopathology, as indicated by "intrusive signs of emotional activities such as obsessions [and] phobias" (p. 51). Psychodynamic psychotherapy hypothesizes that bringing unconscious emotional memories into consciousness will help to relieve related symptoms. In trying to understand the mechanism of exposure-based treatments, behavior therapy researchers have also focused on the experience of affect. For example, Lang, Melamed, and Hart (1970) found that phobics who were most able to have fearful imagery during therapy benefited most from systematic desensitization, an exposure-based procedure of documented efficacy for phobias, and Lang (1977) suggested that emotional responding during imagery might be a "key to the emotional processing which the therapy is designed to accomplish" (p. 863).

An information processing approach to understanding the mechanism of exposure-based treatment for anxiety disorders, including OCD, was elaborated by Foa and Kozak (1985, 1986). In developing this idea, they adopted Lang's (1977, 1979) construal of fear behavior as founded in propositional networks of emotional imagery or "fear structures." Accordingly, these networks can be analyzed into three kinds of information, coded as "propositions" (cf. Pylyshyn, 1973). *Stimulus propositions* code information about the feared stimulus situation. *Response propositions* code information about behavioral and physiological responses. *Meaning propositions* code information about the relationship of stimuli and responses and semantic interpretations of their meaning. In Lang's theory, the fear structure is essentially a network of these three kinds of propositions, which serves as a program for escape or avoidance.

Viewed as a program to escape or avoid harm, a fear structure must involve propositions about harm, ie, that something about the feared situation is dangerous. Two elements, perception of danger and preparation to escape, distinguish fear structures from other emotional images, and when a fear structure is active, a person is afraid. Ordinarily, the presence of a real threat gives rise to a mental representation

(fear structure) of the details of the situation (stimulus propositions), which also includes a perception that aspects of the situation are dangerous (threat meaning propositions), and contains "commands" for physiological preparation, such as heart rate increase, to escape, or avoid (physiological response propositions). Normally, if the potential for harm is somehow removed, if the individual escapes, or if some new stimulus information changes the perception to one of safety, the mental representation changes accordingly, and fear decreases. Problems arise when the fear structure does not match the reality of the situation but does not change to become more realistic, and the fear persists in the absence of danger. When the fear is intense and persistent in the face of information that it is unrealistic, it is considered pathological (Foa & Kozak, 1985, 1986). In theoretical terms, a pathological fear structure contains stimulus propositions, response propositions, and meaning propositions that do not accurately represent real objects and events.

A theoretical mechanism of emotional processing, by which exposure therapy is hypothesized to change pathological fear structures, was elaborated by Foa and Kozak (1985, 1986). Accordingly, two conditions are required for fear reduction via exposure treatment. First, fear-relevant information must be presented to the patient in a way that will evoke the pathological fear structure. This presentation must also include information that is incompatible with the pathological fear structure (cf. Lang, 1977) and must be incorporated into the pathological emotional representation that has been evoked by the exposure task. The corrective incorporation of new information into a pathological fear image is a kind of learning that is hypothesized to operate in emotional processing. A successful exposure therapy for OCD must employ confrontations with situations that evoke the obsessive fear, but which are not dangerous, so that the corrective information can influence the pathological fear structure.

There are three putative indicators that an exposure treatment is promoting therapeutic emotional processing. The first indicator is evidence that the pathological fear structure is being evoked by the exposure task. Successful evocation of the fear structure is evidenced by the patient's becoming afraid when doing the exposure. This is usually evident in the patient's report of being anxious, and in physiological responses like those that the patient typically experiences when afraid. The second indicator is a gradual decrease of fear within the exposure session, despite persistent confrontation with nondangerous fear-relevant stimuli. The third indicator is decrease in fear response across exposure sessions. Evidence for the validity of these indicators, especially the first and third, can be found in studies of treatment outcome for a variety of anxiety disorders (for review, see Foa & Kozak, 1986). Evidence for the indicators continues to accumulate. For example, in a psychophysiological assessment of speech anxious volunteers, Schwartz and Kaloupek (1987) found that fear activation during exposure, and decreasing fear within and between exposures sessions predicted good treatment outcome. Studying the psychophysiological process of EX/RP for OCD, Kozak et al. (1988) found that activation of fear during exposure, and decreases in evoked fear across exposure sessions, predicted good treatment outcome. The validity of these indicators of emotional processing seems to be one of the more robust findings available in the process of therapy literature, and lends substantial support to the hypothesized mechanism of emotional processing for the effects of exposure-based treatment for anxiety disorders.

PHARMACOTHERAPY FOR OCD

In addition to data demonstrating the efficacy of EX/RP, evidence has accrued for the efficacy of pharmacotherapy with the serotonin reuptake inhibitor (SRI) clomipramine (Anafranil), and a number of selective serotonin reuptake inhibitors (SSRIs). Clomipramine is probably the most established pharmacotherapy for OCD, with a responder rate of about 60% and symptom reductions of about 39% (Clomipramine Collaborative Study Group, 1991). The SSRIs have also been found to be effective. For example, controlled studies of fluvoxamine (Luvox) have found OCD symptom reductions in the range of 17–25% (eg, Goodman, Kozak, Liebowitz, &White, 1996; Price, Goodman, Charney, Rasmussen, & Heninger, 1987). Symptom reductions of about 25% have been found in controlled trials of sertraline (Zoloft: Chouinard et al., 1990; Greist et al., 1995a), and fluoxetine (Prozac: Tollefson, Birkett, Koran, & Genduso, 1994). A double blind placebo controlled study of paroxetine (Paxil) found OCD symptom reductions of about 31% (Goodman, Steiner, Bushnell, Gergel, & Wheadon 1996). Citralopram (Celexa) is a more recently available SSRI that is less well evidenced than the other above-mentioned compounds, but is of interest for OCD (Montgomery, 1998).

The repeated demonstrations of the potency of serotonergic compounds for reducing OCD symptoms present an impression of substantial efficacy. However, like EX/RP, pharmacotherapy of OCD has limitations. Pharmacotherapy works for a subset of patients with OCD, and mean symptom reductions are smaller than from EX/RP. For example, a meta-analysis of major multicenter studies of the anti-obsessional compounds found decreases in OCD symptom scores (Y-BOCS) of 4–10 points from pretreatment scores of 22–26 (Greist, Jefferson, Kobak, Katzelnick, & Serlin, 1995b). Although these are noticeable improvements, even responders typically have clinically significant residual symptoms. Outcomes in routine clinical practice may be more modest, given that many patients outside of controlled trials might receive suboptimal doses because of side-effects. Another consideration is that as with EX/RP, treatment refusal is a limitation for pharmacotherapy. Although satisfactory estimates are not generally available, Kozak (1999) noted a 7% refusal rate for clomipramine. Relapse after medication discontinuation is another limitation. Relapse estimates vary from 24% after discontinuation of sertraline (Koran, Hacket, Rubin, Wolkow, & Robinson, 2001) to between 31% and 89% after discontinuation of clomipramine (Pato, Zohar-Kadouch, Zohar, & Murphy, 1988; Ravizza, Barzega, Bellino, Bogetta, & Maina, 1996), figures notably higher than the 12% relapse following completion of EX/RP (Simpson et al., 2003).

It is clear that a substantial number of patients with OCD realize a good response to medication but that the response is typically partial. (For a thorough review, see Simpson & Franklin, this volume.) Because of the limitations of drug monotherapy and of EX/RP alone, there has been interest in combination treatments. Unfortunately, controlled studies of pharmacotherapy augmentation by other medications are not very encouraging (Barr, Goodman, Anand, McDougle, & Price, 1997; McDougle, Price, Goodman, Charney, & Heninger, 1991; McDougle et al., 1993). Studies of combined EX/RP and pharmacotherapy have provided mixed results. Some studies have found modest additive effects from combining EX/RP and pharmacotherapy in treating OCD (Cottraux et al., 1990; Hohagan et al., 1998; Marks, Stern, Mawson, Cobb, & McDonald, 1980; Neziroglu, 1979), although others have failed to find additional

benefits of combining these two treatments (Kozak et al., 2000; Van Balkom et al., 1998).

What are the implications the pharmacotherapy literature for EX/RP? Individually potent pharmacotherapy might be a useful adjunct to exposure. It is possible that premedication with a potent SRI could increase the acceptability of EX/RP to otherwise reluctant individuals. Given the high efficacy of intensive EX/RP *for completers*, and the consequential proportion of EX/RP treatment refusers, a finding that premedication reduces the EX/RP refusal rate would be an important advance. However, only experimental designs that circumvent the "problem" of high EX/RP acceptance and responder rates could be expected to reveal such an adjunctive capacity. Alternatively, one might expect that EX/RP could help patients who respond partially to pharmacotherapy, and because of the ample proportion of pharmacotherapy partial responders, a study designed to detect such effects could be realized. These approaches offer promise that combining pharmacotherapy and EX/RP might enhance outcomes.

COGNITIVE THERAPY FOR OCD

Treatment refusal and nonpartial responding are limitations of EX/RP. Pharmacotherapy can be a useful alternative or perhaps, supplementary treatment. However, as discussed above, pharmacotherapy has its own limitations, and seems to be generally less potent than EX/RP. Also as discussed above, it is yet to be established that the combination of EX/RP and pharmacotherapy is superior to EX/RP alone. Partly in response to the limitations of pharmacotherapy and of EX/RP, CT has been of interest for OCD.

Cognitive approaches to OCD stem from clinical observations that the patient's thoughts are important elements of the pathology of OCD, and the assumption that changing pathological thoughts could be a mechanism of therapeutic change. There are various theoretical approaches to the nature of cognitive pathology in OCD, and different therapy procedures that are derived from them. For example, Carr (1974) suggested that erroneous estimation of threat characterized thinking in OCD, and that it involved overestimates of the probability of harmful events and of their cost. Rituals are hypothesized to reduce perception of threat. The therapeutic implication was that a procedure that corrected the threat estimates could be effective in ameliorating OCD. Meichenbaum's (1975) hypotheses about the importance of negative-self statements led to the use of self-instructional training to counter negative thoughts (Emmelkamp, van der Helm, van Zanten, & Plochg, 1980). McFall and Wollersheim (1979) analyzed threat estimation into two stages of appraisal: a primary stage in which potential harm is compared to coping capacity, and a secondary stage in which consequences are appraised. These appraisals are hypothetically influenced by various pathological beliefs, such as perfectionism. Compulsive rituals are hypothesized to circumvent other coping with the perceived threat. The therapeutic implication is that CT techniques that have been used to modify beliefs could be used to reduce OCD symptoms by modifying beliefs that support pathological appraisals. These hypotheses have been adopted in the development of CT procedures along the lines advocated by Ellis (1962), in which the therapist helps the patient identify and challenge irrational beliefs that are supposed to be central to the OCD (eg, Emmelkamp & Beens, 1991). Cognitive

biases about threat have been found for a variety of anxiety disorders, including OCD (for review see Amir & Kozak, 2002), but the above-mentioned cognitive approaches to OCD do not accommodate the complexity of these findings, or provide a cognitive account of differences among anxiety disorders.

Perhaps because the cognitive approaches cited above were not found alone to be superior to EX/RP, or in combination, to enhance the effects of EX/RP, they have not become established treatments for OCD. More recent cognitive hypotheses introduced by Salkovskis (1985, 1989) and Rachman (1997, 1998) have guided much of the contemporary work on cognitive treatment for OCD. This approach posits pathological interpretations of obsessive intrusions, and hypothesizes that these erroneous appraisals of the intrusions are central to OCD. Derivative treatment procedures focus directly on modifying the hypothesized pathological intEXRPretations (eg, Freeston, Rheaume, & Ladouceur, 1996).

Cognitive therapy typically begins with presentation of a rationale for treatment. A rationale following the hypotheses of Salkovskis might be that the intrusive thoughts experienced in OCD are not themselves harmful or illustrative of anything important. Rather, problems arise when the patient perceives them as significant in some way that is unacceptable. Accordingly, it is the way the patient *interprets* the intrusions that causes distress, and leads to reactions that inadvertently reinforce the intrusions. This conceptualization formally resembles a prevailing cognitive conceptualization of panic disorder (Clark, 1986), which hypothesizes that it is not interoceptive sensations or their physical instantiation that are of primary interest, but rather, the catastrophic meanings mistakenly ascribed to them by the patient. In other words, regardless of the origin of a distressing sensation, without misperceived threat, there can be no panic disorder. The therapeutic implication is that correcting the relevant misperceptions will obviate panic. A parallel formulation of the pathology in OCD assumes that unwanted intrusions are normal (Rachman & deSilva, 1978), and it is their misinterpretation that causes OCD. A rationale for CT is that it will ameliorate OCD by helping the patient reinterpret the unwanted intrusions as nonthreatening.

Various techniques are used to help patients correct erroneous ideas, but a common thread is rational discussion of problematic ideas by patient and therapist, with the therapist guiding the patient to challenge the ideas by questioning and by evaluation of relevant evidence. Other techniques besides rational discussion are also used however. For example, Clark (1999) noted that because the Socratic method does not fit well with the cognitive rigidity of many OCD patients, the use of "behavioral experiments" might be especially important. Such "experiments" are characteristic of traditional CT for depression (Beck, Rush, Shaw, & Emery, 1979) and for panic disorder (Clark, Salkovskis, & Hackman, 1994) and can be construed as a kind of hypothesis testing that involves gathering evidence about the erroneous belief. This testing is accomplished by the patient's entering and observing situations that exemplify the idea under scrutiny. Although the rationale for behavioral experiments in CT is somewhat different than that for the exposure exercises of EX/RP, there is overlap, and the essential difference between the two procedures can be difficult to resolve.

Evaluation of the efficacy of CT for OCD is saddled with the common problem of varied treatment procedures across different studies. Evaluation of the relative efficacy of CT and EX/RP is additionally complicated by problems of procedural overlap: CT routinely includes behavioral experiments that seem tantamount to the exposure exercises of EX/RP, and EX/RP routinely includes discussions of risk and

consequences that seem tantamount to the Socratic dialogue of CT. There is a risk that comparison studies designed to preserve the integrity of idealized EX/RP or CT programs would sacrifice the capacity to disentangle mechanisms of action, and that studies that "purify" treatment programs to isolate procedural components would sacrifice the integrity of the treatment programs in ways that could compromise them by depriving them not only of some potent individual components, but also of the interactive efficacies of these components.

Cognitive therapy repeatedly has been found to reduce OCD symptoms. For example, a CT regimen that targeted erroneous estimates of threat, without employing exposure or behavioral experiments, was found superior to a no-treatment period, and reduced OCD symptoms by about 20% (Jones & Menzies, 1998). However, this is considerably less improvement than would be expected with the best available EX/RP regimens. Cognitive therapy was compared to EX/RP in two studies by Emmelkamp and colleagues (Emmelkamp & Beens, 1991; Emmelkamp, Visser, & Hoekstra, 1988), in which the two procedures yielded equivalent improvements in OCD symptoms. The CT procedures in these studies resembled Ellis' rational emotive therapy, and did not include exposure-like behavioral experiments, but unlike the massed, therapist-guided exposure sessions that have been found to yield the best available outcomes, the EX/RP condition did not involve in-session therapist-guided exposure, but rather, self-guided exposure homework. Another comparison of CT with suboptimal EX/RP was conducted by van Oppen et al. (1995), which evaluated CT that included behavioral experiments, and an EX/RP procedure that involved weekly therapist sessions and self-guided exposure. This CT procedure with exposure-like elements was found to be at least as good as the EX/RP condition. Van Balkom et al. (1998) study used procedures and samples that overlapped with those of the van Oppen et al. (1995) study, and found that the cognitive procedures and EX/RP were superior to a no-treatment period, and did not differ from one another. The results of these treatment evaluations support a conclusion that some CT procedures can be as potent as certain suboptimal EX/RP procedures.

In a review of 17 controlled studies of EX/RP, CT, and their combination, Abramowitz et al. (2002), noted that six studies comparing CT and EX/RP found equivalent efficacy, but that the cognitive procedures included exposure-like behavioral experiments, and many of the EX/RP procedures involved nonsupervised exposure. For example, one study that compared CT to intensive exposure (4 weeks of two 2-h sessions followed by 14 weeks of 40-min booster sessions every other week; Cottraux et al., 2001) found similar response rates for intensive behavior therapy and CT. However, the CT included behavioral experiments, and patients were encouraged to confront feared situations to modify their thoughts. Thus, the cognitive procedure involved exposure. Abramowitz et al. (2002) concluded that the efficacy of CT alone is equivalent to "substandard" exposure. Average OCD symptom reduction for EX/RP was 48%, for CT with behavioral experiments, 30%, and for EX/RP combined with CT, 39%. An ongoing controlled trial of CT with brief behavioral experiments (Wilhelm, Steketee, Fama, & Golan, 2003) is yielding preliminary findings of a larger reduction in OCD symptoms than has typically been reported for CT but their interpretation must await the completion of the study. Overall, CT has been found to reduce OCD symptoms more than no-treatment, and, when it includes exposure-like behavioral experiments, it competes well with suboptimal EX/RP. However, neither CT alone, nor in combination with EX/RP, competes well with intensive, therapist-supervised

EX/RP and rigorous abstinence from rituals, for example, as described in the treatment manual by Kozak and Foa (1997). This, rather than no-treatment, stress-management, SRIs, or compromised EX/RP, seems to be the procedure to beat in comparative outcome trials.

As reviewed above, the available evidence suggests limited potency of CT for OCD. Serotonin reuptake inhibitors appear to be at least as potent as CT, and EX/RP, more potent. Exposure and response (ritual) prevention has been subject to dismantling studies (eg, Foa, Steketee, & Milby, 1980; Foa et al., 1984; Grayson, Foa, & Steketee, 1986) that have indicated separate contributions of its exposure and antiritual components. Although similar dismantling studies are not available for CT, one might make a weak inference from the van Oppen et al. (1995) trial that the most palpable OCD symptom reductions occurred with the instigation of the exposure-like behavioral experiments of the cognitive procedures employed. Assessment of the psychophysiological process of fear reduction during exposure exercises for OCD has yielded results that converge with those of assessments of exposure with other anxiety disorders, and which support hypotheses that fear activation and reduction between and within sessions are mechanisms of action for EX/RP. This is one of the more reliable findings in the psychotherapy process literature. Although hypotheses abound for the mechanisms of CT for OCD, parallel experimental support from treatment trials has not been forthcoming.

For EX/RP, various findings point to habituation of fear within and between sessions as mediators of the treatment effects. For CT for OCD, there is some evidence that it produces cognitive changes (Bouvard, 2002; Emmelkamp, van Oppen, & Van Balkom, 2002), but experiments that demonstrate the mediation of outcome by hypothesized cognitive mechanisms are not available. Conventionally, demonstration of mediation is considered to require that changes in the hypothesized mediator occur after the experimental manipulation has been instigated, and that such changes are associated with the outcome variable. Although cognitive changes have been found after CT (and EX/RP) for OCD, changes in cognitive variables such as though–action fusion, excessive responsibility, disastrous interpretation of intrusions, and so forth that have been central to cognitive theories of OCD have not been established to be mediators of the effects of CT. One ongoing controlled study of CT for OCD (Wilhelm, Steketee, Fama, & Golanl, 2003) has yielded some preliminary results supporting cognitive mediators of outcome, and might eventually provide convincing evidence in this regard.

THE ROLE OF COGNITIVE THERAPY IN TREATING OCD

Given the relative limitations of CT for treating OCD, what useful role might it have in such treatment? As reviewed above, it is clear that although SRIs and EX/RP are potent treatments, it is also clear that there is a nontrivial proportion of nonresponders and treatment refusers, and responders to the established treatments often have enough residual symptoms to provoke further treatment-seeking. Attempts to achieve better results from the established monotherapies by applying them in combination with each other, or with nonestablished treatments, have not solved the problem. Nevertheless, there are some reasons to believe that CT could be helpful. Cognitive

theorists might note that although evaluations of EX/RP routinely focus on the parameters of exposure and abstinence from rituals, they are generally inattentive to other aspects of the procedure, including discussions of risk and consequences that are not themselves exposure exercises, and which bear formal resemblance to some of the techniques of CT. As noted above, there are findings that a CT procedure that targeted erroneous estimates of threat, without employing exposure or behavioral experiments, reduced OCD symptoms by about 20% (Jones & Menzies, 1998). This finding suggests that CT techniques themselves can produce improvement.

Although the repeatedly observed findings that activation of fear and its decrease within and between sessions predict good outcome of EX/RP, these results leave unexplained substantial proportions of the variance of prediction. It is unclear how much of the unexplained treatment outcome variance stems from method error, and how much stems from important variables that were not measured. Thus, although it is clear that fear activation and its decrease during exposure indicate successful emotional processing, it not clear that this kind of emotional processing is the only possible pathway to reduction of pathological fear. It is reasonable to speculate that the 20% OCD symptom reduction obtained by Jones and Menzies did not involve activation of fear and its decrease with prolonged exposure. On the other hand, one could also speculate that discussions of risk, consequences, and responsibility that characterize contemporary CT techniques operate by persuading individuals to confront on their own, feared objects and situations for prolonged periods without ritualizing, thus recruiting the mechanisms of fear reduction via exposure. The available evidence does not afford resolution of these competing hypotheses about the potential contributions of CT techniques. However, they might operate primarily by preparing patients for exposure and thus, unleashing its power to reduce fear.

DECISION MAKING ABOUT TREATMENT

Preparing patients to accept and persist with treatment in the face of their uncertainty about it remains an informal aspect of EX/RP. It has not been subject to the same attention and study as the specific contributions of exposure and abstinence from rituals. There are no established standards for how to persuade fearful individuals to confront feared situations: this persuasion remains more of an art than a technology for exposure-based treatment. As noted above, it is possible that some CT procedures operate by persuading patients to confront mistakenly feared situations. Such procedures might support exposure in the form of prescribed behavioral experiments, and might lead to nonprescribed self-exposure as well.

In addition to what is known about documented CT procedures, other information might be available that could inform therapist attempts to engage patients in EX/RP. Clinical writings (eg, Kozak & Foa, 1997; Steketee, 1993) present examples of preparing the patient for therapy for OCD. Such examples generally emphasize psychoeducation from an expert (the therapist) as a means to facilitate acceptance of the proposed maintaining factors for OCD, and therefore logically lead to the recommended treatment via EX/RP. Many clinicians working with OCD also advocate providing patients with data supporting the efficacy of EX/RP, summarizing the existing treatment outcome literature (eg, Steketee, 1993, p. 96). Although there is some research demonstrating the benefits of providing a treatment rationale (Marcia, Rubin,

& Efran, 1969; McReynolds & Tori, 1972; Oliveau, Agras, Leitenberg, Moore, & Wright, 1969), experimental studies of what information to present and of how to present it have not been forthcoming. The experimental literature on persuasion might provide additional guidance. A body of empirical work has delineated routes of persuasion and variables that influence the effectiveness of such messages. This work suggests some approaches that might increase the likelihood that a patient in need would decide to accept EX/RP.

Methods of psychoeducation and providing empirical support for the efficacy of EX/RP are consistent with persuasion methods targeting what has been called a "central route" of processing. According to Petty and Cacioppo (1986) persuasion via the central route depends upon argument strength, and involves critical consideration of the validity and strength of the arguments presented. Importantly, this assumes a kind of rationality that is sometimes lacking in anxious patient's thinking about threats: relative unresponsiveness to information about safety, presented in an ordinary rational way, is a hallmark of pathological fear. Explaining to a germ phobic that others routinely touch a particular surface without subsequent decontamination and do not get sick is not sufficient to eliminate the fear. Otherwise, no special therapy procedures would be of interest. Contemporary treatment rationales do not seem explicitly oriented to the "peripheral route" of persuasion, which is less direct, and involves decision heuristics and relevant incidental cues, such as the attractiveness of the presenter, the perceived authority of the presenter, reactions of others, or the number of arguments presented.

Numerous factors are proposed to influence processing route. For example, cognitive models (Petty & Cacioppo, 1986) propose that when motivation and ability to process a message are both high, subjects are likely to utilize the central route, evaluating the strength of an argument. However, when motivation and/or ability to process the message are low, subjects are proposed to rely on peripheral cues (eg, attractiveness of the source, expertise of the source, number of arguments presented, reactions of others. . .) or a heuristic to determine the validity of a message. Within this framework, one could hypothesize that OCD patients are likely to be highly motivated to find relief from treatment and that they would therefore be likely to engage in central processing. However, it is also feasible to posit that OCD patients would be anxious meeting with a therapist, and that their anxiety could interfere with their ability to process messages, therefore biasing them to engage peripheral processing. While processing of persuasive messages in OCD has not been tested, a small body of research has examined the influences of anxiety. For example, DeBono and McDermott (1994) found that "trait anxiety may be significantly related to people's ability to process a persuasive message (p. 404)." Specifically, they found that high-trait anxious subjects were persuaded by the attractiveness of the source of information, while low-trait anxious subjects were persuaded by the strength of the argument. Traditionally, emotional arousal is discussed as impairing motivation to critically evaluate messages. However, Jepson and Chaiken (1990) showed that chronic fear also affected ability to process messages. Specifically, they found that subjects with chronic levels of fear engaged in less systematic processing of message content. Given the chronic course of OCD it is reasonable to argue that it represents a chronic fear and may have a similar impact on processing to that demonstrated by Jepson and Chaiken's subjects.

Level of fear or anxiety can interact with expectations in important ways. There is some reason to believe that an anxious OCD patient who is hopeful that the therapist

will provide relief would accept a treatment rationale more readily than a patient who is pessimistic. Gleicher and Petty (1992) found that under conditions of moderate fear, expectations about a proposed solution influenced processing. Specifically, individuals with clear expectations that the proposed solution would work accepted the solution regardless of the argument quality. However, when subjects were unclear in their expectations of whether the proposed solution would work they evaluated the solution based on the quality of the arguments presented. Gleicher and Petty (1992) discuss their results in the context of attempts to obtain reassurance and conclude that subjects appear to be motivated to be reassured and will engage or avoid based on which strategy best allows them to satisfy this goal (p. 97). These conclusions are particularly important given the reassurance seeking and distaste for uncertainty in OCD.

What does the persuasion literature suggest for maximizing engagement of EX/RP? Persuasion is a complicated process, influenced by multiple factors such as motivation, resources, and emotional arousal. Research is needed to examine persuasion in anxious subjects, to delineate the conditions that maximize patient acceptance of EX/RP and specific exposure exercises. However, until this research is completed what can be done? The literature suggests potential for more systematic attention to peripheral routes of persuasion to accept treatment. Because most OCD patients find the prospect of exposure treatment threatening and anxiety provoking, persuasion procedures that make use of peripheral processing merit consideration. In addition to presenting logical arguments, measures to enhance the perceived authority or credibility of the presenter and to present multiple arguments from various sources might be advantageous. Therapists could seek to convey their qualifications via verbal information, written information, and materials in their office such as diplomas or awards. Providing information about the extent of therapist experience with OCD treatment and data about outcome of the treatment could enhance credibility. Instead of focusing extensively on one particular argument, therapists might do well to present multiple lines of support. Multiple sources of information, including some with prima facie credibility, such as patients who have successfully completed EX/RP, or scientific publications, could be more persuasive than a single presentation of a coherent treatment rationale. Therapists can seek to communicate clear expectations that the proposed solution will work under given conditions. Regardless of the specific techniques employed, therapists are encouraged to recognize their persuasive influence on patients (Ojanen, 1996; Strauman, 1998) and to seek to maximize the degree to which these influences can be arranged to facilitate the patient's progress.

Research on decision-making under conditions of uncertainty seems to converge (cf. Simon, 1986) with the literature on persuasion to indicate that the coherence and validity of a treatment rationale is not the only factor that could be important in treatment acceptance. The work on decision-making heuristics (Tversky & Kahneman, 1981) has pointed to variables that account in part for the irrationality of decisions involving risk and valence. For example, negative outcomes receive disproportionately negative weight than positive outcomes of the same magnitude. An apparent implication is that presenting accurate information about the likelihood of a positive outcome to treatment seeker is probably more likely to provoke treatment acceptance than presenting equally accurate information framed as the complementary likelihood of a negative outcome. Emphasizing accurate estimates of positive outcomes might be more effective before and during exposure treatment than emphasizing equally

accurate complementary estimates of negative outcomes. This approach is not strictly "rational," but it could enhance treatment acceptance or completion if employed at various points before and during EX/RP.

The observation that experimental evaluations of EX/RP have been inattentive to potentially active aspects of the procedures is not limited to discussions of risk, consequences, and responsibility. Unleashing the power of exposure entails encouraging the patient to tolerate the distress of the feared confrontations: if the patient declines to do the exposure exercises, EX/RP is impotent. Clinical experience suggests that patient trust in the wisdom and benevolence of the therapist affects whether a patient accepts and completes EX/RP treatment, but the apparently pivotal therapy relationship has received minimal attention in research on EX/RP (cf. Grayson, 1999).

The quality of the therapeutic relationship is one of the most robust available correlates of psychotherapy outcome. Unfortunately, outcome and dismantling studies of EX/RP have emphasized EX/RP parameters, with inattention to the working alliance. However, decades of research point to the importance of the therapy relationship (Horvath, 2001; Lambert & Barley, 2001). For example, in reviewing over 100 studies examining predictors of outcome Lambert and Barley (2001) found that common factors, including the client–therapist relationship, accounted for 30% of the variance in outcome. On the basis of a review of over 2000 process-outcome studies, Orlinsky, Grave, and Parks (1994) identified several therapist variables related to client outcomes. The importance of the therapeutic relationship has been documented for cognitive-behavioral therapy. For example, Williams and Chambless (1990) found that patients with agoraphobia who rated their therapists as more caring, involved, and self-confident, were significantly more likely to improve from exposure therapy. Recently, Klein et al. (2003) provided support for the causal impact of alliance on outcomes from cognitive-behavioral therapy. Controlling for prior and concurrent symptom levels, comorbidity, and numerous patient characteristics. They showed that therapeutic alliance early in therapy predicted depression changes. Although the large contributions of exposure and abstinence from rituals have been clearly established, there remains unexplained variance and the problem of EX/RP treatment refusals. Given the findings about the importance of therapeutic alliance, it would be surprising if more systematic attention to relationship factors could not enhance the potency of EX/RP.

What can therapists do to develop and maintain a strong therapeutic relationship? Initially, we believe that recognizing the importance of the relationship serves as an important foundation. Ideally, cognitive-behavioral therapists can seek to balance attention to the therapeutic relationship and specific therapeutic techniques. Beyond this, therapists can work to be open and flexible throughout therapy. Indeed, much empirical evidence demonstrates the benefits of an open, flexible stance, in contrast to rigid expectations or taking charge of sessions (Horvath, 2001). Therefore, therapists should seek to respond flexibly to patients, incorporating their observations and comments into the ongoing process. In addition, therapists should seek to be respectful of clients and their viewpoints, remembering that rejecting or negative interactions are particularly hurtful to therapeutic alliance and thereby, therapeutic outcomes (Horvath, 2001). Within these broad parameters, therapists could do well to modify the nature of their relationships to meet the specific needs of each client (see Wright & Davis, 1994, on therapeutic relationships in cognitive-behavioral therapy).

Certain patient variables might be important in relation to treatment acceptability. For example, work on readiness to change (eg, Prochaska, 1994) might suggest ways to match persuasion methods to the inclinations of the patient to make use of information presented: the readiness research indicates that "timing" matters, and perhaps assessments of readiness can be used to inform the timing and type of information provided to the individual in need Alternatively, work on individual differences in threat-information processing style (eg, Miller, Fang, Diefenbach, & Bales, 2001) that has been applied to facilitate decision making about treatment for cancer might be extended to enhance acceptance and adherence to EX/RP regimens.

Some obsessive-compulsive foci can appear to present special problems for which special persuasive effort might enhance the outcome of EX/RP. These include hoarding, certain types religious obsessions, and delusions. In the first two of these, the patient often demonstrates a clear commitment to certain aspects of obsessive-compulsive patterns, and in the third, adheres strongly to a patently false belief. The accumulation of items that occurs in pathological hoarding, for example, can be appetitive: the popularity of "collecting" hobbies attests to the appetitive nature of certain kinds of hoarding. Because collecting is socially acceptable and can be satisfying, pathological hoarders sometimes come very late, or not at all, to clear insight about the costs of this activity. Often, they are brought for treatment by family members whose threshold of intolerance for the costs is lower than that of the hoarder. Perhaps systematic psychoeducation that specifically highlights the negative personal and interpersonal consequences of hoarding increases the likelihood of EX/RP acceptance and completion (cf. Frost & Steketee, 1999).

Some obsessions are found in religious tenets that garner substantial social support and that are integrated into the patient's *Weltanschaung* in a way that makes dislodging these theological impediments an impractical approach to treatment. When religious devotion inspires rejection of EX/RP for pathological obsessions of a religious nature, special efforts toward persuasion might be helpful. Indirect approaches, as suggested above, might be particularly useful. For example, rather than offering arguments against the religious tenets, it might be more helpful to arrange social support from a respected religious authority, or from other members of the patient's religious denomination. If the patient can be persuaded to accept the treatment, exposure exercises can be developed for all manner of supernatural fears, including those involving eternal damnation.

An area ripe for research is the development of transportable packages of persuasive material that could be made available to clinicians and their patients. What might be the form of such a package? One could imagine a convenient set of audio–video modules that could be matched to individual symptoms, age and cultural background of the treatment seeker, information processing style, and readiness to change. The credibility of the presentation might be enhanced by including well-credentialed and articulate experts in the psychopathology and treatment of OCD, as well as individuals with OCD who have had experience with the treatments being considered. The *imprimatura* of professional or consumer groups might further enhance the credibility of the presentation. Favorable use of decision-making heuristics could be employed, with attention to the likelihood of positive outcomes from treatment acceptance, and the likelihood of negative outcomes from treatment avoidance. Demonstrations of fear-relevant exposure exercises by successfully coping patients could be included. The package could first be presented when the potential therapist is available to

answer questions and demonstrate expertise. It could then be made available for repeated review by the treatment seeker, so that there would be ample time for prolonged exposure to the information, and so that habituation of fear associated with threat related information, and for learning and persuasion to occur. The goal would not be to automate the presentation simply to reduce expensive interpersonal contact with the therapist, but rather to introduce a standard set of materials of established quality and efficacy, which could amplify the effects of EX/RP by increasing treatment acceptance.

CONCLUSION

Exposure-based treatment has proven to be a very powerful technique for fear reduction, and when combined with abstinence from ritualizing (aka, response prevention), is a treatment that has been found more effective for OCD than any available alternative. It is arguable whether the Achilles Heel of EX/RP is treatment refusal or the limited availability of such treatment by knowledgeable practitioners. Nonetheless, it is clear that a nontrivial proportion of patients decline to accept EX/RP treatment when it is available. Because pharmacotherapy by SRI medication is of documented efficacy, is more generally available than EX/RP, and is more acceptable to some patients, it is an important and valuable alternative to EX/RP (Greist et al., 1995b). The comparative efficacy of cognitive techniques for OCD remains unclear: CT for OCD seems typically to involve behavioral experiments that resemble the exposure exercises of EX/RP, and when "purified" of exposure-like elements, it compares unfavorably to optimized EX/RP. On the other hand, EX/RP consists of more than its namesake elements, and persuading the patient to accept the treatment is prerequisite to its success. Many factors, including CT techniques, are likely candidates for causal factors in this persuasion. More systematic attention to the mechanisms of persuasion, including research on putative persuasive factors in EX/RP holds some promise to yield methods that can enhance the efficacy of this powerful method.

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Reply to Kozak and Coles:

EXPANDING THE CONCEPTUALIZATION OF COGNITIVE THERAPY AND ITS THERAPEUTIC POTENTIAL

Jeanne Fama and Sabine Wilhelm

Kozak and Coles' thoughtful review of the exposure and response prevention (EX/RP) treatment literature helps refine expectations about the variance in symptom reduction that accompanies EX/RP under circumstances that vary according to therapist training (eg, cognitive-behavior therapy [CBT] trained therapist versus generalist therapist versus self-guided exposure); treatment intensity (eg, massed, 90 min sessions, several times per week versus 1 h weekly sessions); and patient variables (eg, "responders" versus nonresponders). We agree with Kozak and Coles that, although not efficacious for all OCD patients, EX/RP is undoubtedly a highly potent treatment for OCD. We also agree that the delivery of suboptimal EX/RP along with high refusal, noncompliance, and drop out rates attenuate EX/RPs potential for a substantial number of OCD sufferers, some of whom might otherwise benefit greatly from EX/RP. Efforts to improve EX/RP delivery, adherence, and compliance via decision-making strategies could prove very useful and await empirical investigation. Efforts to do so via cognitive techniques are likely very promising; however, we disagree with Kozak and Coles' interpretation of the potential utility of CT alone, namely, that OCD patients generally can expect considerably less improvement from CT than from the best available EX/RP.

Kozak and Coles state that CT alone is as efficacious as is "substandard" exposure, citing Abramowitz, Franklin, and Foa's (2002) meta-analytic review of CT and EX/RP treatment studies. Support for this statement is based on two observations. First, CT versus EX/RP comparisons that deem CT and EX/RP equally efficacious, often include studies employing CT with behavioral experiments, which some consider a form of exposure, one of the core elements of EX/RP (Abramowitz et al., 2002). Second, such comparisons also employ "substandard" EX/RP that diverges, to varying degrees, from optimal therapist-guided, prolonged sessions conducted several times a week. However, playing devil's advocate, one could argue that many treatment

studies employ EX/RP that includes core elements of CT (eg, discussions of risk, consequences, and uncertainty), the omission of which may diminish EX/RP's efficacy (Abramowitz et al., 2002). Furthermore, some might argue that the CT versus EX/RP comparisons studies are obfuscated by a relative lack of CT research (Abramowitz et al., 2002), and may be distorted because they have included not only suboptimal EX/RP, but suboptimal CT as well. Indeed, unlike Kozak and Coles, Abramowitz et al. acknowledge several limitations in the extant literature that preclude "definitive conclusions" about the differential efficacy of CT and EX/RP. Conclusions that have been drawn about the differential efficacy of CT and EX/RP are premature and therefore potentially misleading. As we discuss below, such conclusions are flawed partly because of the overlap between CT and EX/RP and partly because of the paucity of research directly comparing the effects of standardized protocols for EX/RP and contemporary CT, as measured by uniform outcome measures.

PROCEDURAL OVERLAP

The overlap in CT and EX/RP procedures has been discussed in this volume and elsewhere, and their potentially overlapping mechanisms of change is receiving increased attention (Abramowitz, 1997). As Kozak and Coles point out, some reports suggest that a substantial portion of EX/RP's success is mediated by exposure-induced modification of fear structures (eg, Foa & Kozak, 1986). However, as Kozak and Coles also note, some of the variance in EX/RP efficacy remains unexplained. Indeed, some of this variance may be attributable to changes in beliefs, as demonstrated by research documenting that some belief change may follow EX/RP (Emmelkamp & Beens, 1991; McLean et al., 2001). Similarly, research suggests that cognitive change may act as a mediator in the relationship between successful CT and decreased severity of distorted beliefs (Wilhelm, Steketee, Fama, & Golan, 2003). However, it is possible that such change may reflect the modification of fear structures thought to underlie EX/RP-induced change. That is, CT, like EX/RP, may work partly by modifying fear structures evoked, not through exposure per se, but through discussion of distorted beliefs about anticipated consequences.

RESEARCH ON OPTIMAL CT AND OPTIMAL EX/RP

It is likely that experimental comparisons between CT and EX/RP are somewhat distorted, quite simply, because of the paucity of well-controlled research on CT and EX/RP. For example, Abramowitz et al. (2002) indirect comparison of no-treatment-control conditions versus EX/RP and CT, respectively, ostensibly favors EX/RP: Abramowitz et al. found that the across-study, composite EX/RP versus no-treatment-control effect size was larger than was the composite CT versus no-treatment-control effect size. However, as Abramowitz et al. rightly point out, this comparison is potentially unreliable given that the CT effect size estimate was based on only two CT studies. When Abramowitz et al. examined the five studies that directly compared CT (with and without behavioral experiments) and EX/RP (likely, with and without discussions of risk, etc.) they found both treatments to be equally

efficacious. Although Kozak and Coles rightly question the interpretation of studies employing suboptimal EX/RP, they do not acknowledge that many such studies have quite possibly employed suboptimal versions of CT as well.

Kozak and Coles provide a somewhat limited description of CT, mentioning just two techniques: Socratic questioning and behavioral experiments. Although they note that contemporary CT derives from the work of OCD researchers such as Salkovskis (1985, 1989) and Rachman (1993, 1997), some of the studies they review employed early or somewhat unconventional versions of CT that may be viewed as substandard relative to contemporary CT protocols derived from these and other increasingly refined theories regarding the cognitive underpinnings of the wide range of OCD symptoms (Obsessive Compulsive Cognitions Working Group [OCCWG], 1997; Wilhelm, 2001; Wilhelm et al., 2003, in press).

For example, Kozak and Coles cite Emmelkamp and colleagues (Emmelkamp & Beens, 1991; Emmelkamp, Visser, & Hoekstra, 1988), who conducted two seminal studies on CT for OCD from which they concluded that CT and EX/RP were equally efficacious. We agree with Kozak and Coles that the EX/RP employed in these studies was likely was substandard in that it relied on self-controlled exposures. However, the nonspecific CT employed in these studies derived from Ellis' (1962) RET and did not target several of the cognitive belief domains considered by leading cognitive researchers such as the OCCWG (1997) to be integral to the understanding and treatment of OCD. Hence, by contemporary standards, such CT would likely be regarded as substandard. The purity of the CT administered in Emmelkamp and colleagues' studies has been questioned given that some CT patients engaged in exposure exercises on their own. However, the EX/RP administered could be questioned on similar grounds: EX/RP patients likely engaged in some form of cognitive restructuring on their own, as they demonstrated some degree of posttreatment belief change in one of the studies (Emmelkamp & Beens, 1991).

Kozak and Coles also cite van Oppen et al. study, which showed that CT was at least as effective, and possibly more so than EX/RP. Van Oppen et al. (1995) methods have also been questioned on the basis that they too employed self-controlled EX/RP. However, it should be noted that CT in that study employed mainly targeted just two of the six OCCWG belief domains. Thus, both forms of treatment were likely administered "suboptimally." This notwithstanding, results showed that 6 weeks of pure CT (without behavioral experiments) was as efficacious as 6 weeks of pure EX/RP (without discussions of risk and consequences). Hence, Kozak and Coles appear unjustified inferring that a substantial portion of CT's success may have been attributable to behavioral experiments added to CT in the later weeks of treatment and that CT without behavioral experiments may be of "limited potency."

Kozak and Coles cite Jones and Menzies (1998) study on Danger Ideation Reduction Therapy (DIRT) as evidence for the relatively limited effects of CT. Jones and Menzies studied the efficacy of DIRT in a group of 11 OCD patients with washing concerns. To characterize DIRT as optimal CT would be misleading for several reasons. First, the DIRT protocol comprised six components (eg, filmed interviews, corrective information, microbiological experiments with discussion of excessive risk expectancies, etc.), just one of which was an RET/cognitive restructuring component, derived from Mattick, Peters, Clarke and Christopher (1989) and Menzies and Clarke (1989), and one of which targeted patients' unrealistic probability estimates of risk. Second, given DIRT's emphasis on decreasing unrealistic estimates of dangerous

outcomes, it ignores most OCCWG belief domains (eg, responsibility). DIRT mainly addresses just one OCCWG domain (threat/risk), a domain that is also targeted in EX/RP if given optimally (Abramowitz et al., 2002). Third, DIRT patients received only 8 h of treatment . This would be considered suboptimal by both CT and EX/RP standards. Fourth, DIRT was delivered in group format and there is cause to suspect that because of the heterogeneity of cognitive distortions encountered in the population of OCD patients, group CT may not be the optimal format in which to undertake cognitive restructuring (Jones & Menzies, 1998; McLean et al., 2001). Finally, because the Jones and Menzies study treated only washers, we are unable to generalize from these patients to the great number of OCD patients with nonwashing symptoms. Hence, DIRT may decrease OCD symptoms by approximately 20% in OCD washers who undergo 8 h of group treatment. However, it seems unwise to extrapolate from this the extent to which the population of OCD patients would benefit from an adequate trial of individually administered, contemporarily informed CT.

Given the lack of data that would enable us to make accurate differential comparisons between optimal CT and EX/RP, it appears unjustified to characterize CT as considerably less efficacious than EX/RP. As is apparent from a review of the CT treatment literature, CT is still evolving. Research on contemporary CT techniques (eg, Wilhelm et al., 2003, in press) suggest that these may be quite powerful. What will ultimately constitute optimal CT should depend on what future research shows to be the most effective components of CT.

Optimal EX/RP is undeniably efficacious, but several factors detract from its potency. The use of decision-making techniques and cognitive techniques as EX/RP aids may partly counteract the high drop out, refusal, and noncompliance limiting EX/RP's potency. Kozak and Coles review some of the decision-making literature and provide suggestions about how we may draw from it to persuade patients to engage in EX/RP. On the other hand, elaborate persuasion techniques are not needed to convince patients to undertake CT, an efficacious treatment in its own right. Moreover, it is unlikely that decision-making techniques will counteract all factors impinging upon EX/RP's optimal delivery (eg, the shortage of available EX/RP trained therapists needed to disseminate optimal massed, prolonged exposure sessions). In our experience, such factors do not detract from CT's efficacy or "real world" effectiveness. Characterizing CT as a treatment of limited potency may curtail the very research needed to ascertain which protocols would be most optimal for the wide range of treatment-seeking OCD patients. Furthermore, characterizing CT as a substandard treatment option seems unwise given that many of the factors detracting from EX/RP's effectiveness (eg, poor compliance, lack of trained therapists, inadequate funds for time intensive treatments) have not been demonstrated to interfere with CT. Cognitive therapy techniques may well add to EX/RP's potency. However, it seems imprudent to relegate CT to an augmentation strategy, when research is increasingly demonstrating it to be an efficacious treatment in its own right. Indeed, recent research suggests that when CT is administered optimally, symptom reduction rates are in the range of those found with EX/RP (Wilhelm et al., 2003, in press). Continued research on the various components of CT, CBT, and EX/RP conducted in various patients subgroups, in various treatment settings, is needed before blanket statements can be made about comparative potency. More importantly, such research is needed to identify the symptoms-subtypes, comorbidity, treatment preferences, and so forth of those

patients who would benefit most from EX/RP, those who would benefit most from CT, and those who might benefit most from flexible, individually tailored, modular treatments comprising both CT and EX/RP components.

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Reply to Fama and Wilhelm:

COGNITIVE THERAPY AND EXPOSURE TREATMENT FOR OCD:CONTRAST AND RAPPROCHEMENT

Michael J. Kozak and Meredith E. Coles

Two chapters in this volume evaluate the status of cognitive therapy and exposure and response (ritual) prevention (EX/RP) as treatments for obsessive-compulsive disorder, and offer different perspectives on their potential. This reply compares our interpretations and conclusions with those of Fama and Wilhelm to highlight similarities and differences in our claims.

The two chapters appear to diverge more in interpretation and emphasis than in claims about facts. For example, the chapters converge in an assumption that OCD involves erroneous concepts of threat that must be modified in the treatment of the disorder. However, citing Foa and Kozak (1986) in support of this assumption, Fama and Wilhelm do not engage Foa and Kozak's suggestion that low susceptibility to corrective information presented in a rational manner in part defines the pathology of OCD. Inattention to this hypothesis illustrates a divergence between the chapters in their optimism about the extent to which variants of rational discourse can suffice to modify certain pathological concepts of OCD. The formal cognitive therapy techniques described by Fama and Wilhelm can be construed as variants of rational discourse and encompass role play, graphic illustration of responsibility attribution, instruction in the normality of unwanted intrusions, guided reevaluation of beliefs about the immorality of intrusive thoughts, behavioral experiments, guided estimation of the contingent probabilities of feared events, and guided consideration of the expected values of various good and bad fear-relevant events. Our own chapter focuses on the parameters of exposure, and sees it as a better-established method of pathological fear reduction than the panoply of cognitive therapy techniques.

The chapters also converge in their conclusion that studies of cognitive therapy for OCD support its efficacy for OCD, but differ in their emphases. Fama and Wilhelm interpret published findings that cognitive therapy reduces OCD symptoms to indicate its efficacy. Our own analysis also reviews the outcome literature and acknowledges that cognitive therapy can reduce symptoms, but interprets the evidence

to indicate that exposure-based treatment is more potent than cognitive therapy. Fama and Wilhelm do not tout the superiority of exposure to cognitive therapy, perhaps because they do not interpret the available evidence to indicate such superiority. The argument for the superiority of exposure is elaborated at length in our chapter, and therefore will not be reiterated here. Although Fama and Wilhelm acknowledge that work on cognitive therapy is in its early stages, their conclusion "that cognitive therapy is an effective and pragmatic treatment for many OCD patients" seems more straightforward and emphatic than our qualified interpretation that emphasizes relative efficacy and concludes that cognitive therapy has been found as potent as suboptimal exposure treatment.

How might the two chapters interpret the relative efficacy of cognitive therapy and exposure differently? Some of the difference between the two chapters seems to lie in their differing views of the evidence for the mechanism of action of exposure therapy, and in the potential contribution of exposure to the efficacy of cognitive therapy procedures. Our analysis gives substantial weight to the weaker outcomes of cognitive procedures that exclude behavioral experiments than of those that do not, arguing that (*a*) behavioral experiments resemble exposure procedures, and (*b*) cognitive therapies that exclude behavioral experiments do not compare well to exposure procedures. Fama and Wilhelm seem to weigh these weaker outcomes less heavily, and to emphasize the comparisons that show equivalent efficacy, which we discount on the grounds that they compare suboptimal exposure therapy to cognitive therapy that includes behavioral experiments that resemble exposure exercises.

Assumptions about the nature and mechanisms of action of behavioral experiments are debatable, and constitute points of divergence between the two chapters. We argue that behavioral experiments resemble exposure exercises of established potency, and suspect that they operate like exposure exercises. They point to weaker outcomes of cognitive therapies that do not include behavioral experiments. Fama and Wilhelm distinguish behavioral experiments of cognitive therapy from prolonged exposure on the basis of the brevity of the behavioral experiments that are "usually brief, and used only to test patients' maladaptive predictions against other, more rational predictions."

The concept of functional exposure complicates Fama and Wilhelm's brevity distinction: the effective length of the exposure depends upon the length of time that the patient focuses on the exposure event without undoing it in some way, rather than simply on the duration of the initial confrontation with the stimulus. Accordingly, a patient painted with a contaminant might not be "functionally" exposed if the patient does not think about the event, which would be tantamount to actually undoing the exposure. On the other hand, an extremely brief stimulus event can function as a very long exposure if the patient dwells on it for a long time and does not undo the event. The implications for understanding "brief" behavioral experiments are clear: although a behavioral experimental confrontation itself might be temporally brief, the functional exposure could be prolonged if the patient does not undo the stimulus event. The descriptions of behavioral experiments in reports of outcome of cognitive therapy do not provide enough detail to permit resolution of questions about the extent of associated functional exposure. Because of the frequent inclusion of exposure-like behavioral experiments in cognitive therapy regimens, and the ambiguity about the extent of the associated functional exposure, it is reasonable to suspect that many cognitive therapy studies include important exposure components whose contributions

have not been disentangled from those of more reason-based procedures. Indeed, incorporation of exposure-like behavioral experiments seems necessary for cognitive therapy to approach the efficacy of suboptimal exposure-based treatment.

Although Fama and Wilhelm's discussion of the thought suppression test component of cognitive therapy for OCD is neither convergent nor divergent with our chapter, it merits some comment in relation to the techniques of exposure therapy and the above-mentioned issue of functional exposure. As described in their chapter, the thought suppression test is a demonstration exercise that requires the patient to practice thinking and not thinking about an affectively neutral stimulus, and to record the frequency of thoughts about the stimulus under each of the two practice conditions. The purpose of this exercise is to demonstrate that attempts at thought suppression are counterproductive.

Discussion of the counterintuitive effects of attempts to avoid obsessive intrusions are routine in exposure-based treatment, but are accompanied by instructions to entertain fearful obsessive intrusions for prolonged periods. This is a clear instruction to confront the feared thought rather than to avoid or undo it via rituals. It would seem that the therapeutic effect of the neutral thought suppression demonstration described by Fama and Wilhelm would depend upon application of the relevant principles by the patient to obsessive intrusions: if the patient does not abandon attempts to avoid or escape obsessive intrusions via thought suppression, one would expect no effect of the thought suppression demonstration. Notably, however, Fama and Wilhelm's discussion of cognitive therapy describes no systematic efforts to persuade the patient to apply the thought suppression principle to obsessive intrusions. It is not clear why the logic of this application would be left implied and unexplicated by the cognitive therapist, leaving the patient to guess how to generalize the thought suppression principle to the problem of obsessive intrusions. Presumably, some patients who have experienced the thought suppression demonstration do discover the intended application, with or without explicit instruction from the cognitive therapist. If these patients practice confronting obsessive thoughts, rather than trying to suppress them, they would seem to be doing the kind of feared confrontation that is routinely prescribed in exposure treatment, but without the neutral thought suppression test. If this is true, then putative action of the thought suppression test in cognitive therapy might depend on an exposure component. Accordingly, the neutral thought suppression demonstration might be a useful tactic to persuade patients to confront obsessive intrusions.

Whereas we maintain that activation of fear and its habituation during exposure are among the most well-established mechanisms of psychological treatment, Fama and Wilhelm note that "the extent to which CT and ERP work via these alleged mechanisms is unclear" owing to "the absence of studies employing research methods to adequately assess mechanisms of change." Although the chapters agree that the available evidence does not resolve the extent to which CT versus exposure depend upon fear activation and habituation of associated physiological responding, we seem much more confident about the importance of certain hypothesized mechanisms of exposure than are Fama and Wilhelm. We reiterate Foa and Kozak's (1986) well-supported hypotheses that fear activation and physiological habituation are fundamental to emotional processing of fear via exposure for a variety of anxiety disorders, including OCD. We admit to ambiguity about the extent of the variance of outcome prediction accounted for by fear activation and habituation, but assert

that the involvement of the hypothesized mechanisms has been clearly established for exposure treatment. It would not be logical to discount the strong evidence for involvement of fear activation and habituation in exposure on the grounds that the mechanisms of cognitive therapy have not been established, or on the grounds that the parameters of extent have not been precisely ascertained.

In their review of studies comparing cognitive therapy and exposure for OCD, Fama and Wilhelm attend carefully to details of the exposure procedures that complicate interpretation of findings about the efficacy of cognitive therapy vis-a-vis EX/RP. The exposure procedures studied variously involved reliance on self-directed exposure, brief exposure sessions, and spaced, rather than massed, exposure sessions. The best results for exposure treatment have been obtained with therapist-guided exposure in massed sessions of long duration. In accord with Fama and Wilhelm's interest in particular cognitive hypotheses, these authors show more interest than we do in several small cognitive therapy effects that do not achieve significance. We weigh these various limitations more heavily than do Fama and Wilhelm, and to a less favorable view about the efficacy of cognitive therapy, especially in comparison to optimal regimens of EX/RP.

There are two apparent domains in which Fama and Wilhelm appear to argue for the superiority of cognitive therapy: decreasing irrational thoughts and decreasing comorbid conditions. The clinical importance of the cited findings on irrational thoughts is not clear, in that the causal relationship with overall symptom reduction has not been established. Fama and Wilhelm's suggestion that cognitive therapy might be superior to EX/RP for reducing comorbid symptoms, and in particular, depression, is provocative, and we do not address this question in our chapter. It is notable that EX/RP has been found superior to pill placebo and similar to the antidepressant imipramine in reducing OCD with comorbid mild-to-moderate depression (Foa, Kozak, Steketee, & Mccarthy, 1992). It is also notable that the studies cited by Fama and Wilhelm do not strongly support a conclusion of the superiority of cognitive therapy. For example, the means and change scores in Cottraux et al. (2001) seem to indicate that the two treatment groups have similar depression at follow-up, and the van Oppen et al. (1995) study did not yield significant differences in depression outcome between cognitive therapy and EX/RP when initial depression levels were taken into account. It is probably premature to draw conclusions about the relative efficacy of cognitive therapy and EX/RP for comorbid symptoms.

Fama and Wilhelm argue that EX/RP is difficult to administer, and that because of the demanding nature of the therapy, many candidates decline to participate. The chapters converge in their acknowledgment that the effectiveness of EX/RP is substantially diminished by treatment refusal. Our chapter focuses on potential methods to increase the effectiveness of EX/RP, whereas Fama and Wilhelm focus on an alternative to EX/RP. Fama and Wilhelm assert that it is difficult to administer EX/RP, and that insufficient numbers of trained therapists are available. It seems prudent not to overinterpret Fama and Wilhelm's survey of the percentage of training programs offering EX/RP for OCD to that of programs providing training in Beck's cognitive therapy for depression. A survey of training in exposure therapy generally, or of cognitive therapy for OCD specifically, might yield a different picture.

Critics have sometimes suggested that behavior therapy offers little intellectual challenge or opportunity for creativity compared to more traditional psychotherapy methods focused on intrapsychic conflicts and interpersonal relationships. This

suggestion rings ironic in light of Fama and Wilhelm's observation that EX/RP is difficult to administer by a scarcity of skilled providers. Although Fama and Wilhelm hope that cognitive therapy might help address the insufficient numbers of expert EX/RP providers, it is not at all clear that optimal cognitive therapy requires less skill and experience with the OCD population than does optimal EX/RP, or that it is any more convenient or efficient to train expert cognitive therapists than expert providers of EX/RP. The techniques of EX/RP are well worth learning, and well worth providing, even if their mastery requires courage, dedication, creativity, and supervised practice.

A striking point of convergence between the two chapters is the proposal that cognitive therapy techniques could constitute one element in a technology of persuasion that might increase the effectiveness of EX/RP. For example, Fama and Wilhelm suspect that cognitive therapy might enhance treatment acceptance and adherence, and might afford a successful augmentation strategy". A central motif of our chapter is that EX/RP is highly potent for those who accept and complete it, but that there is a need for techniques that enhance its acceptability for treatment refusers. The two chapters converge in their conclusions that it would be worthwhile to explore the efficacy of cognitive therapy techniques for increasing treatment acceptance and compliance.

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Chapter 16

THE ROLE OF THE THERAPIST IN BEHAVIOR THERAPY FOR OCD

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BEHAVIOR THERAPY FOR OCD

Cognitive-behavioral interventions are considered a first-line treatment of choice for OCD (Franklin & Foa, 1998; March, Frances, Carpenter, & Kahn, 1997). Although cognitive-behavioral therapy has many forms, the strongest evidence base is for exposure and response prevention (ERP). Exposure and response prevention consists of gradual, prolonged exposure to fear-eliciting stimuli or situations, combined with strict abstinence from compulsive behavior. In practice, this means that a patient with contamination concerns would be encouraged to touch progressively more contaminated objects while simultaneously refraining from washing or cleaning. Similarly, a patient with obsessive concerns about harming other people while driving might be encouraged to drive in increasingly congested areas without looking in the rear-view mirror. The purpose of exposure exercises is to allow the patient to experience a reduction of their fear response, recognize that these situations are not excessively dangerous, and learn that their fear will not persist indefinitely. Thus, ERP's mechanisms of action may include modification of maladaptive cognitions as well as the "behavioral" mechanisms of habituation and extinction (Foa & Kozak, 1986).

The "classic" model of ERP involves daily sessions (Kozak, Liebowitz, & Foa, 2000), although researchers have tested less intensive forms of treatment delivery such as twice-weekly sessions (Abramowitz, Foa, & Franklin, 2003) and group therapy (McLean et al., 2001). Numerous studies attest to the efficacy of ERP in adults (eg, Cottraux, Mollard, Bouvard, & Marks, 1993; Fals-Stewart, Marks, & Schafer, 1993; Kozak et al., 2000; Lindsay, Crino, & Andrews, 1997; van Balkom et al., 1998) and in children and adolescents (eg, Benazon, Ager, & Rosenberg, 2002; de Haan, Hoogduin, Buitelaar, & Keijsers, 1998; Franklin et al., 1998; March, Mulle, & Herbel, 1994; Thienemann, Martin, Cregger, Thompson, & Dyer-Friedman, 2001; Wever & Rey, 1997). Approximately 75% of patients treated with ERP improve significantly, usually defined as 30–50% improvement, and remain so at follow-up (Franklin & Foa, 1998).

LIMITATIONS OF BEHAVIOR THERAPY

Despite its documented efficacy, most OCD patients never receive this treatment. One likely reason for this is the fact that there are relatively few professionals who have the specialized training needed for competent treatment administration. Thus, ERP is difficult to obtain. Illustrative of this problem, the 1988–1989 American Psychiatrist Activities Survey (American Psychiatric Association, 1989) revealed that the vast majority of psychiatrists (90%) do not practice behavior therapy. Even in specialty anxiety treatment centers, only 28% of OCD patients are treated using exposure-based techniques (Goisman et al., 1993). Specialized training in ERP is not a component of most graduate programs (Crits-Christoph, Chambless, Frank, Brody, & Karp, 1995; Davison, 1998), causing resources for treatment with ERP to be limited. Therefore, mental health service providers may choose not to administer this treatment due to their lack of competence with this approach. Another possible explanation for the underutilization of ERP is the perception that this treatment is not cost-effective.

As is evident from our description of ERP, the treatment sessions are time consuming, requiring an estimated 30 h of direct clinician time, and expensive (in the short term), with a 1995 survey showing an average cost of $4370 (Turner, Beidel, Spaulding, & Brown, 1995). Although behavior therapy is less expensive over time than are longer-term psychotherapy and medications (Otto, Pollack, & Maki, 2000), this still represents a considerable expense. Even when cost is not an option (eg, in clinical research trials), approximately 25% of OCD patients still refuse ERP (Franklin & Foa, 1998), presumably because of apprehension about the difficulty and intensity of the treatment. Thus, although ERP is clearly efficacious, several obstacles prevent the majority of patients from receiving this treatment.

THE POTENTIAL ROLE OF SELF-ADMINISTERED TREATMENT

Given the current obstacles to therapist-administered ERP, it might be argued that self-administered ERP programs should be explored for those patients who cannot or will not receive the full treatment. Self-help programs have existed for decades, perhaps reaching an apex in the 1970s and 1980s (Glasgow & Rosen, 1978, 1984). Resources containing self-help programs can be found in most bookstores, and with the increasing popularity of the Internet, they are even easier to access. Self-help programs are usually low-cost (often no more than the price of a book), and therefore accessible to persons with low incomes who could not afford behavior therapy.

Self-administered treatment programs have been found to be superior to no treatment, wait list, or placebo treatment in the areas of assertiveness training (Rakos & Schroeder, 1979), smoking cessation (Curry, Ludman, & McClure, 2003), binge eating disorder (Peterson et al., 1998), insomnia (Riedel, Lichstein, & Dwyer, 1995), and chronic headache (Larsson, Daleflod, Håkansson, & Melin, 1987). Within the anxiety disorders, self-administered exposure-based programs have been shown to be superior to no treatment for specific phobias (Moss & Arend, 1977; Rosen, Glasgow, & Barrera, 1976), public speaking anxiety (Marshall, Presse, & Andrews, 1976), and panic disorder (Gould, Clum, & Shapiro, 1993; Lidren et al., 1994). There have been comparatively few controlled assessments of self-administered OCD treatment. Fritzler

et al. (1997) compared partially self-administered treatment (a self-help book plus five sessions of therapist contact to supplement the readings) to wait-list. Treated patients showed a superior outcome, although only 25% met criteria for clinically significant improvement. Another partially self-driven ERP program is the BT-STEPS program (Baer & Greist, 1997), in which instructions for conducting self-administered ERP therapy are delivered via a computerized telephone administration system. Although BT-STEPS is not purely self-directed (exposure instructions are determined by the computer based on a decision-making algorithm using the patient's anxiety ratings), there are no in-person meetings between the patient and a therapist. Open trials found this treatment to be both acceptable to, and clinically effective for, patients with OCD (Bachofen et al., 1999; Baer & Greist, 1997). BT-STEPS was also superior to relaxation training, with 38% versus 14% of patients considered treatment responders, respectively (Greist et al., 2002).

COMPARISONS BETWEEN SELF-ADMINISTERED AND THERAPIST-ADMINISTERED TREATMENT

As reviewed above, the available evidence suggests that self-administered ERP may be superior to no treatment or to a placebo treatment. However, other evidence suggests that patients receiving self-administered treatment do not fare as well as those who receive treatment from a therapist. In a meta-analysis of therapist-administered ERP for OCD, patients whose exposures were therapist-controlled (eg, the therapist led the patient through exposure exercises) showed a significantly greater decrease in OCD symptoms and general anxiety than did those whose exposures were self-controlled (eg, the therapist provided a list of exposures that the patient completed on his/her own; Abramowitz, 1996). Although none of the sampled studies investigated purely self-administered treatment, among therapist-administered treatments clinical outcome was linked to degree of therapist involvement. Because these meta-analytic results were not based on studies in which patients were randomly assigned to treatment conditions, they may not correspond to the outcome of direct treatment comparisons (LeLorier, Gregoire, Benhaddad, Lapierre, & Derderian, 1997). A clearer understanding of the comparative efficacy of therapist-administered and self-administered treatment is derived, therefore, from randomized controlled trials.

Self-administered treatment has been compared to therapist-administered treatment for anxiety disorders other than OCD. Earlier studies of specific phobias failed to show significant differences between self-treated and therapist-treated patients (Moss & Arend, 1977; Rosen et al., 1976); however, in a more recent study, only 10% of patients receiving home-based exposure manuals showed clinically significant improvement, compared to 80% of patients receiving a single session of therapist-directed exposure (Hellström & Öst, 1995). A study of undergraduates with public speaking anxiety found that neither self-administered relaxation nor therapist-administered imaginal exposure therapy led to decreases in behavioral manifestations of anxiety, although both treatments were superior to no treatment and placebo treatment in terms of subjective anxiety. A higher discontinuation rate (45% versus 18%) was noted among patients in the self-administered treatment group compared to those in the therapist-administered treatment group (Marshall et al., 1976). Self-administered

treatment for panic disorder was shown to yield comparable effects to therapist-administered coping skills training (Gould et al., 1993) and to therapist-administered behavior therapy incorporating exposure (Hecker, Losee, Fritzler, & Fink, 1996). Intriguingly, in the former study, 73% of self-administered treatment patients, versus 67% of therapist-administered treatment patients, met criteria for clinically significant improvement at posttreatment. Similarly, in the latter study, 40% of patients receiving self-administered treatment, compared to 29% of patients who received therapist-directed treatment, met criteria for good end-state functioning. In the latter study (Hecker et al., 1996), however, we note that the "self-administered treatment" patients had three sessions with a therapist to supplement their use of the self-help manual, and this may have influenced the results. A study of agoraphobic patients found no differences in outcome between self-administered and therapist-administered treatment (Ghosh & Marks, 1987); however, all patients received detailed psychoeducation and preliminary instructions from a therapist, and the "therapist-administered" treatment was largely self-controlled with some instructions from the therapist. Thus, the treatments may have been too similar to represent a true comparison of self-administered and therapist-administered treatment.

To date, there have been few comparisons of self- versus therapist-administered treatment for OCD. Emmelkamp and Kraanen (1977) found no difference in outcome between "therapist-controlled" and "self-controlled" ERP; however, "self-controlled" treatment was still directed by the therapist during ten 1-h office visits. The difference between treatments was that the therapist was not physically present during the exposure exercises in self-controlled ERP. In an augmentation study in which all patients received the serotonin reuptake inhibitor antidepressant clomipramine, patients received 8 weeks of self-exposure instructions (in the form of a workbook with therapist instructions) (Marks et al., 1988). Half of these patients then received 9 weeks of therapist-assisted exposure; the other half continued self-administered exposure for another 9 weeks. Overall, there were few differences in outcome between patients who did and did not receive the therapist-administered treatment. However, the design of this study precludes clear conclusions about the necessity of a therapist in ERP. That is, all patients had already received medication and self-administered exposure instructions; it may be that by that point, any additional treatment would have a negligible effect. A clearer (and larger) comparison using the BT-STEPS program was recently conducted (Greist et al., 2002). Patients were randomly assigned to receive self-administered treatment (BT-STEPS), therapist-administered ERP, or relaxation (placebo treatment). After treatment, 58% of patients in the therapist-administered treatment group, versus 38% of those in the BT-STEPS group, were considered responders. Therapist-administered treatment patients showed a 30% reduction on the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS), compared with a 23% reduction for the BT-STEPS group (effect sizes = 1.22 and 0.85, respectively). Interestingly, when only treatment–adherent patients were sampled, the therapist-administered and BT-STEPS groups showed similar outcomes, suggesting that the reason for the attenuated results in the BT-STEPS group may have been related to nonadherence to the treatment instructions in that group.

We are currently examining the use of self-help manuals in the treatment of OCD patients who have not responded or have only minimally responded to medications. In this study, patients who have received an adequate trial of a serotonin reuptake inhibitor, but have not experienced satisfactory improvement, are randomly

assigned to 15 sessions of therapist-administered ERP, or to the self-help manual *Stop Obsessing* (Foa & Wilson, 2001). Patients assigned to self-help treatment are given a schedule recommending weeks in which chapters and chapter assignments should be completed; yet no other therapist suggestions or contacts are provided. Patients are given 6–8 weeks to complete the self-help treatment. Therapist-administered treatment is administered using a flexible dose schedule, with treatment lasting from 3 to 7 weeks. All patients meet with an independent evaluator, who is blind to treatment condition.

Preliminary results ($N = 15$, self-help $n = 9$, therapist assisted $n = 6$) indicate that both groups achieve significant improvement on the Y-BOCS. However, patients receiving treatment from a therapist showed lower posttreatment Y-BOCS scores (controlling for pretreatment scores) than did self-help patients. The average reduction in Y-BOCS scores for patients in the therapist condition was 53.7% (range 38–64%). The average reduction in Y-BOCS score for the self-help condition was 20.8% (range 0–53%). On the Clinical Global Impressions (CGI) scale, a clinician summary of the severity of OCD and all other psychiatric illness, patients in the therapist group demonstrated significant improvement, whereas those in the self-help condition did not. The average reduction in CGI severity ratings for those in the therapist condition was 36% (range 25–50%). The average reduction in CGI severity ratings for those in the self-help condition was 7.9% (range 25–60%). The average score on the CGI improvement (0 = "very much worse," 3 = "no change," 6 = "very much improved") scale at posttreatment was 5.5 (range 5–6) for the therapist condition and 4.11 (range 2–6) for the self-help condition. Examination of clinical significance (Jacobson & Truax, 1991) revealed that 66.7% of patients receiving therapist-administered treatment met criteria for clinically significant change, compared to only 22.2% of those receiving self-administered treatment.

In summary, our early results suggest that patients receiving therapist-administered ERP improve to a greater degree than do those receiving the same form of treatment in a self-help format. A therapist may add variables that a self-help program cannot provide, or can only provide with limitations. In addition to the preliminary steps of ensuring the proper diagnosis and the appropriateness of ERP, a therapist may contribute the following to treatment: education, consultation, support, modeling, motivation, and accountability. Below, we discuss each of these contributions using illustrative case examples.

ROLES OF THE THERAPIST

Education

Educational functions of the therapist include ensuring that the patient has a thorough understanding of OCD, ERP, and the rationale for treatment. Typically, this involves teaching the patient a cognitive-behavioral model of the disorder. Such understanding is generally considered a critical prerequisite to successful treatment. Most self-help books on OCD include some form of psychoeducation (eg, Baer, 2000; Foa & Wilson, 2001; Hyman & Pedrick, 1999; Schwartz, 1997); however, the education provided in self-help programs may be limited. One potential limitation is the inability of a self-help treatment to discern whether one adequately understands the material. A therapist has the opportunity to assess the patient's understanding of OCD and

ERP, and to correct misconceptions that may lead to patient mistakes in completing ERP or that might put the patient at risk for noncompliance.

Self-help manuals may not adapt well to the wide range of situations that can arise in therapy, and the specific educational needs that arise out of these situations. In the course of therapy, a clinician has the opportunity to assess specific situations in which additional education or review of past material is necessary. Abramowitz, Franklin, and Cahill (2003) note several areas of education they utilize to aid patient understanding of OCD and ERP, including the normality of intrusive thoughts, the thought suppression paradox, and cognitive errors such as thought–action fusion and intolerance of uncertainty.

Self-administered treatment programs may also be difficult to adjust to a patient's education level. Some patients can understand a great deal of the complex research into OCD, whereas others may only be able to understand very basic information. By understanding the patient, a therapist can adapt the information given to the patient to minimize confusion and maximize understanding.

CASE EXAMPLE

Mr A is a 37-year-old man whose OCD involved intrusive images of harm coming to him and family members. One of the most troublesome images for Mr A was of his teenage son being maimed by a lawnmower. As a result, he would not permit his son to earn money mowing lawns, as he feared his son would be involved in an accident in which his hand or foot would be caught by a lawnmower blade or a rock would fly from underneath the mower and strike him in the eye. The treatment plan involved imaginal exposure to images of his son being harmed. Initially, Mr A was resistant to this treatment, as he believed that it was necessary for him to suppress these images in order to control his anxiety. In order to proceed with treatment, Mr A required "normalization" of his discomfort toward imagining harm coming to his son, as well as further education about the relationship of cognitive and behavioral avoidance to the maintenance of these intrusive images. First, Mr A was taken through a "thought suppression" exercise in which he was asked not to think of a white bear for 30 s. He reported that while trying not to think of a white bear, he actually thought about it more often. This enabled him to understand that his attempts to suppress the thoughts were paradoxically increasing them. Next, the expected pattern of habituation was reviewed in the context of previous exposure exercises. After this exercise and subsequent discussion, Mr A proceeded through the imaginal exposure exercises designed to weaken his anxiety associated with intrusive thoughts.

Whereas such educational modules might be present in self-help programs, the therapist applied them at a critical time in Mr A's treatment. Were he utilizing a self-help program, he might not have referred back to material on the thought suppression paradox or habituation. Treatment did not proceed until he fully understood the rationale behind it. Without this piece of information, Mr A may have avoided this exposure, abandoned it prematurely, or performed it improperly. In this case, the therapist was able to deliver the education in response to the patient's immediate needs.

Consultation

In addition to educational functions, the therapist acts as an ongoing consultant or "coach" by making sure that the patient understands how to complete ERP tasks

properly. To fulfill this function, the therapist must look for opportunities during treatment to provide corrective information. One opportunity for such coaching is when patients wait for an exposure opportunity to occur naturally (eg, waiting for one's hands to become dirty during natural activities), rather than purposefully bringing on the feared situation (eg, deliberately touching things that seem dirty). In this case, the therapist can assist by pointing out the importance of sustained, deliberate exposure exercises. Another common misconception held by patients is that ERP exercises should mimic "normal" behavior. For example, a patient may express a desire to shower once per day, because *most* people he/she knows do this. In this case, the therapist can explain that ERP exercises are designed to weaken the overestimations of risk and intolerance for uncertainty that underlie OCD, and to help the person systematically overcome their fears. Therefore, overcoming his/her fear of contamination may necessitate engaging in activities that are decidedly "abnormal," yet of acceptable risk, such as going for prolonged periods without showering. Patients also make the mistake of engaging in partial exposures that do not adequately activate their fears. For example, a patient might touch a feared object quickly and then stop, or touch a contaminant and touch their whole body except their face, leaving their face as a "safe zone." Another patient may attempt to mentally "freeze" a contaminated spot on their hands, preempting the perception of spreading of contamination. Other patients might substitute subtle mental rituals for overt behavioral compulsions. These common "safety behaviors" are akin to compulsive rituals and may serve to reinforce the obsessive thought that the feared object is dangerous, or to block the natural habituation process. Consultation may also include helping the patient to differentiate OCD symptoms from everyday concerns, thus helping to identify appropriate targets for treatment.

CASE EXAMPLE

Mr B is a 41-year-old man who feared that he would attack other people. Due to these fears, he avoided prolonged time in public places. One of his initial exposures involved walking through a crowded supermarket. Mr B reported that his fear remained high during exposure and did not improve as he repeated the assignment. Reviewing the manner in which he conducted the exercise revealed that he was engaging in mental rituals (saying silent prayers) to prevent himself from hurting others in the supermarket. The therapist was able to point this out and direct Mr B to repeat the exercise without the mental rituals. When he repeated the same exposure without ritualizing, he experienced a gradual reduction in anxiety.

Support

Exposure and response prevention is a challenging treatment, and many patients require a great deal of support and encouragement during this process. Certainly one's family and friends can be an important source of support, and this is strongly encouraged in therapy. However, given the fact that these people are often personally affected by the patient's OCD and at times may not be able to provide adequate support, patients usually find it helpful to receive additional support from the therapist. In the early stages of treatment, the therapist sets a collaborative tone in which the therapist and patient will "team up" to fight OCD. Initially, the patient may be embarrassed by obsessions that may contain sensitive content they have never revealed,

such as fears of hurting others or doubts about one's sexuality. A therapist who does not reject a patient for these thoughts and instead acknowledges them with acceptance provides a context for an individual to begin to confront their fears. Often, we find it helpful to remind patients that such thoughts are common even among people without OCD (Rachman & de Silva, 1978).

As a patient begins to face their feared situations, anxiety tends to increase. At this point in treatment, it is easy to retreat because one believes it is too hard or it is causing too much distress. A therapist at this point can help the individual understand that this increase is expected and offer encouragement that the anxiety will dissipate as they continue ERP. The therapist can offer real life success stories of other patients whose anxiety subsided as they faced their fears. While a self-help program can offer stories as well, hearing it from someone who is connected to the struggling patient and to the success stories they describe may be more credible to the patient. During this stage of treatment, the therapist praises the patient for his/her efforts, rather than for success. As the patient progresses to more difficult items on the exposure hierarchy, the therapist can use examples from the patient's earlier exposures to demonstrate that his/her fear will not last forever, and that he/she is capable of doing difficult exposures.

CASE EXAMPLE

Mr C was a 62-year-old man who suffered from intrusive thoughts that he was homosexual. This fear was particularly challenging for him, as an adult male had sexually abused him when he was a child. Mr C had never discussed this history, even with his wife of 40 years, and was reluctant to do so in therapy. When he did, however, he reported a sense of relief and was surprised that his therapist did not look at him with shame and disgust. Listening to his story with empathy and without judgment allowed for a more complete assessment of rituals and avoidance and allowed the therapist to discuss exposures to this obsession. Although he was willing to complete these exposures, they were still difficult for him and required a great deal of encouragement.

Modeling

Another role of the therapist is to model exposures. A therapist can demonstrate a willingness to perform ERP, as well as the proper implementation of ERP exercises. As previously mentioned, ERP often requires one to go beyond what is considered "normal" behavior. For instance, an individual who fears contracting diseases from public bathrooms may be expected to conduct exposures to touching toilets and then rubbing his/her hands over his/her body. As might be expected, many patients are initially reluctant to complete this exercise, even after completing other exercises successfully. The therapist can demonstrate the appropriateness and acceptability of such exposures by completing them with the patient. In our clinical practice, we have even helped patients to understand the acceptability of not showering for 3 days (response prevention) by doing so ourselves. These modeling activities demonstrate that we do not consider exposures to be dangerous, and that we are not asking patients to do anything that we are not willing to do ourselves.

In addition to being a model of willingness to engage in ERP, a therapist can model how to perform ERP tasks appropriately. The tendency of OCD patients to be overly cautious may lead them to ERP exercises of low intensity. By performing

exercises with a patient, the therapist can demonstrate the most appropriate ways in which to perform them. For instance, a patient can observe the therapist touch a contaminant and touch his/her clothing, hair, and face, as well as objects that others may touch. Modeling also allows us to demonstrate appropriate coping with feared activities (eg, "This is pretty disgusting and I feel very dirty, but I also know that this is unlikely to hurt me"). Therapists must walk a fine line in these cases, taking care not to provide compulsive reassurance via their modeling. For example, some patients have told us that they knew an object was safe to touch because they had seen us do it, and no harm came to us. While this kind of inference from modeling may be useful in the early stages of exposure, it eventually becomes counter-productive as the patient comes to rely on the therapist as a "safety check." Therefore, in many cases, after consulting with the patient, we have decided it best for the patient to engage in an exposure *without* watching the therapist do it first.

CASE EXAMPLE

Ms D was a 32-year-old woman who had the unusual fear of being contaminated by items from Asia. She found exposures to become increasingly difficult as she got closer to her highest fears. One of her highest exposures involved touching a souvenir a friend had brought back from China. Her therapist agreed to conduct an exposure along with her to aid the process. As Asian items would not cause the therapist discomfort, he revealed his dislike of touching velvet. The patient and therapist agreed that he would hold onto a piece of velvet while she completed an exposure to the Chinese souvenir. Although the therapist's experience was personally uncomfortable, it did not reach the same level of fear and discomfort as did Ms D's exposure. However, she reported that it was easier to tolerate her own discomfort, knowing someone else was willing to experience discomfort as well.

Motivation

Although patients usually dislike having OCD and look forward to a life that is not ruled by obsessions and compulsions, the prospect of facing feared situations and abstaining from rituals to achieve this goal may overshadow their desire to engage in treatment. Other patients may be ambivalent or unmotivated to seek treatment for OCD, and deny that they have a serious problem. Still others may adopt a hopeless outlook, believing that no treatment can help them. Thus, another role of the therapist is to bolster the patient's motivation to begin and continue with ERP.

To help therapists accomplish this aim, Maltby, Tolin, and Diefenbach (2002) have developed a four session readiness intervention for fearful patients who initially decline ERP. This program consists of psychoeducation, a videotape example of an ERP session, motivational interviewing techniques (Miller & Rollnick, 1991), and a phone conversation with a former ERP patient. Preliminary data from a study on the effects of this program ($N = 12$) indicate that 71% of patients subsequently chose to begin ERP, whereas only 20% of patients in a wait-list condition entered ERP.

Fear of performing particularly anxiety-evoking exposures can contribute to patients' ambivalence toward ERP. Thus, one role of the therapist is to aid patients in making appropriate judgments about exposure. We often use the principle of "acceptable risk" in designing possible exposures with a patient. Patients are informed that exposures may contain some degree of risk, yet the risk is low and similar to those

taken in everyday life. For example, a patient who does not want to complete an exposure to touching a toilet without washing may be asked to compare the risk of this exercise with that of a camping trip where cleanliness is delayed for days or weeks, or to driving on a highway to get to the therapist's office. It is common for individuals with OCD to perceive any risk to be excessive. However, the therapist can help patients to adopt a strategy of assuming, in the absence of contrary information, that the exposure situation is low risk. Taking a new perspective toward risk may improve willingness to engage in feared behaviors.

As patients struggle during ERP, they may worry about their prospects for successful outcome. What a patient may see as a failure, a therapist may see as a typical response to ERP or as merely a temporary setback. A patient having slow progress in reducing their anxiety may receive encouragement to keep going when their therapist points out that their performance is appropriate and their progress is in the right direction. Patients may also view setbacks, such as ritualizing during ERP, as a failure. Again, a therapist may help the patient reinterpret the event as a learning experience and a chance to work harder on overcoming OCD.

CASE EXAMPLE

Mr E entered therapy with fears that that he would be responsible for harm coming to others. His compulsions included picking up objects he thought might be harmful, such as rocks or twigs on the sidewalk. Mr E was initially wary of suggested exposures such as dropping thumbtacks onto roads. At this point in treatment "acceptable risks" were reviewed. Discussion included a review of materials commonly seen on the roadways such as rocks, broken glass, potholes, and nails that have the potential to cause harm. The therapist pointed out that all driving assumes some degree of risk of tire damage and subsequent accidents; however, the risk of harm is actually quite small as evidenced by the relative rarity of flat tires. The risk of tossing thumbtacks onto the road was then compared to the risk of putting nails through a board so they can be laid out on the road straight up. Compared to nails sticking straight up, the risk of dropping thumbtacks on the road appeared much smaller. Mr E was able to acknowledge this continuum of risk and engage in the exposure exercise.

Accountability

The presence of a therapist introduces an additional degree of accountability for the patient. In a self-help program, one can easily make excuses that result in treatment delay or outright cessation. For example, we have often heard patients tell us that they purchased a self-help manual but did not read it completely because their lives became too stressful, or they convinced themselves that they would read the book at some future, unspecified date (but never got around to it). The result of this process is that patients may not follow through with self-administered treatment. Therapist-administered ERP, on the other hand, involves contracting with the therapist regarding specific goals, expected treatment-related behaviors, and a time frame for treatment (Otto, Reilly-Harrington, Kogan, & Winett, 2003). This contract, whether formal or implicit, may enhance motivation and elicit appropriate behaviors from the patient, particularly during high-stress periods in treatment. For example, the expectation that homework assignments will be completed by the next session

may serve to increase compliance. A therapist can emphasize that treatment requires a strong commitment that the patient must be willing to prioritize.

CASE EXAMPLE

Mr F, who participated in our study comparing self-help treatment to therapist-administered treatment, was assigned to the self-help condition that included recommendations to complete certain assignments by weekly due dates. At the outset of treatment he was enthusiastic about following the program; however, when he returned for his posttreatment evaluation, he indicated that he had not done any of the assignments and proceeded to list stressful life events that impeded his progress (his sister got married, he needed to plan a business trip, and he was moving into a new house). After the study was completed, Mr F was referred to our outpatient clinic. The therapist helped Mr F review priorities and pointed out that life stress may at times be overwhelming to the point in which it is necessary to postpone treatment until the situation has resolved. However, it may also be the case that the patient is waiting for their life to be completely devoid of stress before beginning ERP. Mr F came to recognize that waiting for a completely stress-free time was unrealistic, and decided to move forward with therapist-administered ERP.

INTEGRATION OF SELF-ADMINISTERED AND THERAPIST-ADMINISTERED TREATMENT: THE ROLE OF STEPPED CARE

As is evident from the above discussion, we view the presence of a therapist as a critical component of treatment for most patients. However, this does not imply that we see no role for self-administered treatment. Indeed, many patients prefer to try self-help before investing the time, energy, and money into therapist-administered treatment. This sequential implementation of multiple treatment modalities represents a *stepped care* model of treatment. We are currently testing the feasibility and utility of a stepped care program for OCD. In this program, patients are first supplied with a self-help program to be completed without the aid of a therapist. Patients not meeting criteria for treatment response after this step are entered into the second step of treatment. In this condition, patients meet with a therapist to discuss their progress with ERP. Exposures are not modeled during these sessions, but the patients are given feedback on the exercises they performed on their own. The therapist's main role at this stage is as consultant, although education, support, accountability, and motivation are appropriate roles as well. Patients not meeting response criteria at the conclusion of this step enter the third step, which consists of therapist-administered ERP. While this study is still ongoing ($N = 10$), preliminary results are encouraging. To date there has been a 20% response rate to step 1 (self-administered ERP), with a mean 31% reduction in Y-BOCS scores. Patients who did not respond to step 1 showed a 28.6% response rate for step 2 (therapist support), with an additional 3.5% average reduction in Y-BOCS scores. Among patients who did not respond to steps 1 and 2, there have been two step 3 (therapist-administered ERP) completers, both of whom met responder status at the end of treatment. Step 3 treatment completers experienced an additional 75% reduction in YBOCS scores. These data are preliminary, but suggest that stepped care may be an effective and cost-effective method of ERP administration.

The advantage of a stepped care program is that it is self-correcting in its ability to provide patients with their optimal level of care and no more. Patients that perform well on their own may not need a therapist. Thus, using a self-help program may save both time and money for the patient. However, therapist support and direction are available for patients who need additional assistance and our early data suggest that therapist involvement (particularly modeling and directing exposures) may produce substantial incremental efficacy for patients who did not respond to self-administered treatment.

CASE EXAMPLE

The case of Ms D, discussed previously, provides an example of how the roles of the therapist were essential in treatment. Ms D initially refused ERP and was entered into our readiness intervention, during which the therapist discussed the concept of "acceptable risk," helped her to explore her ambivalence toward ERP, and facilitated a conversation with one of our former ERP patients. After completing this program, Ms D agreed to enter into the stepped care program. During step 1, she read the materials but did not properly engage in response prevention and was trying exposures to items that were higher on her hierarchy than was appropriate for initial exposures. Thus, she proceeded to step 2. At the start of this step, the therapist and Ms D reworked her hierarchy to approach easier fears first. In addition, she was encouraged to abstain more strictly from rituals. While Ms D did engage fully in ritual prevention, she did not complete exposure exercises on her own. Ms D's motivation began to wane at this time; however, with support and encouragement from her clinician, she agreed to enter the final stage, therapist-administered ERP. The first job for the therapist was to demonstrate how to conduct the exposures and to model them for the patient. As Ms D observed the therapist and followed what he was doing, she noticed her anxiety beginning to decline. These initial treatment successes increased her motivation to continue.

One of her highest exposures was to a souvenir a friend had brought back from Asia. While this item was readily available to her, as her friend lived in her neighborhood and told her she could borrow it, she never went forward with this exposure on her own. During therapist-administered treatment, she was given the assignment of bringing the souvenir to a session. This made it easier for her, as she would do the exposure along with the therapist; it also set a deadline for the exposure.

Consultation was essential in adjusting how she performed exposures. In her home, exposures often involved encouragement to push into her "safe zones." One particular exposure that proved difficult was bringing Asian objects into her bedroom. After placing an object on her pillow, she struggled to go to sleep that night and engaged in rituals. Ms D wanted to leave her own bed out of exposures, as it had always been a safe haven from OCD and seemed too difficult to tackle. Education was utilized by discussing, from a context of her symptoms, how initial exposures often do not completely eliminate anxiety; rather, repeated exposures are needed. A plan was developed to work with the pillow outside of the bedroom and begin the exposure before bedtime to allow some initial anxiety reduction. Ms D was encouraged to tolerate a few nights of bad sleep as an "investment" in her recovery. She did so, and reported sleep improvement over time. For this patient the therapist was a necessary variable in treatment. The therapist provided education, consultation, support, motivation, and accountability. While obstacles to improvement were apparent throughout treatment, she was able to overcome them, evident from her reduction in Y-BOCS score from a 25 at intake to 3 at posttreatment.

CONCLUSIONS

Self-help programs may offer an alternative treatment option for patients with OCD. Traditional therapist-administered behavior therapy may be difficult to access due to a lack of trained professionals and costs associated with treatment. Given these challenges, some patients may prefer to attempt treatment on their own. Self-help programs are readily available to patients who may not be able to afford or find appropriate treatment. As self-help has been demonstrated to be effective for some patients with OCD, it may be a sufficient treatment option for certain individuals who cannot access or afford traditional therapist-administered treatment. However, other evidence suggests that therapist assistance is critical to successfully implement ERP, as a therapist provides several variables that may otherwise be absent. In particular, the therapist may provide education, consultation, support, modeling, motivation, and accountability.

The question of who requires therapist-administered treatment and who is able to utilize a self-help program still remains and further research into predictors of treatment success in both domains is still needed. Until we have a clearer understanding of who is able to succeed with self-help and who requires therapist-administered treatment, a stepped care model of treatment may be the most appealing treatment option. This model allows a flexible model of care in which a patient, along with a therapist, proceeds incrementally through more intensive forms of treatment until they have reached an optimal level of care providing symptom reduction. Flexible treatment programs such as stepped care may be the most cost-effective way to integrate the low cost of self-help with the particular skills of a therapist.

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Chapter 17

SELF-DIRECTED EXPOSURE IN THE TREATMENT OF OCD

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Since Victor Meyer's (1966) first report on two successfully treated OCD patients, the behavioral intervention of exposure and response prevention (ERP) has become firmly established as a highly effective method for reducing symptoms of the disorder (see Abramowitz, 1997; van Balkom et al., 1994, for reviews). However, as several authors have noted (Foa & Kozak, 1986; Steketee & Barlow, 2002), no one is entirely sure why exposure is effective. It appears that an extended period of confronting a feared situation permits the emotional discomfort associated with that situation to dissipate and subsequently provokes a less intense reaction. As a result of repeated exposure to obsessional material, coupled with the prevention of those rituals that serve the initial function of neutralizing their associated fears, the individual's attitudes and expected negative outcomes are altered. As Steketee and Barlow (2002) conclude, "... the major procedural issue in treatment is arranging for sufficient exposure to occur without interruption from neutralizing strategies" (p. 540).

Many outcome studies report the use of intensive ERP treatment protocols. Given the intensity and frequency with which such treatment is delivered, the clinician is actively involved in the planning, delivery, and monitoring of ERP. Therapist-directed intensive interventions include sessions that can last for up to 2 h and can be as frequent as on a daily basis (eg, Abramowitz, Foa, & Franklin, 2003; Foa, Kozak, Steketee, & McCarthy, 1992; Franklin, Abramowitz, Kozak, Levitt, & Foa, 2000). Interestingly, the duration and optimal frequency of ERP have not been clearly established. Studies suggest that habituation is associated with improvement following ERP (Kozak, Foa, & Steketee, 1988) and that approximately 90-min of continuous exposure is needed for anxiety to be sufficiently reduced and for a decrease in the urge to ritualize to occur (Foa & Chambless, 1978; Rachman, De Silva, & Roper, 1976). Steketee and Barlow (2002) suggest that 20 sessions of exposure are necessary but the frequency of sessions varies.

While there is considerable research supporting the use of therapist-directed exposure, treatment can be successfully delivered with very limited involvement on the part of the clinician (eg, Emmelkamp & Kraanen, 1977; Emmelkamp, van den Heuvell, Ruphan, & Sanderman, 1989). Methods involving no therapist participation that, instead, utilize self-treatment guided by a manual and computer-conducted telephone

interview have been reported as well (Bachofen et al., 1999; Marks et al., 1998). Whereas the latter reports constitute the extreme with regard to self-directed exposure, the larger proportion of treatment studies involves both therapist-directed treatment that is conducted during face-to-face ERP sessions coupled with self-directed exposure that is assigned as homework between therapy appointments.

The focus of this chapter is on the self-directed aspects of ERP for the treatment of OCD. Specifically, we will be discussing the benefits as well as the impediments to self-administered exposure treatment. After providing an overview of self-directed ERP, the chapter will address how manuals as the standard of care integrate self-directed exposure, the transition from therapist-directed to self-directed exposure (ie, therapist fading), issues related to treatment adherence, how comorbidity affects self-directed treatment, and finally, the benefits and limitations of this approach.

OVERVIEW

The literature provides several descriptions of self-guided treatment for other anxiety disorders such as agoraphobia (Edelman & Chambless, 1993; McNamee, O'Sullivan, Lelliott, & Marks, 1989; Michelson, Mavissakalian, Marchione, Dancu & Greenwald, 1986) and specific phobia (Hellstrom & Ost, 1995). Michelson et al. (1986) suggested that self-directed exposure was strongly related to endstate functioning. However, subsequent studies did not entirely support this finding. Edelman and Chambless (1993) found that in the treatment of agoraphobia, more time spent with homework exposures was associated with greater decreases in anxiety sensitivity and avoidance. However, these authors concluded that it might not be the homework itself that was the critical factor. Instead, clients who are receiving ample amounts of therapist-directed exposure or a particular client type who is more prone to complete homework may, then, be more likely to improve. McNamee et al. (1989) reported that agoraphobics who participated in telephone guided self-exposure did not respond as well to treatment as did subjects in previous studies involving self-directed or self- plus therapist-accompanied exposures (eg, Ghosh & Marks, 1987) possibly due to severity of symptoms and motivational issues. In a comparison of one-session therapist-directed exposure versus two forms of manual-based self-directed exposure for the treatment of spider phobia, 80% of the patients who received therapist-directed exposure significantly improved as compared with 63% in a specific manual-based treatment conducted in a clinic, 10% for a specific manual-based treatment completed in the home, 9% for a general home-based manualized treatment, and 10% for a general clinic-based manualized treatment (Hellstrom & Ost, 1995).

Much as in the treatment of agoraphobia or specific phobia, there is some evidence suggesting that OCD treatment can be administered with limited therapist involvement. Evaluations of the presence of a therapist during treatment have yielded inconsistent results, thereby suggesting that the therapist's contribution is, at best, ambiguous (Foa & Franklin, 2001; Kozak & Foa, 1997). A meta-analytic review (Abramowitz, 1996) indicated that therapist-directed exposure was more effective with regard to improvement in both OCD and other anxiety symptoms compared to self-controlled exposure. However, in a frequently cited study that directly compared therapist-assisted to self-exposure, Emmelkamp and Kraanen (1977) reported that there were no differences in efficacy at posttreatment or at follow-up. Unfortunately,

small sample sizes in each condition of this latter study make it difficult to draw a firm conclusion. A report examining the use of an OCD self-help book coupled with meetings with therapists that involved no therapist-directed exposures over the 12 weeks of the treatment yielded significant pre-post changes on the Yale-Brown obsessive-compulsive scale (Y-BOCS) and another anxiety measure (Fritzler, Hecker, & Losee, 1997). Again, this was a small sample of nine subjects: all nine subjects demonstrated statistically significant change, whereas only three subjects met criteria for clinically significant change. The authors suggest that more severely impaired OCD patients may be better suited for therapist-directed exposure treatment and that hoarders, in particular, may benefit less from a self-directed approach. They also noted that two of the three therapists had no experience with OCD treatment, which also may have been a factor that influenced their outcome. A recent OCD effectiveness study relied heavily on self-directed exposure assignments (only 3 of 19 patients received either in office in vivo or therapist-assisted out-of-office in vivo exposure) (Warren & Thomas, 2001). These authors found significant reductions in Y-BOCS scores with 84% of their patients demonstrating clinically significant change on the total Y-BOCS score, thus supporting the utility of self-directed ERP.

One novel approach, called BT STEPS, involves the use of computer-assisted treatment and an interactive voice response system (Bachofen et al., 1999; Marks et al., 1998; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002) with no therapist involvement whatsoever. In some cases, subjects had contact with a treatment coordinator but this did not involve assistance with exposures. Research in both the United States and Great Britain found that the number of self-directed exposure sessions in which subjects engaged was correlated strongly with a decrease in their symptom severity. Moreover, Marks et al. (1998) noted that improvement did not occur simply by completing the self-assessment component of the program. Not surprisingly, improvement occurred only if subjects continued in the study and engaged in self-directed exposure practice. The authors noted that motivation appears to be a factor in the successful implementation of computer-based treatment (Bachofen et al., 1999). Thus, those subjects who completed the four self-assessment calls quickly were more likely to complete two or more self-directed exposures. Whereas a substantial portion (76%) of patients completed the self-assessment module of the program, only 43–48% went on to do two or more ERP sessions and improve significantly (Bachofen et al., 1999; Marks et al., 1998, respectively). Compared with dropout rates in other OCD studies, it would appear that fewer subjects with OCD participated in the active treatment phase of this self-directed approach. Predictors of poor outcome for the self-directed approach included poor insight into the senselessness of obsessions and compulsions, more ego-syntonic symptoms, less willingness to disclose symptoms, decreased motivation, and the presence of religious or sexual obsessions (Mataix-Cols et al., 2002).

Descriptions of OCD treatment typically include a self-directed component in the form of hierarchically arranged, between-session homework exposures (eg, Foa & Franklin, 2001; Foa & Kozak, 1997; Foa & Wilson, 2001; Hyman & Pedrick, 1999; Steketee, 1993, 1999). It would appear that self-directed exposure provides a mechanism for maximizing the patient's engagement in treatment while at the same time, insuring that rituals are being blocked across a broad range of situations, at least some of which may not be accessible or practically replicated in the context of a therapy session. In addition, McGinn and Sanderson (1999) note that individuals who are able

to attribute their improvement to themselves are more apt to do better than those patients who make external attributions for their treatment gains.

It is also likely that self-directed treatment is important for preventing relapse (Greist, 1994; Steketee, 1993). One explanation for this involves the short- and long-term effect of massed versus spaced learning and retrieval on memory. Schmidt and Bjork (1992) suggest that longer and more varied intervals between practice sessions, such as with self-directed exposure, impede learning during the acquisition phase, but enhances long-term retention through more opportunities to practice retrieval in a variety of contexts. It stands to reason that in order for treatment gains to be maintained, the OCD patient cannot be under the watchful eye of the therapist in perpetuity. What is learned in the context of a therapist-directed exposure needs to be generalized to the patient's day-to-day life when there is no therapist present to provide guidance.

THERAPIST FADING

Early in the process of therapist-directed ERP, it is common for the therapist to model exposure to feared situations for the patient during sessions. This provides the patient both with information regarding normative behavior and assurance that the patient is not being asked to perform a behavior that is dangerous or anything the therapist him or herself would not agree to do. In this way, the therapist can also assess patient comprehension of exposure therapy and can help motivate the patient to begin what can be a very difficult process. Direct therapist involvement in exposures also allows for the assessment of treatment integrity, for example, that the patient is participating in exposures correctly, is remaining in the exposure long enough to allow for habituation, and is not engaging in subtle ritualizing during the exposure (Abramowitz, 1996). After the first few sessions, and when it is clear that the rationale for, and the process of, ERP is well understood, patients should be required to complete exposures independently in session. By encouraging patients to engage in exposures independently, the likelihood of dependence on the therapist is reduced. Furthermore, patients then are more likely to attribute treatment gains to themselves, resulting in improved self-efficacy (McGinn & Sanderson, 1999). Thus, as Steketee (1993) concluded, when therapist-assisted ERP is utilized it should be focused and time-limited to avoid clients' dependence on the therapist.

Self-directed exposures and homework also make generalization to home and other natural settings more likely. Sometimes attempts at replicating feared situations in the therapist's office are ineffective in producing anxiety due to the artificial nature of the exposures or the perceived "safety" of this setting. Incorporating self-directed exposures and homework assignments each day can result in more realistic and beneficial fear-evoking experiences for the patient. If appropriate, a family member or friend can serve as a "coach" to provide support during these exercises. This, too, should be faded quickly so that dependence does not develop and care should be taken to assure that responsibility is correctly attributed to the patient.

Exposure practice is more likely to produce therapeutic levels of anxiety if the client is not able to shift responsibility to the therapist (Steketee, 1993) or other support person (eg, friend or family members). When patients must take responsibility for the feared outcome of the exposure it is more likely to result in beneficial anxiety, allowing

for subsequent habituation and consolidation of corrective information about the feared situation or stimuli. Maximizing the clients' role in making decisions about exposure tasks and response prevention rules during treatment is a way of transitioning patients to the independent, self-directed therapy necessary to maintain gains following treatment. In later sessions, clients should be asked to choose their own exposures in session as well as for homework assignments.

TREATMENT ADHERENCE

One obstacle to treatment success that clinicians may occasionally encounter is the patient's failure to follow through with self-directed exposures assigned for homework practice. Although compliance with ERP procedures is among the best predictors of long-term success for OCD patients treated with behavior therapy or combined behavior therapy and pharmacotherapy, roughly 25% of patients fail to comply with instructions provided by their therapist (Fals-Stewart & Schafer, 1993; McLeod, 1997; although see Abramowitz, Franklin, Zoellner, & DiBernardo, 2002). Thus, an important consideration is the identification of factors that influence a patient's decision to engage in, and comply with, instructions for self-directed exposure. As noted previously, homework serves to allow patients to generalize their ostensibly positive experiences with therapist-directed ERP to the situations they encounter on a regular basis that cause them to ritualize (Araujo, Ita, & Marks, 1996). In addition, self-directed exposure encourages the development of patients' confidence in their own ability to manage their symptoms without needing to depend on their therapist. As their confidence builds, successively more anxiety producing exposures can be introduced and therapist involvement can subsequently be tapered off. Typically, this rationale for homework is conveyed early in treatment when psychoeducational information about OCD is being imparted. For example, Foa and Franklin (2001) indicate that when describing their intensive treatment program, time is spent in the second information-gathering session to inform patients that homework will require 2–3 h of the their time in addition to the therapy session, and is to be conducted at the patient's home or other treatment relevant locations.

There appears to be a relationship between in-session and out-of-session homework compliance. Abramowitz et al. (2002) found a moderately strong correlation between compliance with in-session exposures and both homework exposures and subjects' understanding of the rationale for treatment. They also reported that understanding the rationale for ERP and compliance with both in-session and homework exposures were associated with less severe posttreatment OCD symptoms. For the 64.3% of their subjects who met criteria for clinically significant improvement, these same variables again proved significant. Thus, it would appear that placing a strong emphasis during the initial phase of treatment on insuring that patients understand the rationale for treatment may result in their greater willingness to engage in activities which they find challenging, if not highly anxiety producing.

Despite even the most precise and compelling explanation of the treatment rationale, there are some individuals who may actively participate in exposures when their therapist is present, but fail to engage in self-directed exposures. Clinically, we have found that some of these patients may not retain an understanding of why exposure homework is important. It is certainly possible that they approach each exposure as

an isolated event and struggle to generalize their experience of turning a doorknob to turning on a faucet, for example. Thus, each exposure is almost like starting treatment anew, it never becomes easier or more comprehensible. One might speculate that individuals with attentional biases to threat relevant cues, lack of confidence in memory, or diminished cognitive flexibility (Amir & Kozak, 2002) might find working independently of their therapist more challenging given the absence of an authority to review or reinforce the rationale for exposures, or help them to generalize their ERP experiences. It also has been hypothesized that impaired executive functioning in OCD may contribute to difficulty in retrieving new memories and may result in difficulty prioritizing or planning behavior as well as in initiating strategic actions (Savage, 1998). Any of these factors may impede the successful execution of self-directed exposures.

Another impediment to successful self-directed exposure is the presence of mental rituals and neutralizing strategies. These may not have been previously identified or, as exposures become increasingly difficult, patients may incorporate them into their repertoire. For example, a patient attempting exposure to a cemetery might perform a cognitive ritual such as praying to God for protection. Such a ritual would disrupt the exposure because it would prevent the patient from experiencing corrective information that cemeteries are not dangerous places. Similarly, a patient may try to absolve him/herself of responsibility within the context of the exposure, a form of neutralizing, by transferring liability for possible catastrophes onto the therapist. For example, one patient reasoned that because the therapist was "making" her imagine her child becoming ill, he would be ultimately responsible if this occurred. In such instances, patients may report completing exposure assignments, yet will not benefit since the point of the exercise is to confront the possibility of being responsible for causing illness.

Alternatively, some patients are willing to devote the time to exposures during treatment sessions but find a multitude of reasons to justify why they do not have the time to participate in self-directed exposures at home, or why such exposures are unsuccessful. There may be several explanations for such behavior. Studies have suggested that better treatment compliance is associated with less severe OCD symptoms (eg, Abramowitz et al., 2002), thus it may be more difficult for patients with highly acute symptoms to engage in exposures without a therapist present. Clearly, if exposure to an anxiety producing stimulus is overwhelming, the patient may become immobilized, simply avoid the stimulus, or perhaps continue ritualizing. Our clinical observations also suggest that if a patient's OCD symptoms are not severe enough or no longer impair functioning, the patient may be less motivated to engage in homework. Discussing possible impediments to self-directed exposure and reinforcing that the order of difficulty of hierarchy items is not immutable may help to circumvent on-going problems of this kind.

Often the therapist's best intentions have an unexpected impact on the patient's ability to perform an exposure. Araujo, Ito, Marks, and Deale (1995) noted that one of their patients refrained from doing imaginal exposure homework due to the depressing nature of the exposure script. Once the script was revised, the patient followed through with the homework. Typically, imaginal scripts present more extreme variations on the patient's fears and the related consequences. In an effort to be rigorous (and zealous), it is not inconceivable that the script becomes so overwhelming that the patient avoids the homework assignment. Of course, this is a fine line to walk since avoidance of important exposure material can hinder treatment response.

Family members may sometimes be incorporated into the ritualizing process and this can unwittingly detract from treatment compliance (Carmin & Wiegartz, 2000). When this happens, the patient becomes aware that his or her obsessions can be neutralized in absentia. Likewise, reassurance-seeking rituals can be subtle and family members may be unaware that they are reinforcing the patient's OCD symptoms. Thus, although the patient is not directly avoiding or performing compulsive rituals, violations of response prevention are occurring and efforts to implement self-directed exposures are thwarted. Should it appear that in-session exposures are not generalizing at a pace consistent with the therapist's experience, a visit to the patient's home while members are present may be the best method to observe how the patient and his or her family interact. If such a home visit is not feasible, taking therapy time to educate family members, and involving them as "coaches" or "support people" by instructing them in how to set limits, may then facilitate self-directed treatment.

The fundamental nature of exposure therapy is that patients are asked to confront the very situations and stimuli that they fear and have been working hard to avoid. It is understandable that it takes considerable motivation to face what is a perceived catastrophe. Even though a therapist-directed exposure may begin to provide evidence to refute the consequences of not completing a ritual, patients may be reluctant to complete self-directed exposures. This gives rise to the possibility that some subjective change needs to occur in order for an OCD patient to maximally engage in exposures on his or her own. We know very little about who succeeds at self-help approaches. Further research into both self-help and the characteristics of those individuals who are resistant to participating in self-directed exposures is needed to better address these issues.

COMORBIDITY

Patients diagnosed with OCD comprise a heterogeneous group. As researchers converge on the several robust dimensions that emerge from structural analyses of obsessions and compulsions (eg, Baer, 1994; Leckman, Grice, Boardman, & Zhang, 1997; Summerfeldt, Richter, Antony, & Swinson, 1999; Wu & Watson, 2003), clinicians are reporting that different symptom domains respond differently to treatment efforts. For example, Mataix-Cols et al. (2002) found that OCD patients presenting with sexual/religious obsessions had poorer outcomes with behavior therapy than did OCD patients presenting with other primary symptoms.

In addition to consideration of such within-disorder heterogeneity, OCD also is observed to be frequently comorbid with a variety of other Axis I and II disorders, particularly mood and other anxiety disorders (eg, Steketee, Eisen, Dyck, Warshaw, & Rasmussen, 1999; Steketee, Henninger, & Pollard, 2000). Reports have suggested that at least one-third of OCD patients present with major depression and many more show clinically significant depressive symptoms (eg, Rasmussen & Eisen 1998; Steketee et al., 2000). Other disorders seen frequently in patients with OCD include panic, social phobia, eating disorders, disorders that frequently are referred to as OC Spectrum (eg, hoarding, body dysmorphic disorder, trichotillomania) and personality disorders (eg, Black & Noyes, 1990; Crino & Andrews, 1996; Mineka, Watson, & Clark, 1998; Rasmussen & Tsuang, 1986; Ricciardi & McNally, 1995). Each of these conditions holds the potential for influencing a patient's ability to engage in and complete behavioral

treatment, albeit therapist- or self-directed. In general, the available evidence suggests strongly that patients with multiple, comorbid conditions present a pattern that is more severe and often more difficult to treat than "pure" disorders (Abramowitz & Foa, 2000; Clark, Watson, & Reynolds, 1995; Mineka et al., 1998; Steketee, Chambless, & Tran, 2001).

Whereas research in this context typically has not targeted OCD and its treatment, there is mixed evidence to suggest that this pattern holds for OCD. For example, Steketee et al. (2001) reported that comorbid GAD in patients with OCD predicted treatment dropout and comorbid depression predicted poorer short-term outcome for patients who completed behavior therapy. Similarly, Gershuny, Baer, Jenike, Minichiello, and Wilhelm (2002) noted that patients diagnosed with comorbid *DSM-IV* OCD and PTSD showed significantly less symptom improvement following ERP than did patients diagnosed with OCD only. This blunted improvement was found not only for obsessions and compulsions (as measured by the Y-BOCS), but also for depressive symptoms (BDI scores). These general findings notwithstanding, a review of the literature reveals that virtually no attention has been paid to the influence of comorbidity on the success of self-administered exposures for OCD.

Clinically, it is clear to see how some conditions may affect the execution of successful self-exposures. For instance, when patients present with significant depression, they may be less hopeful that treatment will work and, thus, be less motivated to comply when not being encouraged directly by the clinician. Likewise, the anergia and fatigue associated with depression may impede the patient's best intentions. Clark (2002) suggests that there are cognitive features common to both OCD and depression. In particular, maladaptive beliefs involving the significance of the intrusive thoughts, their overimportance, self-blame, and the need to meet exaggeratedly high personal standards may in combination with cognitions specific to depression (eg, thoughts of failure, negative self-appraisals, beliefs related to isolation or non-acceptance and failed mastery, achievement or independence) serve to intensify OCD symptoms and complicate treatment.

If, as Clark (2002) suggests, there is a mutual reinforcing cycle of OCD and depression-related cognitions, then behavioral experiments that target such negative cognitive content need to be developed. What is, however, unclear are whether cognitive elements need to be addressed prior to ERP or vice versa. One could argue that focusing on cognitions first might alleviate both anxiety and depression. On the other hand, behavioral interventions might provide the patient with an early opportunity to experience success with exposure, both therapist-directed and self-directed, and may provide corrective data that inherently alter maladaptive cognitions. Independent of the order of the interventions, the therapist should be alert to balancing the supportive needs of the patient with respect to his or her low self-esteem, low confidence, or low motivation without either compromising the integrity of the behavioral protocol or providing excessive reassurance that could interfere with the patient's experience of the stressor as anxiety-provoking.

For OCD patients who struggle with co-occurring social anxiety or agoraphobia, it may be important for the treatment plan to incorporate exposure to situations relevant to these other disorders in order to facilitate compliance with self-directed treatment instructions. Consider a patient whose primary OCD symptoms involve contamination and who also has severe social anxiety. If OCD is the target for treatment, then exposures involving touching toilets or floors, handling money, or shaking

someone's hand may be highly appropriate. Unfortunately, the comorbid social anxiety may interfere with the patient's ability to engage successfully in the latter exposure. It would not be surprising that a patient with this presentation would then avoid those self-directed exposures that activated his or her comorbid fears. Although obvious, it bears stating that for OCD patients, it is important to complete a comprehensive diagnostic evaluation in order to assess whether there is comorbidity and if so, to be attentive to the behavioral and cognitive components relevant to the conditions present. A collaborative approach in which the patient and therapist work together in treatment planning may reveal the need to incorporate elements of multiple disorders into a single exposure hierarchy, or alternatively, to focus on one area of anxiety before addressing the other one.

Various personality traits and/or disorders might also complicate a patient's efforts at self-exposure. For example, an OCD patient who presents with comorbid OCPD might avoid self-exposure due to the rigidly held belief that his self-exposures must be completed in a manner that he sees as perfect, or "just so." Early discussions that dispel the notion of a "perfect" exposure might help the patient work toward more realistic goals and expectations for each task and, consequently, allow a reasonable opportunity for success. As another example, patients who present with self-entitled or narcissistic traits might resent a therapist who is particularly directive when it comes to planning homework exposures. These patients may rebel and not adhere to what they perceive as the therapist's plan for treatment or make the assumption that they know what is the best course for treatment and should not have to comply with instructions. Such patients might be invited early on by the therapist to help play a major deciding role in planning self-exposures and the therapist might repeatedly underscore the collaborative nature of treatment throughout. Provided tasks remain appropriate and within the realm of what the therapist believes to be indicated, this time and effort might go a long way in establishing rapport with the patient and helping them to take ownership over and engage in between-session self-exposures more fully.

In summary, then, comorbid conditions have the potential to influence various facets of a behavioral protocol. Thus, an important step in early treatment planning is to properly assess for comorbid conditions and to use this information when staging exposures and developing the hierarchy (or hierarchies) of self-directed homework exposures. The better able we are to gage patient's strengths and weaknesses, the better able we are as clinicians not only to improve our patient-specific interventions, but also the chances that a patient will be properly motivated to engage the protocol fully.

CONCLUSIONS

Clearly, there is a role for self-directed exposure in a comprehensive OCD treatment protocol. However, what research exists reflects considerable ambiguity as to just how critical a role homework, or other self-directed tasks, may play in influencing treatment outcome. There appear to be a handful of variables that may moderate response to self-directed exposure. Symptom severity as well as comorbid conditions diminished insight and/or the presence of ego-syntonic symptoms (as sometimes occurs in hoarding), and the presence of religious or sexual obsessions appears to interfere

with self-directed treatment. Likewise, motivation and treatment engagement are key variables. A thorough initial assessment may facilitate a better understanding of the nature of the patient's symptoms as well as help to identify what may be impediments to progress during the course of treatment. For example, assessments should inquire into whether family members are involved in rituals, particularly if reassurance seeking is present. On-going assessment may be necessary as well, particularly if treatment is not progressing. In this case, it may be important to assess for the presence of covert rituals or forms of distraction that interfere with habituation.

Providing a clear rationale for self-directed exposure also may be instrumental in facilitating adherence to treatment. Repeated presentations of the educational aspects of treatment should be a consideration if there is a diminished participation in homework. The importance of self-directed exposure and challenging oneself on a regular basis may need to be reviewed in order to enhance the patient's understanding of the rationale for assignment, treatment adherence, and motivation. Research also needs to focus on the role of cognitive variables and how they influence the self-directed components of OCD. Excessive attention to threat-related cues, decreased confidence in memory, and diminished cognitive flexibility all may have an impact on just how effective ERP may be. As our understanding of the mechanisms underlying the effects of cognitive therapy for OCD continues to grow, these same variables may be important with regard to their influence on how the obsessional fear structure is challenged. Thus, engagement in, and the effects of homework that involves cognitive restructuring is an area that needs to be examined further.

Very little has been said about the therapist variables and how they may interact with homework completion. Foa and her colleagues (Foa & Franklin, 2001; Kozak & Foa, 1997) noted that the contribution of the therapist to OCD treatment is ambiguous. To be sure, we know very little about what factors contribute to an OCD patient's success when he or she is involved in a purely in a self-help approach. Nonetheless, it is reasonable to assume that patients will continue to seek out therapy and that behavior therapists will assign homework. This being the case, therapist variables such as experience level may need to be considered. This is particularly germane given that OCD treatment often occurs in academic settings where novice therapists are involved. It would not be unreasonable to assume that an inexperienced therapist may be overly zealous or too timid with regard to assigning either in vivo or imaginal exposures that are too challenging. Likewise, trainees may not be sufficiently sensitive to just how specific exposure hierarchy items need to be in order to control for extraneous variables and to safeguard against unexpectedly difficult exposure tasks. In one study that noted the level of therapist experience (Fritzler et al., 1997), therapists had no previous experience with OCD treatment which may have accounted for the limited treatment success of their subjects. Unfortunately, confidence in the therapist and its relationship to self-directed exposures were not examined. In a more recent study, Franklin, Abramowitz, Furr, Kalsy, and Riggs (2003) found no relationship between therapist experience and outcome of exposure therapy. However, the assignment of patients to therapists was not random; thus, firm conclusions regarding the relationship between experience and outcome cannot be drawn at this time.

Whereas all of the above suggest that there are a myriad of challenges to the OCD patient's successful participation in self-directed exposures, there are obvious benefits for encouraging patients to do so. First and foremost is that cognitive behavior therapy (CBT) is a relatively short-term approach that encourages the patient

to assume an instrumental role in his or her treatment. The commonsense nature of CBT embraces the patient becoming independent of their therapist, hence the role of homework. As the therapist decreases his or her involvement in the exposure process, patients are encouraged to make internal attributions for their success at ERP. Hopefully, this results in an increase in patients' self-confidence, willingness to continue on to greater challenges, and generalization of their successes to day-to-day experiences. In explaining the rationale for homework, it is not unusual to point out that even with intensive treatment, patients spend only a small percentage of their week working with a therapist. Self-directed exposure helps to decrease the potential for relapse since the patient has acquired the skills necessary for completing ERP. Should they experience an increase in symptoms at some point after therapy concludes, they need to be well-equipped to implement these skills. Thus, it is critical for patients to spend time engaged in self-directed exposures for treatment to succeed and for gains to be maintained over time.

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Reply to Carmin et al.:

WHAT IS IN A NAME? THE DISTINCTION BETWEEN SELF-DIRECTED AND SELF-CONDUCTED TREATMENT

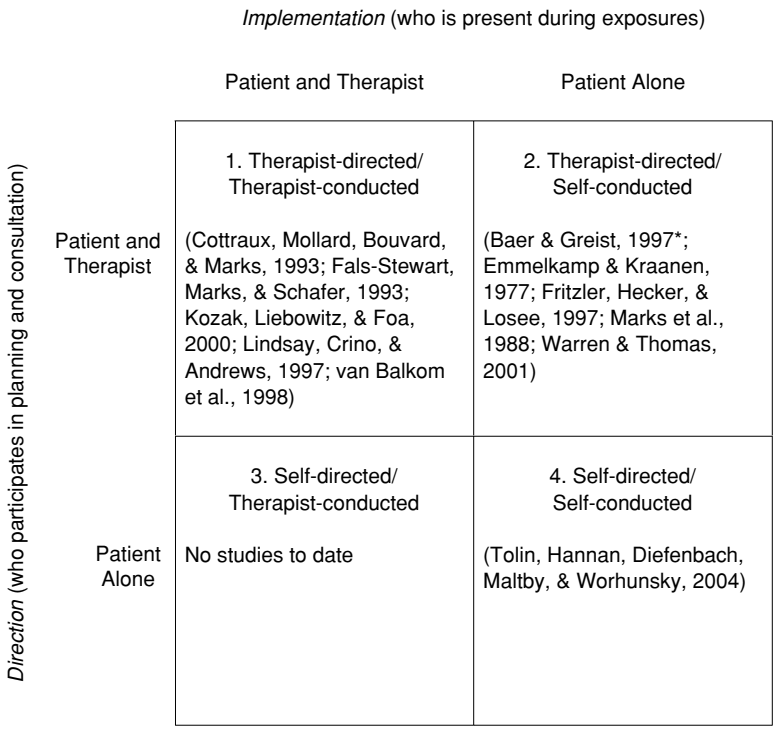
David F. Tolin and Scott Hannan

THE DIRECTION AND IMPLEMENTATION OF EXPOSURE

Carmin, Wiegartz, and Wu address appropriate points in regards to the necessity of a therapist during exposure for OCD. Rather than arguing that the therapist is unnecessary, they make a case for integrating self-directed exposures with therapist directed exposures, and consider how and when a therapist can be most useful in treatment. We concur with this general idea, and will return to this topic later.

One issue, however, requires clarification. Carmin and colleagues refer to several studies as demonstrating the efficacy of "self-directed" ERP. However, closer examination of these studies suggests that in many cases, the therapist's involvement was much greater than is suggested by this label. We propose that rather than the global term "self-directed," it may be more accurate (and useful) to think of the therapist's level of involvement along two dimensions: (*a*) "self-directed" versus "therapist-directed" and (*b*) "self-conducted" versus "therapist-conducted." Self- or therapist-*directed* exposure implies that the patient or therapist takes primary responsibility for determining what exposures will be conducted. Self- or therapist-*conducted* exposure implies that the patient or therapist is responsible for actually conducting the exposures. Thus, if the therapist guides the patient toward certain exposure exercises, but leaves the actual implementation of the exercise to the patient (as is frequently done with homework assignments), we would consider this to be therapist-directed/self-conducted exposure. This is more than simply a semantic issue; we suggest that examining the role of the therapist in the planning and implementation stages of treatment will clarify where and how a therapist is needed.

Figure 17.1 shows this conceptualization of therapist and patient direction in ERP. Box 1 in the figure (upper left) shows therapist-directed/therapist-conducted



*Note: BT-STEPS is included in this box because exposure decisions are made by a computer-based algorithm, rather than by the patient. However, BT-STEPS might also be considered a form of self-directed/self-conducted treatment.

FIGURE 17.1. The roles of the therapist and patient in the direction and implementation of ERP (and exemplary studies).

treatment. This reflects the "classic" model of ERP treatment upon which most randomized controlled trials have been based. In this model, the therapist takes primary responsibility for determining which exposures will be done, and assists the patient in the implementation of these exposures. For example, the therapist works with the patient to develop an exposure hierarchy and then accompanies the patient through most of the exposures.

Box 2 of the figure represents therapist-directed/self-conducted exposures. This is the form of ERP described by researchers such as Emmelkamp and Kraanen (1977), and includes most of the studies that Carmin et al. describe as "self-directed treatment." However, examination of these studies shows that a therapist *was* involved in decisions about the timing and implementation of exposures. In this type of treatment, the patient meets regularly with the therapist. The therapist's role in such treatment may include providing psychoeducation regarding OCD, aiding the creation of an exposure hierarchy, developing specific assignments for exposure homework, and consultation regarding a patient's performance during exposure exercises. However, the therapist is not physically present during the exposures. Yet, although not physically present during exposure, the therapist is intimately involved with all other aspects of treatment.

The therapist-directed/self-conducted treatment described by Emmelkamp and Kraanen (1977) included a large degree of therapist contact. Although therapists were not present during exposure exercises, patients met with therapists for ten 1-h office visits. No difference between therapist-controlled and self-controlled treatment was found. Warren and Thomas (2001) followed a similar procedure in which patients met regularly with a therapist who provided homework assignments and feedback. Some patients were also assisted in completing exposure exercises.

BT-STEPS (Baer & Greist, 1997) represents a special case; however, we suggest that it too is best represented as therapist-directed/self-conducted treatment. There is no "therapist" involved in BT-STEPS; rather, a computer program instructs the patient via the telephone as to which exposures to perform and when, based on a decision-making algorithm using the patient's anxiety scores (Greist et al., 2002). In addition, "patients may also record a personal message to a behavior therapist and receive the therapist's recorded answer within 72 hours" (Greist et al., 2002, p. 140). Thus, BT-STEPS cannot be considered a truly self-directed treatment, although it is self-conducted. The computer is essentially taking over the consultation (and perhaps accountability) roles that a therapist would ordinarily play; however, we also recognize that the computer cannot provide the support, motivation, and modeling that a live therapist can. Some degree of therapist involvement is possible within BT-STEPS; it is not known what percentage of patients access this feature, or how frequently.

Examination of treatment outcomes for therapist-directed/self-conducted ERP suggests that as the degree of therapist contact is decreased, a pattern of decreasing benefit appears. Treatment response rates between 38% and 60% are reported (Bachofen et al., 1999; Baer & Greist, 1997; Greist et al., 2002), and are lower than those using more therapist involvement, with 38% of BT-STEPS patients versus 60% of live therapist-directed/self-conducted ERP patients classified as responders, respectively (Greist et al., 2002). Further removal of the therapist, although still considered therapist-directed/self-conducted exposure, is seen in Fritzler, Hecker, and Losee (1997). In this study, patients were given a self-help book with periodic meetings with a therapist to review treatment progress and to offer feedback. Clinically significant change was found in 33% of patients.

Box 3 of the figure reflects self-directed/therapist-conducted exposure. To our knowledge, this model of treatment has not been studied empirically. Treatment of this kind would consist of a patient-generated exposure hierarchy, with the patient deciding which exposures would be done and when. However, the therapist would accompany the patient during exposures, thus providing modeling, consultation, support, motivation, etc. To some extent, this model is similar to a treatment model we have been using clinically in group therapy. Each patient is responsible for generating their own exposure hierarchy and deciding which exposure they will do on any given day. The therapist then assists the patient with the chosen exposure. However, in our groups the therapist still provides input into the design of the exposure hierarchy, and will occasionally suggest specific exposures; thus, this model of treatment has elements of box 2.

Box 4 of the figure represents "pure" self-administered treatment. This treatment is both self-directed, meaning the patient generates their own exposure hierarchy and decides which exposures to do and when; and self-conducted, meaning the patient engages in exposure exercises without the assistance of a therapist. Bibliotherapy

without any contact from a therapist is one (although not necessarily the only) form of self-directed/self-conducted treatment. The self-help treatment in our study of patients with OCD (Tolin, Hannan, Diefenbach, Maltby, & Worhunsky, 2004) can be considered a self-directed/self-conducted treatment. With the exception of an initial assessment and assignment to treatment condition, the patient has no contact with a therapist. Psychoeducation is derived from the book and patients develop their own hierarchy of exposures. At no point in treatment do patients receive feedback from a therapist. To date, 22.2% of patients receiving this treatment have experienced clinically significant change (Jacobson & Truax, 1991), compared to 66.7% of patients receiving therapist-directed/therapist-conducted (box 1) treatment.

MERGING SELF-ADMINISTERED AND THERAPIST-ADMINISTERED TREATMENTS

The arguments presented by Carmin and colleagues appear to promote the use of self-directed exposure without arguing against the usefulness of a therapist. We concur with this general line of reasoning. Self- and therapist-directed exposure, and self- and therapist-conducted exposure, may all play a significant role within the treatment setting. In terms of directing exposures, it is useful for both the therapist and patient to be involved in the decision-making process. In terms of conducting exposures, although the therapist may accompany the patient during initial exposures, it is helpful for the patient to engage in other exposure exercises without a therapist present. This is most in line with current clinical approaches in which the patient takes on increasing responsibility for determining and following through with exposures, and reducing dependence on the therapist. Essentially, treatment progresses from that which is described in box 1 to a combination of boxes 2 and 3 and ultimately to box 4.

We suggest that optimal treatment may involve a combination of treatment models shown in Figure 17.1. In the stepped-care model, patients begin with a low-intensity treatment such as bibliotherapy (box 4). Patients who do not respond to this intervention may move-up to a somewhat more intensive treatment such as therapist-directed/self-conducted ERP (box 2). If this treatment proves ineffective, patients are then given therapist-directed/therapist-conducted ERP (box 1). A stepped-care approach maximizes cost-effectiveness of treatment and, by virtue of its self-correcting nature, provides the patient with the least restrictive form of therapy needed for symptom improvement. However, stepped care may lead to somewhat higher dropout rates (Tolin, Diefenbach, Maltby, Hannan, & Muller, 2003). This issue requires further empirical study. A second possibility, suggested by Carmin and colleagues as well as by other authors (eg, Steketee, 1993), is to begin treatment with therapist-directed/therapist-supported ERP, and gradually introduce self-conducted and then self-directed ERP. The advantage of this procedure is that therapist involvement is gradually faded, decreasing the patient's reliance on the therapist and allowing the patient to attribute his/her success to internal, rather than external, factors. Increasing self-directed and self-conducted activity may also facilitate the spacing of sessions, which may improve long-term outcome (Rowe & Craske, 1998) but not short-term outcome (Tsao & Craske, 2000); however, we note that this could also be done within a therapist-directed setting.

In summary, self-administered exposures may play a substantial role in the strength and durability of ERP outcome. However, it is important to note that different studies use different definitions of "self-administered" treatment. Some use this term to indicate that patients have frequent sessions with a therapist, but complete exposures unaccompanied; others, to indicate that patients are responsible for all aspects of their treatment. Examination of study results suggests that the more active the role of the therapist, the better outcomes tend to be. Additional research is needed to determine how self- and therapist-administered exposures can be integrated into a unified treatment program.

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Reply to Tolin and Hannan:

SELF-DIRECTED VERSUS THERAPIST-DIRECTED TREATMENT: ADDITIONAL CONSIDERATIONS

Cheryl N. Carmin, Pamela S. Wiegartz, and Kevin D. Wu

While the purpose of this response is to highlight differences between issues raised in our chapter and those raised by Tolin and Hannan, we find there are more areas of agreement than disagreement. For example, neither chapter takes the position that successful OCD treatment is either entirely therapist-directed or entirely self-directed. Rather, we see the choice as between placing initial emphasis on one or the other strategy, and that this choice may be predicated on a variety of patient-based factors. As in most forms of CBT, we incorporate self-directed homework assignments that take place outside of the therapy session, but within the overall therapist-directed treatment plan. This practice does not appear to be at odds with Tolin and Hannan's approach. That being said, three points warrant further discussion.

AVAILABILITY OF TREATMENT

Tolin and Hannan note that one of the fundamental limitations of behavior therapy is that there are relatively few clinicians trained in CBT and even fewer who are skilled at treating OCD using ERP. It is difficult to take issue with this point, especially when we concede that our group (located in the third largest city in the United States) often has difficulty finding adequately trained clinicians to whom we can refer OCD patients. Further, given the scarcity of well-trained clinicians, Tolin and Hannan generally share our experience of having few openings for new patients and typically find it necessary to maintain a substantial waiting list. To complicate matters further, the greater a patient's distance from a metropolitan area or a major university/university medical center, the less likely it is they will find a clinician who is knowledgeable about (or practices) ERP for OCD.

On the other hand, it is worth noting that many of the studies Tolin and Hannan cited were published several years ago. Within the ensuing years, there has been an increasing amount of attention given to the need for evidence-based treatment both in clinical psychology and psychiatry. As an example, Nathan and Gorman

(1998) assembled leading treatment outcome researchers from both disciplines who reviewed and analyzed an enormous amount of information from several diagnostically specific literatures. Regarding OCD, Franklin and Foa (1998) reported that ERP has received the strongest empirical support and consequently is considered the psychosocial treatment of choice for this disorder. More recently, the Division of Clinical Psychology of the American Psychological Association formed a taskforce to examine what treatments are effective for specific disorders (see Chambless & Ollendick, 2000). While the findings of the taskforce received some criticism, the emphasis on consistent, positive, clinical research outcomes to support the use of an intervention has had an impact both on the mental health field and on the consumers. The Surgeon General's Report on Mental Health (Satcher, 2000), in fact, brought this information to the public in an understandable format. For anxiety disorders in particular, the evidence supporting CBT is compelling.

What, then, has the impact been of this movement toward evidence-based treatment? Over the years, university psychology departments have, at least in some cases, developed specialty clinics focusing on the treatment of anxiety disorders. Students who are trained in these settings are, at the very least, exposed to CBT. There are some graduate programs whose primary model of training now embraces CBT. Granted, even should the latter be the case, there is no guarantee that all graduate students will be assigned to directly treat a patient with OCD. There is, however, the potential that they will participate in a supervision group where at least one of their peers will be treating an OCD patient. Some students will have the opportunity to hone their skills in providing ERP, whereas others will have the opportunity to learn vicariously. Even if little has changed since the most recent of the studies noted by Tolin and Hannan (ie, Davison, 1998), there have been over 5 years worth of clinical psychology students who may have been trained in CBT and/or ERP. While our numbers may not be legion, they are growing.

Of note, the shift to an evidence-based perspective has not been the sole domain of clinical psychology. The accrediting body for psychiatry residency education now mandates that psychiatry programs include not only training in psychodynamic methods of psychotherapy, but that residents receive training in CBT, as well. Unfortunately, how this mandate has been interpreted varies from program to program with some residencies offering as little as a few months of exposure to CBT. At the other end of the spectrum, programs such as the one at our university integrate CBT theory and practice across all 4 years of psychiatric residency education. Since the article by Goisman et al. (1993) predated this change in residency requirements, there are many psychiatrists who have received at least some degree of training in CBT. As with their clinical psychology counterparts, there is still no guarantee that a resident will have the chance to treat an OCD patient.

The entire issue of how to best train psychologists and psychiatrists in ERP is a topic that receives minimal attention in the literature. In addition, one of the issues that often go unaddressed is how to best provide intensive treatment and how to generalize this method to the broader range of practice settings. In training clinics, it is far easier to assemble a team of clinicians or behavior technicians who can assist with providing the required number of treatment hours. In our setting, for example, intensive outpatient ERP may involve 10 or more hours of therapists' time. For most of our staff, it would be impossible for one person to be providing all these treatments single handedly. Rather, we utilize a team model including permanent staff, psychology

trainees, and psychiatry residents who are taught how to provide ERP. Once a trainee masters the necessary skills, he or she can assume leadership of a team and add OCD patients to his or her caseload. This method of treatment delivery clearly is demanding on the clinician and may not be possible for a solo practitioner. Rather, it is in this situation that therapist-directed exposure, by necessity, must incorporate self-directed exposure.

PATIENT EDUCATION AND TREATMENT ENGAGEMENT

Information about OCD treatment has become far more available to consumers. There are organizations (eg, Obsessive Compulsive Foundation, Anxiety Disorders Association of America) that are very active in providing up-to-date information to OCD sufferers and their families. What this often means is that the individual calling a clinic seeking treatment has some idea about what treatment may involve. To the extent that the OCD sufferer has read one of the several consumer-oriented books available that individual may even be quite knowledgeable.

There is, however, a problem associated with being self-educated about ERP. Given that the basic premise of treatment is to face one's fears, the notion of handling a contaminated object or purposefully abstaining from compulsive rituals that are believed to prevent some unwanted harm to a loved one may be overwhelming. For the purpose of self-managing their symptoms, many patients work hard to avoid such experiences and recoil at the thought of facing their fears head on. It would not be surprising if this concern contributed to why many OCD patients suffer for years before summoning the courage to seek out treatment. Thus, such education should not happen without due consideration of the patient's needs or fears. On the other hand, as clinicians, we sometimes do ourselves a disservice by providing too much education too soon about what treatment entails. As an example of this issue, a colleague was concerned that he was having a difficult time getting new patients who were calling to make inquiries about treatment to actually schedule an appointment. He noted that he was spending a considerable amount of time on the phone answering questions and providing information about the clinic and about treatment. When he outlined what he was telling new patients, it became clear that the detailed information about ERP that he was providing was scaring these potential patients away.

There is clearly a balance that must be struck between how much information to provide and also when and how to provide it. The issue of how to engage patients in treatment, whether therapist- or self-directed efforts, is an important consideration for treatment to be effective. If the patient feels overwhelmed with the treatment that is presented, it is likely that he or she will decline to initiate treatment, fail to appropriately engage treatment, or, as in the example above, drop out prematurely. It is with such patients that we see a clear benefit to initial therapist-led efforts. That is, the attentive therapist who notes patient apprehension or low confidence would be in a position to provide the necessary encouragement needed to begin treatment. In these situations, it might be the skill of the therapist that ultimately provides the prospective patient with the confidence to undergo this difficult and demanding therapy. A completely self-directed treatment (ie, via self-help manual) would not offer the patient such support or guidance.

In both of the preceding chapters, we underscored the importance of patient education. Certainly, self-help books and other sources of information can add to one's becoming a well-informed consumer. However, we have all had patients who have bought the books and then not followed the treatment plans outlined in these texts. Similarly, we have all worked with patients who did well when working with their therapist only to not actively participate in self-directed exposures. Another advantage we see of working with a therapist is that a clinician can adapt ERP to the individual patient's needs. Consider the preceding example of the patient hesitant to begin treatment: The therapist might tailor the initial exposures such that the patient stands the best chance to experience early success with the habituation process. This might involve selecting an exposure item that is lower on the hierarchy than would be the usual starting point for the more prepared patient. Such early success might then improve the patient's confidence and serve to help the patient engage more fully in treatment.

Additional benefits of therapist-led exposures include the opportunity to make "real time" assessments of the patient's speed of progress and the opportunity to provide specific and timely assistance if an impediment to treatment is encountered. Likewise, the therapist can assess whether the patient understands the nature of treatment and is able to generalize the basic principles of ERP to self-direct an increasing amount of treatment. Based on these assessments, the therapist then would have opportunity to make any appropriate changes to the treatment plan, including providing more education regarding the importance of the patient assuming more responsibility for his or her treatment. We view the shifting of emphasis from therapist- to patient-led exposures as critical to the long-term success of ERP. As such, determination of when and how to best achieve the shift toward greater patient responsibility for his or her exposure is extremely important and often requires clinical skill.

Thus, therapist-directed exposure clearly has certain advantages. Whereas it is possible that some computerized programs may be able to emulate some of the above aspects of the therapist's role, even a program as elaborate as BT STEPS is still not functioning at this level. *For some patients*, the flexibility and skill of the clinician affords the patient the best chance for success through an appropriately individualized treatment.

STEPPED MODELS OF CARE

Tolin and Hannan outline a program they are currently assessing, whereby initially patients participate in a self-help program. If a patient does not respond to this first phase, the patient then meets with a therapist who functions primarily as a consultant. Should treatment not be progressing at the second phase, the patient is then offered therapist-directed ERP. A clear advantage of this model is that it is cost-effective for patients who benefit from self-help efforts. However, the authors noted that their patients experienced only a 20% response rate with self-help treatment. Their results are consistent with the preliminary data they reported previously, whereby patients participating in therapist-directed treatment fared better than those engaged in self-directed treatment (ie, more than 50% reduction in symptoms versus 20% reduction).

Despite this concern, the notion of a stepped model of care is intriguing given how little we know about whether self- or therapist-directed treatment is preferential and for whom which of these approaches is best suited. It is noteworthy that a

different model of stepped care has been available for some time and is based on intensity/frequency of treatment. In this model, the degree to which the patient's OCD is disabling is what determines whether inpatient, intensive outpatient, or traditional outpatient treatment is recommended.

This latter interpretation of stepped care draws attention to the issue of symptom severity. Perhaps, a further refinement of the program described by Tolin and Hannan would involve a finer grained assessment of their prospective patients, followed by an attempt to assign/recommend treatment modality by degree of impairment. Those patients with relatively mild symptoms could be directed either to a self-help program or, if needed, to a program whereby the therapist functions as a consultant. More symptomatic patients might be better served by starting with a therapist-directed program that then gradually evolves into a more self-directed approach.

As we noted in our chapter, symptom severity, the presence of hoarding, religious or sexual obsessions, over-valued ideation, lack of family support, and/or comorbid disorders can mitigate against the use of a self-directed approach to treatment. These same patients are also likely to have the greatest difficulty engaging in treatment and maintaining a level of motivation that would ensure their on-going participation in a self-directed program of ERP. A thorough assessment to identify key variables that would serve to derail self-directed treatment may not only add to the cost-effective nature of the program that Tolin and Hannan outlined, but also enhance patients' motivation. Assigning severely disabled OCD patients to a self-help program would seem to have the potential of underscoring their inability to effectively self-manage their symptoms or, in some cases, to reinforce fears or maladaptive beliefs by means of aversive conditioning. Thus, patients may be destined to fail at the outset of their recovery process, which could then have a substantial impact either on their motivation or on their future engagement in or adherence to treatment.

Nevertheless, the stepped model of care that Tolin and Hannan have outlined has the advantage of potentially answering several questions we are raising: Is starting all OCD patients in a self-help program cost-effective? Should level of care be assigned based on variables such as symptom severity or other factors that indicate the need for a therapist-directed program? Is there a means of determining when is the best time to initiate therapist fading and thereby move from therapist-directed to self-directed treatment? Perhaps the most useful information to be learned through such study involves a better understanding of the specific variables that play a role in determining which patients will benefit from which of the different levels of care. We anticipate that these variables will include at least the domains of cognitive abilities, personality traits, and the presence of motivation-disabling comorbid conditions such as depression. Through fuller understanding of the combination of each of these potentially important variables, we might be in a position to offer a program of treatment that is maximally suited to serving the individual patient's needs and abilities.

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Chapter 18

COMBINING PHARMACOTHERAPY AND COGNITIVE-BEHAVIORAL THERAPY IN THE TREATMENT OF OCD

H. Blair Simpson and Michael R. Liebowitz

Two monotherapies are efficacious for adults with OCD: pharmacotherapy with serotonin reuptake inhibitors (SRIs, ie, clomipramine and the selective serotonin reuptake inhibitors) and cognitive-behavioral therapy (CBT) consisting of exposure and response prevention (ERP). Expert consensus treatment guidelines for adults with OCD (March, Frances, Carpenter, & Kahn, 1997) recommend ERP monotherapy be offered to every OCD patient when available and that it be the first treatment used with patients with milder OCD. Serotonin reuptake inhibitor monotherapy or SRI + ERP treatment is recommended for adults with more severe OCD. In this chapter, we examine data supporting the premise that combining SRI and ERP treatment is more effective than either treatment alone. We conclude that for adults with OCD, combination therapy (SRI + ERP) is warranted in specific clinical situations.

COMPARATIVE EFFICACY OF SRIS, CBT, AND THEIR COMBINATION: EVIDENCE FROM RANDOMIZED CONTROLLED TRIALS

Six randomized clinical trials in adults with OCD have examined whether combining SRIs and ERP treatments produced better outcomes than either treatment alone. A summary of findings from these studies appears in Table 18.1. Each study is reviewed in detail below.

Marks and Colleagues (1980)

Marks, Stern, Mawson, Cobb, and McDonald (1980) compared the outcome of 40 OCD patients who were randomly assigned to oral clomipramine (CMI) or pill placebo

TABLE 18.1. Comparisons between combination treatments and monotherapy in randomized controlled trials

Study	Main findings	Treatments (n of completers)	Y-BOCS total score		Comments
			Pre-mean (SD)	Post-mean (SD)	
Marks et al. (1980)	At week 7, CMI + ERP led to more improvement in rituals than ERP + PBO or CMI + R	CMI + ERP ($n = 10$)	—	—	Patients had varying degrees of depression. Neither CMI nor ERP treatment was optimized
		CMI + R ($n = 10$) ERP + PBO ($n = 10$) PBO + R ($n = 10$)			
Marks et al. (1988)	At week 8, CMI + ERP(s) superior to ERP(s) + PBO and CMI + AE on measures of rituals and depression. At Week 23, CMI + ERP(s&t) equal to ERP(s&t) + PBO	CMI + ERP(s&t)	—	—	On average, patients were not depressed. Neither CMI nor ERP treatment was optimized
		CMI + ERP(s) CMI + AE ERP(s&t) + PBO			
Cottraux et al. (1990)	At week 24, no significant group differences on OCD measures. FLV + ERP superior to ERP + PBO on measures of depression	FLV + ERP ($n = 16$)	—	—	Sample included many depressed patients; ERP treatment was not optimized
		FLV + AE ($n = 13$) ERP + PBO ($n = 15$)			
Hohagen et al. (1998)	At week 9, FLV + ERP superior to ERP + PBO on some measures (response rates, Y-BOCS obsession subscale) but not on total Y-BOCS scores; patients with comorbid depression fared better with FLV + ERP	FLV + ERP ($n = 24$)	27.9 (2.9)	12.4 (6.8)	Sample included many depressed patients; ERP included other techniques
		ERP + PBO ($n = 25$)	28.4 (3.8)	15.9 (7.9)	

TABLE 18.1. (*cont.*)

Study	Main findings	Treatments (*n* of completers)	Y-BOCS total score		Comments
			Pre-mean (SD)	Post-mean (SD)	
van Balkom et al. (1998)	At week 16, all active treatments led to significant decreases in OCD symptoms, with no between-group differences	FLV + ERP ($n = 18$)	24.7 (7.9)	12.6 (6.6)	Patients had mild depressive symptoms. Neither FLV nor ERP treatment was optimized
		ERP ($n = 19$)	25.0 (7.9)	17.1 (8.4)	
		CT ($n = 19$)	25.3 (6.6)	13.5 (9.7)	
		FLV + CT ($n = 14$)	27.2 (5.7)	15.6 (5.4)	
		WL ($n = 16$)	26.8 (6.4)	—	
Foa et al. (2005)	At week 12, all active treatments superior to PBO; both ERP + CMI and ERP superior to CMI; ERP + CMI equal to ERP alone on OCD measures	CMI + ERP ($n = 19$)	24.8 (4.2)	11.0 (8.1)	Patients with comorbid depression were excluded; CMI and ERP treatments were optimized (ERP was intensive)
		CMI ($n = 27$)	26.5 (4.8)	18.2 (7.8)	
		ERP ($n = 21$)	24.0 (4.6)	11.0 (7.9)	
		PBO ($n = 20$)	25.8 (4.8)	22.2 (6.4)	

Note. Y-BOCS means (and standard deviations) are presented for patients who completed treatment.
AE = Anti-exposure instructions, CMI = clomipramine, CT = cognitive therapy, ERP = exposure and response prevention, FLV = fluvoxamine, PBO = pill placebo, R = relaxation therapy, s = self-controlled; t = therapist-aided, WL = wait list control, Y-BOCS = Yale-Brown Obsessive-Compulsive Scale.

(PBO) plus 30 sessions of psychotherapy. The patients were adults (mean age: 30), 72.5% female, chronically ill (mean duration of OCD >10 years), and with varying degrees of depression (group mean scores on the 17-item Hamilton Depression Rating Scale [HAM-D; Hamilton 1960] ranged from 13.1 [SD = 8.6] to 18.3 [SD = 9.9]. The study design was complex: during weeks 0–4, patients were randomized to receive either CMI or PBO; during weeks 4–10, all patients were admitted to the hospital and received 6 weeks of inpatient psychotherapy consisting of 30 week-day 45-minute sessions. Half of the patients received 30 sessions of ERP; the other half received 15 relaxation sessions during weeks 4–7 and 15 ERP sessions during weeks 7–10. After week 10, patients were discharged from the hospital but remained on medication until week 36. Exposure and response prevention consisted of in vivo exposure and self-imposed response prevention; relaxation training (R) consisted of the therapist teaching the patient to tense and relax different parts of the body. Clomipramine or placebo was prescribed up to a maximum dose of 225 mg within 2 weeks unless side effects intervened. At week 10, the mean dose for all groups was 183 mg per day. Double-blind assessments focused on severity of rituals (eg, time spent), mood (eg, HAM-D), and general adjustment (eg, social life, leisure activities, and work).

Because of the complex design of this study, a direct comparison of the effects of CMI + ERP, ERP + PBO, and CMI + R could only be made at week 7. Between weeks 4 and 7, patients receiving CMI + ERP had more improvement in rituals (but

not mood or social adjustment) than patients receiving either EXRP + PBO or CMI + R. However, in an analysis of variance testing the effects of therapy, medication, and their interaction at week 7, there was no significant interaction effect. In addition to more improvement in rituals, patients receiving CMI demonstrated better compliance with exposure instructions both during and between sessions than patients receiving PBO. The authors concluded that CMI + ERP had a slight additive effect at week 7 and that CMI enhanced compliance with ERP.

The main limitation of this study is its complex design, making it difficult to evaluate the findings. The design did not provide a direct comparison of CMI + ERP versus CMI monotherapy within the same time period. Moreover, neither the CMI nor ERP treatments were maximized. Because it can take 6–10 weeks for CMI's full effects to appear; comparisons at week 7 likely underestimated the effects of CMI. Moreover, the CMI + ERP group had a mean dose of only 136 mg daily (versus the equivalent of 183 mg per day for the other groups); the dose associated with the greatest improvement in OCD symptoms during multicenter trials is 226 mg per day (Greist, Jefferson, Kobak, Katzelnick, & Serlin, 1995). Likewise, the ERP procedures were not clearly optimal (eg, 45 min sessions, insufficiently strict instructions for response prevention). Other limitations included: lack of detail about the diagnostic procedures, use of obsolete OCD outcome measures and the relatively small sample size that would make it difficult to detect small effects that might be clinically meaningful (only 10 patients per group). Despite these limitations, findings from this study suggest two potential advantages of combining CMI and ERP: (1) slightly improved reduction in rituals and (2) enhanced adherence with ERP procedures.

Marks and Colleagues (1988)

In a separate study, Marks et al. (1988) compared the outcome of 49 adult OCD patients. Once again, the study design was complex. Patients were randomly assigned to one of the following four treatments: 6 months of CMI and 23 weeks of anti-exposure instructions (CMI + AE); 6 months of CMI and 23 weeks of self-controlled exposure (CMI + ERP[self]), 6 months of CMI and 8 weeks of self-controlled exposure followed by therapist-aided exposure from week 8 to week 23 (CMI + ERP[self & therapist]); or 6 months of PBO and 8 weeks of self-controlled exposure followed by therapist-aided exposure from week 8 to week 23 (ERP[self & therapist]). With regards to the anti-exposure and self-controlled exposure therapy procedures, all patients were given homework instructions during the first eight weeks and were seen for 40 min at weeks 1, 2, 4, and 6. Patients receiving anti-exposure instructions were asked to avoid all contact with feared stimuli and to ritualize as much as they wanted. Patients receiving self-controlled exposure were encouraged to make contact with feared stimuli and to refrain from ritualizing for increasingly longer periods. During therapist-aided exposure, the therapist helped the patient do exposure tasks during 2 h weekly sessions; patients received between 5 and 15 of these sessions. Either CMI or PBO was prescribed up to a maximum dose of 200 mg per day unless the patient developed intolerable side effects. The mean dose for all CMI groups was 157 (SD = 49) at week 4, 146 (SD = 54) at week 8, and 127 (SD = 67) at week 17; the mean dose was not provided at week 23. Double-blind assessments were made at multiple time points and included measures of ritual severity (eg, time), mood (eg, HAM-D), and social adjustment.

At week 8, CMI + ERP (self, $n = 25$) produced significantly more improvement than ERP(self) + PBO ($n = 12$) on measures of rituals, depression, and social adjustment. However, at week 23, CMI + ERP (self & therapist, $n = 10$) showed no superiority over ERP (self & therapist) + PBO ($n = 8$). At week 8, CMI + ERP (self, $n = 13$) also produced significantly more improvement than CMI + AE ($n = 12$) on measures of rituals, depression, and social adjustment. However, 9 of the 12 cases receiving CMI + AE did so poorly that they were crossed over at weeks 12–17 to receive ERP (self & therapist), confounding further comparisons. The authors concluded that the combination of CMI and ERP had a small transitory additive effect compared to ERP + PBO.

The main limitation of this study was that treatments were not delivered optimally. The CMI groups achieved doses ranging only from 127 to 157 mg per day, substantially below the 'best' CMI dose for OCD of 226 per day established by Greist et al. (1995). The ERP procedures included either self-controlled exposure or a variable number (ie, 5–15) of therapist-aided sessions delivered weekly. In addition, it is unclear what impact the anti-exposure instructions had on CMI treatment or what effect self-controlled exposure prior to therapist-aided exposure had on ERP outcome. Interestingly, assessors were able to guess correctly 90% of the time whether patients received CMI or PBO, suggesting inadequate blinding procedures. Other limitations included the use of obsolete measures of OCD and the relatively small sample size limiting power to detect small effects that might be clinically meaningful. Despite these limitations, findings from this study suggest that combined CMI + ERP treatment can produce more gains in the short-term than ERP + PBO, but indicate that this gain may be temporary.

Cottraux and Colleagues (1990)

Cottraux et al. (1990) compared the outcome of 60 adults with OCD who were randomized to receive 24 weeks of fluvoxamine (FLV) with ERP (FLV + ERP), FLV with anti-exposure instructions (FLV + AE), or ERP + PBO. Of note, 43% of the patients who completed the study met diagnostic criteria for major depression or dysthymic disorder (mean 17-item HAM-D score was 19). Fluvoxamine was prescribed up to 300 mg per day as clinically indicated. In the anti-exposure condition, patients were instructed to avoid feared situations or stimuli; however, most did not complete their anti-exposure homework. Exposure and response prevention consisted of eight weekly sessions with a therapist that included self-controlled exposure between sessions and imaginal exposure during sessions followed by 16 weeks of therapist-guided exposure. Symptom severity was evaluated by both self-report and assessors blind to treatment condition. Outcome measures included the severity of rituals (eg, duration of rituals per day) and depression. A reduction of greater than 30% in the total duration of rituals per day as reported by the patient was used as a global criterion of treatment effectiveness.

After 24 weeks of treatment, all groups improved on some measures of rituals and depression, but only the FLV + ERP improved on all measures. In addition, FLV + ERP produced significantly more improvement in depression than did ERP + PBO. The combined treatment group had the largest percent reduction in the duration of rituals per day (FLV + ERP = 46%; FLV + AE = 42%; ERP + PBO = 25%) and the largest percentage of patients who, by self-report, had more than a 30% reduction in rituals

per day (FLV + ERP = 69%; FLV + AE = 54%; ERP + PBO = 40%); however, these between group differences were not statistically significant. The authors hypothesized that the lack of between group differences for OCD measures was due to inadequate statistical power.

This study had several major limitations. First, patients in the different groups did not necessarily receive the intended treatment: the FLV + AE group complied minimally with the anti-exposure instructions and other psychosocial interventions were provided "as needed" to patients receiving ERP. In addition, although patients "had to take" FLV up to 300 mg per day and medication compliance was high (as measured by a record of the pills taken), the mean dose prescribed at week 24 for the groups receiving FLV was not reported. Second, the ERP protocol (eg, once weekly sessions of an unspecified duration with no mention of response prevention) was not optimal (Abramowitz, 1996). Third, full data were presented only for the 44 subjects that completed 24 weeks of treatment; thus, the comparisons above were made on only 13 patients who received FLV + AE, 16 patients who received FLV + ERP, and 15 patients who received ERP + PBO. These relatively small sample sizes limit the power to detect significant group differences. Despite these limitations, findings from this study suggest that combination treatment may be somewhat superior to monotherapy in adults with OCD and comorbid depression.

Hohagen and Colleagues (1998)

Using a more straightforward research design, Hohagen et al. (1998) compared the outcome of 58 adults with OCD randomly assigned to receive either FLV + ERP or ERP + PBO. Many patients had comorbid psychiatric conditions including mood, anxiety, and personality disorders (mean 21-item HAM-D score was 19.0). On average, patients had moderate to severe OCD symptoms (mean baseline Yale-Brown Obsessive Compulsive Scale [Y-BOCS; Goodman et al., 1989a, 1989b] total score was in the high 20s), and many had received prior treatment (84% had taken medication, 34.7% had previous ERP). Fluvoxamine was started at 50 mg per day and increased to a maximum dosage of 300 mg within 5 weeks. The mean dose was 288.1 mg (range 250–300 mg), which is above the "best" FLV dose for OCD of 249 mg per day as established by Greist et al. (1995). Exposure and response prevention sessions were held weekly for at least 3 h and included therapist-aided exposure. An assessor who was blind to treatment condition evaluated symptom severity using the Y-BOCS and 21-item HAM-D. Patients were considered responders if they had a Y-BOCS reduction of at least 35%.

After 9 weeks of treatment, both groups showed significant reductions in Y-BOCS scores. However, there were significantly more responders (defined as a Y-BOCS reduction of at least 35%) in the FLV + ERP group (87.5%) than in the ERP + PBO group (60%). Secondary analyses revealed the following: (*a*) both groups improved significantly and comparably on compulsions, but the FLV + ERP group improved significantly more on obsessions; and (*b*) patients with comorbid depression fared better if they received FLV + ERP. Therefore, the authors concluded that combination therapy should be used when obsessions dominate the clinical picture or when a secondary depression is present.

This study also had several limitations. First, patients with poor previous response to ERP were accepted, but their distribution in the two treatment groups was not reported. Second, ERP was a "multimodal psychotherapy," and included not just

exposure, but also the development of "alternative behaviors" and cognitive restructuring; exactly what the latter two techniques entailed, and how much session time was devoted to them, is not clear. Third, whether therapists adhered to the treatment manual was not formally assessed. Despite the limitations, these findings provide empirical support for the use of combined ERP and SRI treatment in OCD patients when obsessions dominate or depression is present.

van Balkom and Colleagues (1998)

van Balkom et al. (1998) compared the outcome of 117 patients randomized to one of the following five conditions: FLV + ERP, ERP, cognitive therapy (CT), FLV + CT, or wait list control (WL). Seventy patients completed the 16 weeks of treatment, and 16 patients completed the 8-week wait list condition. On average, patients had moderately severe OCD symptoms at baseline and mild depressive symptoms. Data on comorbidity were not reported. Fluvoxamine was given to a maximum of 300 mg, although the mean FLV dose at week 16 in both FLV conditions was only 197.1 mg (SD = 82.0 mg). All therapy sessions were 45 min long, and ERP consisted of gradual self-controlled exposure in vivo with gradual self-imposed response prevention. Assessments were conducted by evaluators blind to treatment assignment.

At week 16, all active treatments led to a significant decrease on all OCD measures, and all active treatments except for ERP alone showed a significant decrease on the BDI. There were no significant differences in outcome between the treatment groups on any measures. Because ERP alone was as efficacious as FLV + ERP in this study, the authors concluded there were no reasons to combine SRIs and CBT in OCD adults with overt compulsions and without severe comorbid mood disorder.

This study had several limitations. First, it used a less than optimal ERP protocol including weekly 45 min sessions with all exposure being self-controlled ERP being gradual and self-imposed. Second, the average dose of FLV was less than optimal. Third, the combination group received only 10 ERP sessions, whereas the group receiving ERP alone received 16 sessions. Fourth, full results were only presented for the completer sample, despite the fact that 31 patients dropped out prior to completion, and there were differential dropout rates between the treatment groups. Fifth, many patients had received the study treatments previously; yet there is no mention of whether prior treatment was effective. This raises the possibility that some treatment groups contained treatment-refractory patients. Because of these limitations, this study may not have been a fair comparison of whether combined SRI + ERP treatment can produce superior outcome to ERP monotherapy.

Foa and Colleagues (2005)

Foa et al. (in press) compared the outcome of 122 adult OCD patients randomized to intensive ERP, CMI, CMI + ERP, or PBO. Baseline OCD severity was moderate to severe. Major depressive disorder (with a HAM-D > 18) was a criterion for exclusion; thus, patients on average had minimal depressive symptoms. Clomipramine was given up to 250 mg per day if tolerated and clinically indicated; mean daily doses during the last week for all who entered and for all who completed (respectively) were 196 and 235 mg for CMI patients, and 163 and 194 for CMI + ERP patients. Intensive ERP consisted of two information-gathering sessions, fifteen 2-h exposure sessions

conducted every weekday for 3 weeks, two home visits, and daily ERP homework during the first 4 weeks. This was followed by eight 45-min weekly maintenance sessions during the next 8 weeks. Patients receiving CMI + ERP began both treatments simultaneously. Double-blind assessments were conducted both pre- and posttreatment. The main outcome measure was the Y-BOCS.

At posttreatment (week 12), all active treatments were superior to PBO. A combination of CMI + ERP was superior to CMI alone on all outcome measures, but failed to show superiority over ERP alone in any analyses. Intent-to-treat and completer sample response rates, respectively, were: 70% and 79% for CMI + ERP, 42% and 48% for CMI, 62% and 86% for ERP, and 8% and 10% for PBO ($P < 0.001$). The authors concluded that: (*a*) CMI, ERP, and their combination are all efficacious treatments for OCD; (*b*) intensive ERP is superior to CMI, and thus by implication may be superior to monotherapy with other SRIs; and (*c*) CMI + ERP treatment was not significantly more effective than ERP monotherapy.

This study provided empirical support for the superiority of CMI + ERP combination treatment over CMI monotherapy; however, combination treatment was not significantly better than ERP monotherapy. Nevertheless, several factors may have limited the sensitivity of this study's design to potential combined treatment effects: most patients in the combined group (unlike most in the CMI group) did not achieve the maximum dose of CMI; the potency of intensive ERP alone left little room for further improvement, and the simultaneous instigation of ERP and slow upward titration of CMI meant that the intensive phase of ERP was completed (within the first 4 weeks) before the full impact of CMI could be realized. The added benefit of CMI might be more evident in individuals for whom ERP alone is too distressing and SRI pretreatment might make exposure tasks less difficult, in patients with comorbid depression (who were excluded from the study), or if the ERP protocol consisted of weekly sessions, as is more typical of routine clinical practice. Other limitations of this study include the lack of data on patients who dropped out after randomization and before any treatment commenced, the lack of formal data on interrater reliability, and the absence of any systematic data on prior treatment history. Despite these limitations, this study provides clear empirical support for the superiority of intensive ERP + CMI over CMI monotherapy, although not over ERP monotherapy.

Summary of the Randomized Controlled Trials

In summary, although both ERP and SRI monotherapy have been shown in multiple randomized controlled trials to be efficacious for adults with OCD, only a handful of trials (Table 18.1) directly address whether the combination of SRI and ERP is superior to either treatment alone. Of the few studies that do address this issue, limitations in their design and/or procedures prevent definitive conclusions, as discussed above. Few of the studies had adequate sample sizes to detect small differences between treatments, even if they did exist. Moreover, some studies excluded patients with significant comorbidity (even though comorbidity is common in OCD), and these might be the patients who would show the greatest benefit from combination treatment. Finally, certain design decisions (eg, how the different treatments were delivered) may have prevented the detection of important differences between combination treatment and monotherapy.

Despite these problems with the extant literature, the available data support the premise that combination therapy can be superior to monotherapy in some OCD patients, but is not necessary for all OCD patients. In particular, combination therapy has been shown to be superior to ERP monotherapy in OCD patients with comorbid depression or when obsessions dominate the clinical picture (Hohagen et al., 1998). Combination therapy has also been shown to be superior to SRI monotherapy in OCD patients without comorbid depression (Foa et al., in press).

LONG-TERM BENEFITS OF COMBINATION TREATMENT

In the review above, we focused on the short-term efficacy of combination treatment (SRI + ERP) versus monotherapy (SRI or ERP). An important and related question is whether combination therapy produces superior long-term outcome to monotherapy. There are limited research data that address this issue.

Two important questions regarding long-term outcome include: (*a*) does long-term maintenance treatment that combines ongoing SRI treatment with maintenance ERP sessions produce better long-term outcome than either maintenance SRI or ERP monotherapy; and (*b*) does combination treatment (SRI + ERP) lead to better maintenance of gains than monotherapy after treatment discontinuation. We know of no study that directly examined the former. With regards to the latter, several studies have compared the outcome of adult OCD patients who received ERP, SRI, or combination treatment after treatment discontinuation (Cottraux et al., 1990; Cottraux, Mollard, Bouvard, & Marks, 1993; de Haan et al., 1997; Marks et al., 1980, 1988; van Balkom et al., 1998); most used designs that confounded the different treatments' effects (eg, crossing over CMI nonresponders to ERP or providing patients additional treatment after treatment discontinuation as needed). None of these studies compared the effects of ERP, SRIs, and their combination after sustained treatment discontinuation using evaluators that were blind to treatment assignment.

In a recently completed trial, we compared the posttreatment effects of intensive ERP and CMI (arguably the two most efficacious treatment for OCD) by following treatment responders and assessing relapse blind to original treatment (Simpson et al., in press). In this study, responders to intensive ERP or ERP + CMI had a significantly lower relapse rate and longer time to relapse after treatment discontinuation than did responders to CMI alone (relapse rate of 12% versus 45%, respectively). These results suggest that patients receiving combination therapy fared substantially better after treatment discontinuation than patients who received CMI alone. However, the study was designed to compare ERP and CMI relapse rates only when the two groups receiving ERP were combined. Because the study did not have the power to detect small differences between the ERP and ERP + CMI groups, it is not surprising (and not particularly informative) that no significant differences between these two groups were seen in either relapse rates or time to relapse.

In summary, data comparing the long-term efficacy of combination treatment and monotherapy for OCD are limited. Further study is needed given that OCD is a chronic disorder and patients routinely receive long-term SRI treatment. Extrapolating from the one study that exists (Simpson et al., in press), we conclude that patients who receive combination treatment fare better after treatment discontinuation than

patients who receive CMI alone. However, it is not clear whether combination treatment affords any advantages over ERP monotherapy in its posttreatment effects after treatment discontinuation.

CLINICAL EXPERIENCE COMBINING MEDICATION AND CBT

Randomized controlled trials provide important data on the relative efficacy of specific treatment programs. Overall, the data suggest that there may be some benefit of combination treatment in some OCD patients, although only two studies found unambiguous effects (Foa et al., in press; Hohagen et al., 1998). It is therefore important to emphasize the limitations of this literature and thus the incomplete answer these data provide. First, there are only a handful of well-controlled studies that directly compare combination treatment to monotherapy in OCD. These studies used different ERP protocols, different SRI medications, and different study designs, and none had sufficient power to detect small differences between the different treatment groups. Second, many of the well-controlled studies used a simultaneous treatment design, whereas a sequential design (eg, SRI treatment followed by ERP treatment for SRI partial responders) may better demonstrate the benefits of combined treatment over monotherapy. Third, these studies enrolled all patients with OCD; however, combination treatment may be more beneficial for certain subsets of OCD patients (eg, partial responders to monotherapy, patients with comorbid depression) than for others.

In summary, current data from controlled trials can only partially address whether combination treatment is superior to monotherapy. Moreover, data from randomized controlled trials can never address whether combination treatment is superior to monotherapy for a particular OCD patient. As a result, we review below six situations in which combination treatment is typically used in clinical practice and any evidence that supports this use.

The Treatment of Comorbidity that Might Interfere with ERP

Patients with OCD have high rates of comorbid psychiatric conditions: comorbid depressive disorders and other anxiety disorders are the most common (Fireman, Koran, Leventhal, & Jacobson, 2001; Karno, Golding, Sorenson, & Burnam, 1988; Rasmussen & Eisen, 1992). Some of these comorbid disorders are known to interfere with ERP. For example, depression has been found to be a predictor of poor ERP outcome (Abramowitz, Franklin, Street, Kozak, & Foa, 2000; Steketee, Chambless, & Tran, 2001), and generalized anxiety disorder has been found to predict ERP dropout (Steketee et al., 2001). A recent case series (Gershuny, Baer, Jenike, Minichiello, & Wilhelm, 2002) suggested that comorbid posttraumatic stress disorder may also lead to poor ERP outcome.

These comorbid depressive and anxiety disorders that are common in OCD are also responsive to medication (Foa, Davidson, & Frances, 1999; Gorman 2003; Karasu, Gelenberg, Merriam, & Wang, 2000). As a result, it is logical to assume that treating the comorbid disorder first with medication may promote ERP outcome in these patients. This hypothesis has not been formally tested, but it is supported by findings from one

of the studies reviewed above (Hohagen et al., 1998): depressed OCD patients who received combination treatment (SRI + ERP) had a significantly greater reduction in their OCD symptoms than patients receiving ERP alone.

Enhancing Adherence with ERP

Exposure and response prevention is an efficacious treatment; however, its effectiveness in clinical practice is limited by patient refusal, premature discontinuation, and partial adherence. Patients who poorly adhere to ERP do not improve. It is estimated that in most service settings, up to half of OCD patients either refuse or dropout out of ERP treatment; and clinical experience suggests many that complete ERP do not fully adhere with ERP procedures. Thus, methods that enhance ERP adherence could greatly improve outcome.

There are many possible factors that can lead to poor adherence (Foa, Steketee, Grayson, & Doppelt, 1983b), including patient factors (eg, comorbidity, poor insight, and anxiety sensitivity), therapist factors (eg, lack of empathy leading to poor therapeutic alliance), and situational factors (eg, lack of time or money). In clinical practice, OCD patients with trouble adhering to ERP are often started on SRI medication. The rationale for this decision is that if OCD severity were reduced by SRI treatment (leading to more symptom-free periods, increased insight into the irrationality of OCD fears, reduced anxiety sensitivity, and reduced depression), patients would be better able to comply with ERP. This idea has not been formally tested (and below we discuss the possibility that medication may interfere with ERP). However, one study (Marks et al., 1980) found that patients who received CMI had higher compliance ratings with ERP instructions than those receiving placebo. The mechanism for this improved compliance was not examined.

Treatment of Partial Responders to Monotherapy

Although SRIs and ERP are both efficacious as monotherapies for OCD, the Y-BOCS scores in Table 18.1 indicate that these therapies rarely (if ever) eliminate all symptoms. In fact, in our multisite trial comparing CMI and EX/RP (Foa et al., in press), up to one-third of those patients who responded to CMI still had sufficient residual OCD symptoms at the end of treatment to merit entry into most clinical trials (ie, Y-BOCS $\geq$ 16). Thus, partial response to treatment is common; and complete remission is rare, regardless of the monotherapy.

For patients with partial response to SRIs, treatment guidelines recommend augmenting with ERP. There is some empirical support for this: In an open trial of OCD patients who had received an adequate trial of SRI pharmacotherapy, the addition of twice-weekly ERP led to significant further reduction in OCD symptoms (Simpson, Gorfinkle, & Liebowitz, 1999). These preliminary findings are currently being examined in an ongoing multisite (Columbia University and the University of Pennsylvania) randomized controlled trial in which we are comparing the efficacy of ERP to stress management therapy in patients with residual OCD symptoms despite an adequate SRI trial. Preliminary findings from this trial indicate that ERP can successfully augment response in SRI partial responders. In addition to empirical support for the efficacy of adding ERP to SRI partial responders, this strategy may make

practical sense in service settings where SRIs are more widely available and require less provider time and effort than does ERP.

We are not aware of any studies that have examined whether it is efficacious to add SRI medication to patients who have partially responded to ERP therapy. However, this is a strategy sometimes used in clinical practice when patients are having trouble responding to ERP monotherapy or when they threaten to drop out of ERP due to difficulties adhering to the protocol.

Reduction of Relapse

SRI Relapse

Serotonin reuptake inhibitor relapse rates are reported to be as high as 89% (Pato, Zohar-Kadouch, Zohar, & Murphy, 1988). In our trial comparing ERP and CMI treatment (Foa et al., in press; Simpson et al., in press), we found that 45% of patients who responded to CMI returned to their pretreatment severity level within 12 weeks after treatment discontinuation. In an open-label study, Ravizza, Barzega, Bellino, Bogetto, and Maina (1996) found that the cumulative proportion of patients who relapsed 2 years after CMI, FLV, or fluoxetine discontinuation was greater than 75% and significantly greater than the relapse rates of patients who continued on SRIs. These data indicate that many OCD patients who discontinue their SRI (regardless of which one) will eventually suffer a worsening of their OCD symptoms. Thus, OCD treatment guidelines (March et al., 1997) recommend that patients remain on SRI medication for at least 1–2 years, and sometimes for life.

However, many patients, even if they have a good response to SRI treatment, eventually consider stopping their medication. Reasons include: the intention to become pregnant, troubling long-term side effects (eg, lack of libido or inability to achieve orgasm), and/or the desire to be free of taking daily medication. Cognitive-behavioral therapy has been used to help patients with panic disorder discontinue benzodiazepine treatment (Otto et al., 1993); similarly, ERP might help patients with OCD discontinue SRI treatment without relapse.

There is some data to support this strategy. Baer et al. (1994) described 6 OCD patients who were treated with either fluoxetine ($n = 5$) or CMI ($n = 1$) for at least 6 months, and then received ERP during the tapering and discontinuation of their SRI. Two patients remained well 6 and 9 months after discontinuing their respective medication regimes (Patient 1: fluoxetine, 80 mg per day; Patient 2: CMI, 200 mg per day). Patient 3 remained well for 18 months after discontinuing fluoxetine 80 mg per day, but then suffered a relapse and restarted the medication with good response. Patients 4 and 5 suffered relapses: Patient 4 experienced increases in depression and obsessions 3 and 4 weeks after fluoxetine discontinuation, and Patient 5 suffered a return of OCD symptoms 2 months after fluoxetine discontinuation. Patient 6 tapered fluoxetine from 80 to 20 mg per day (but did not discontinue) and remained well at 8-month follow-up. The authors concluded that some patients who received ERP while discontinuing their SRI could remain well for 6 months or more. However, this strategy did not work for all patients.

In our multi-site trial comparing CMI and ERP (Foa et al., in press), we found that 33 ERP responders (whether on or off CMI) had a significantly lower relapse rate and longer time to relapse 12 weeks after treatment discontinuation than did 11

patients who responded to CMI treatment alone (12% versus 45%; Simpson et al., in press). These data indicate that adding ERP to CMI protects against CMI relapse, at least in the short-term. One difference between our study and that of Baer et al. (1994) is that patients who received combination treatment received ERP in our study while starting CMI (as opposed to while discontinuing CMI). Thus, although both studies suggest that combining SRI medication with ERP treatment can help some patients lower or discontinue their SRI medication, it is not clear when the optimal time is for patients to receive ERP to minimize SRI relapse.

ERP Relapse

Available data indicate that relapse following successful ERP treatment is low. In a review of 16 studies, Kozak and Foa (1996) concluded that patients receiving ERP (with or without concomitant medication) were unlikely to lose their gains: of 376 treated patients, 76% were responders at follow-up (mean, 29 months). One caveat is that some of these patients received additional treatment during follow-up. In our trial comparing ERP and CMI, ERP responders (with or without concomitant CMI) had a combined relapse rate (defined as a return to pretreatment severity) of only 12% in the 12 weeks after treatment discontinuation (Simpson et al., in press). These data indicate that most ERP responders (with or without concomitant medication) can maintain their gains for at least 12 weeks in the absence of additional treatment. At the same time, clinical experience indicates that not all ERP responders maintain their gains long-term. Some data indicate that partial response to acute ERP treatment predisposes one to subsequent relapse (Foa et al., 1983a, 1983b; Simpson et al., in press). Thus, it is possible that adding SRIs for partial ERP responders might protect against relapse, yet this has not been formally tested.

The Treatment of Severe OCD

Treatment guidelines for OCD (March et al., 1997) recommend that when OCD is more severe, combination treatment is preferable to monotherapy. However, severity has not consistently been found to be a predictor of poor ERP outcome (Steketee & Shapiro, 1995). Pilot data from our ongoing trial comparing ERP and stress management therapy as augmentative strategies for SRI partial response indicate that good ERP participation (ie, completion all ERP sessions with good adherence) was associated with good outcome regardless of baseline severity. These data illustrate that ERP monotherapy can be very successful, even for severely ill OCD patients, if patients adhere to the protocol. On the other hand, if SRI medication improves ERP adherence in severely ill OCD patients by any mechanism (see the discussion above), then combining SRI medication and ERP may be a useful strategy and is often done in clinical practice. We do not know all the determinants of ERP adherence.

To Minimize SRI Exposure

There are certain clinical situations in which the goal is minimal exposure to SRI medication (eg, pregnancy and childhood). Data from our trial comparing ERP and CMI (Foa et al., in press) indicate that although the simultaneous combination of intensive ERP and CMI was not superior to ERP monotherapy, it was superior to

CMI monotherapy, despite the fact that the combination group received a lower dose of CMI (mean daily doses for the ERP + CMI versus CMI completers: 194 (48) versus 235 (34)). If only treatment responders are examined, this finding holds: responders receiving ERP + CMI had a significantly lower CMI dose than responders receiving CMI alone. There are two implications of these results. First, if the patients receiving combination treatment had received the maximum dose of CMI, combination therapy may have proved superior to ERP alone. Second, combination therapy can produce superior results to SRI monotherapy with lower SRI doses. Thus, clinical situations that involve SRI treatment but necessitate the lowest possible doses might benefit from combination treatment.

POSSIBLE CONTRAINDICATIONS FOR COMBINING MEDICATION AND CBT

The research and clinical data reviewed above indicate that combining SRI medication with ERP may be an effective approach for certain OCD patients. However, it is worth considering potential contraindications to combining SRIs and ERP.

Does the Addition of Medication Undermine the Long-Term Efficacy of ERP?

One theory explaining how ERP (and other cognitive-behavioral treatments for anxiety disorders) works is based on emotional processing theory (Foa & Kozak, 1986). In this model, fear is conceptualized as a cognitive structure in memory. This cognitive structure contains information about feared stimuli, fear responses, and the meaning of these stimuli and responses. When people face a realistically threatening situation, this fear structure supports adaptive behavior. However, when the fear structure does not reflect reality (eg, threat meaning is attached to harmless stimuli), the pathological fear structure leads to pathological anxiety responses as seen in anxiety disorders. Thus, to treat the pathological anxiety responses, one must modify the threat meaning within the pathological fear structure.

Foa and Kozak (1986) proposed that therapies known to reduce fear, such as ERP for OCD, achieve their effect by modifying pathological fear structures. To do this, two conditions must be present: first, the fear structure must be activated; and second, information that is incompatible with the pathologic aspects of the fear structure must be available and incorporated into the existing structure. On the basis of this model, it is theoretically possible that the addition of medication to CBT may impede its outcome. Specifically, the reduction of anxiety by medication may block fear activation; and without fear activation, the fear structure cannot be modified successfully by CBT.

To examine whether combining treatments influences efficacy of either monotherapy, Foa, Franklin, and Moser (2002) reviewed randomized trials that compared combined treatments (medication + CBT) versus monotherapies (medication or CBT) in various anxiety disorders. They concluded that at posttreatment and follow-up, neither the relative effect sizes nor the percentage responder rates indicated any disadvantage of combined treatment over CBT alone for OCD, social phobia, and generalized anxiety disorder. Their conclusion for OCD is based on four of the studies reviewed above (Cottraux et al., 1990; Foa et al., 2005; Hohagen et al., 1998; van Balkom

et al., 1998) and only addressed concomitant SRI medication. Thus, these data suggest that SRIs do not impede ERP outcome in OCD. However, it is possible that other medications, such as benzodiazepines, might interfere with ERP treatment.

It is worth noting that in the four studies reviewed by Foa et al. (2002), the effect sizes they calculated for the combined SRI + CBT treatments were larger than the effect sizes for any of the monotherapies (Cottraux et al., 1990: FLV + ERP = 1.89, FLV = 1.37, ERP + PBO = 1.00; Foa et al., 2005: ERP + CMI = 2.14, CMI = 1.28, ERP = 2.01, PBO = 0.64; Hohagen et al., 1998: FLV + ERP = 2.97; ERP + PBO = 2.02; van Balkom et al., 1998: FLV + CBT = 1.85, CBT = 1.20). Thus, among OCD patients, combining SRIs and ERP had stronger effects than any monotherapy, even though only one study (Hohagen et al., 1998) found a statistical advantage (on some OCD measures) for combined treatment over ERP alone and only one study (Foa et al., in press) found a statistical advantage for combined treatment over SRI monotherapy.

Are There Patients for Whom the Combination May Be Harmful?

Situations in which the clinician should be cautious about using either ERP or SRI monotherapy would also be situations in which their combination may not be ideal. For example, it is generally not recommended to pursue ERP treatment when the patient has an unstable medical disorder, is severely suicidal, is psychotic, or is undergoing a life crisis. The reason is that ERP treatment itself is stressful and patients need to be willing and able to tolerate the distress that is necessarily triggered by these treatment procedures. Certain patients are not only unlikely to benefit from ERP, but may also be negatively impacted by the procedures. Examples include psychotic patients who could become disorganized during stressful exposure exercises, suicidal patients who might feel more hopeless in the face of this additional source of anxiety, or cognitively impaired patients who could become confused or disoriented by the homework. Such patients, even if stabilized on medications, would need to be carefully evaluated before adding ERP.

Treating pregnant or nursing women with medication always requires careful consideration. Here, ERP monotherapy would be preferable to SRI treatment to avoid any potential negative effects of the medications on the fetus or newborn. If poor response to ERP was encountered and the clinician was considering SRI treatment but wanted to reduce medication exposure, combination treatment may be a better strategy than SRI monotherapy given data that combination treatment can produce a superior outcome with lower SRI doses compared to SRIs alone (Foa et al., in press).

CONCLUSION

Six randomized trials have examined the relative efficacy of SRI pharmacotherapy with and without ERP. One (Hohagen et al., 1998) found that combination treatment (SRI + ERP) was superior to ERP monotherapy for obsessions or OCD with comorbid depression. Another (Foa et al., in press) found that combination treatment was significantly superior to SRI monotherapy, but not ERP alone, when skilled therapists delivered ERP in an intensive protocol to patients who were not depressed. Of the

other four studies, all (Cottraux et al., 1990; Marks et al., 1980, 1988) but one (van Balkom et al., 1998) found that combination treatment (SRI + ERP) resulted in improved OCD outcome when compared to monotherapy. However, these differences were either not statistically significant or did not persist over time. Importantly, each of these studies had limitations that may have precluded detecting significant group differences.

Clinical experience indicates that combination treatment is useful in specific situations: to treat comorbidity that might interfere with ERP, to enhance ERP adherence, to help partial responders to monotherapy, to reduce SRI relapse, to treat severe OCD, or to minimize SRI exposure. Some of these uses have empirical support in the research literature. Together, research data and clinical experience support the premise that combining SRI and ERP treatment in OCD patient is more effective in certain clinical situations than either treatment alone. However, the optimal timing and method for combining SRI and ERP treatments remains largely unknown and deserves further study.

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Chapter 19

COMBINING SEROTONERGIC MEDICATION WITH COGNITIVE-BEHAVIOR THERAPY: IS IT NECESSARY FOR ALL OCD PATIENTS?

Martin E. Franklin

Literature reviews presented in earlier chapters of this book and elsewhere (eg, Franklin & Foa, 2002) have already explicated that cognitive-behavioral therapy (CBT) and serotonin reuptake inhibitors (SRIs) are both efficacious treatments for OCD; yet at the same time, response to either of these monotherapies is neither universal nor complete. One can readily hypothesize an advantage for combined treatment by CBT plus SRI over either monotherapy alone, and prevailing belief in the veracity of this view is already reflected in the field in a number of different ways. Commenting on clinical practice with anxiety disorders more broadly Balon (2004, p. 63) states, "A combination of proven pharmacotherapies and psychotherapies may be the most clinically prudent approach to the treatment of anxiety disorders." Regarding OCD specifically, the OCD Expert Consensus Guidelines (March, Frances, Carpenter, & Kahn, 1997) recommended that for adults and adolescents with an initial Yale-Brown Obsessive-Compulsive Scale (Y-BOCS; Goodman et al., 1989a, 1989b) score of 18 or higher, combined treatment should be considered the first-line option, along with SRI alone for adults (March et al., 1997). Some have advocated even more strongly for the relative advantages of combined treatment over monotherapies (eg, Greist, 1992; Greist & Baer, 2002). For example, on the Obsessive Compulsive Foundation's website there is a feature called "How to Select a Behavior Therapist (http://www.ocfoundation.org/ocf_0003.htm)," written by Dr Michael Jenike, who states that, "Over the last two decades, it has become increasingly clear that optimal treatment for most people with OCD involves the combination of medication plus the behavior therapy techniques of exposure and response prevention (EX/RP)." The purpose of this chapter is to consider whether initial treatment with CBT plus concomitant SRI should indeed be recommended for most OCD patients. In deliberating on this issue below, I have included discussion of practical, theoretical, and empirical

factors, and conclude that the answer to this important question requires at least some qualification.

PRACTICAL CONSIDERATIONS

Cost

Patients who are taking adequate doses of nongeneric SRI medications are usually paying somewhere in the neighborhood of $3600 per year for their prescriptions, at least a portion of which is covered by some if not most insurance companies. Along with the cost for six visits per year to a psychiatrist trained in the use of SRIs for OCD, which includes two initial visits and then four maintenance visits at $150 per visit, the cost of SRI pharmacotherapy is approximately $4500 per year, some of which would be lowered by decreasing the frequency of office visits and by having the general practitioner prescribe the medication.

In contrast, when CBT can be accessed privately, treatment costs for a licensed psychologist in the state of Pennsylvania average approximately $150 per hour. Managed care companies certainly suppresses these costs, but perhaps simultaneously suppresses the level of therapist expertise in OCD available to those enrolled in their plans. Indeed, many OCD experts are reluctant to sign up with managed care outfits that reduce payments to a fraction of their original charge, and thus therapists who are available via managed care may have insufficient expertise in treating OCD to produce outcomes similar to those produced by experts. Because the efficacy of CBT in the managed care context with therapists inexperienced in treating OCD has yet to be established, we will estimate CBT costs based on the going rate for 20 weekly, hour-long visits to a licensed psychologist with expertise in CBT for OCD, which yields $3000 per year. More intensive programs, which offer up to 40 contact hours over the course of 1 month plus some follow-up contacts (eg, Kozak & Foa, 1997), are at least twice as costly. Thus, using these figures as a starting point, CBT monotherapy is the least expensive option at approximately $3000 per year, followed closely by SRI monotherapy $4500 per year, with combined treatment being the most expensive at $7500 per year.

Availability

The second practical matter involves finding these respective treatments in community settings. As mentioned earlier, SRI monotherapy can be accessed through either general practitioners or psychiatrists, with the former easier to find yet the latter preferable in terms of expertise and knowledge of the empirical literature on SRIs for OCD. In contrast, CBT is difficult to find in community practice settings (eg, Freiheit, Vye, Swan, & Cady, 2004; Greist & Baer, 2002), and CBT expertise with pediatric OCD, harder still to find. These problems are likely due to a variety of factors, including the tendency for psychology training programs to de-emphasize the importance of outcome research when selecting modes of psychotherapy in which to train their students and the relatively low base rate of OCD, which limits the amount of experience that psychologists in general practice settings can accrue. Notably, the paucity of CBT expertise in the community affects access to CBT monotherapy and

combined treatment equally; thus, the most available empirically supported treatment for OCD is SRI monotherapy. Because access affects CBT monotherapy and combined treatment equally, this particular factor does not allow us to recommend one over the other.

THEORETICAL CONSIDERATIONS

As has been discussed in detail elsewhere (Foa, Franklin, & Moser, 2002), the logic supporting the possible advantages of combined treatment over monotherapy in OCD is relatively straightforward; and as others have remarked, almost common sense (Greist & Baer, 2002). First, response to CBT or SRI monotherapy is neither universal nor complete, which suggests that incremental improvements would still be possible by combining the two efficacious treatments together. Second, SRIs may address the secondary comorbidity that is so common in OCD, and thereby limit the potential negative impact of comorbidity on treatment outcome. Third, and perhaps most importantly, SRIs may make CBT easier by reducing anxiety to more manageable levels and thereby promoting processing of the corrective information available during exposure, which will result in greater symptom reduction. Importantly, there is an alternative possibility with respect to this latter point, which is that concomitant SRIs will attenuate CBT because they will reduce the anxiety/emotional engagement that is considered essential in exposure therapy (Foa & Kozak, 1985, 1986). Thus, there are theoretical arguments that can be made in support of the advantage of combined treatment over CBT monotherapy, but also arguments can be mounted to support the position that SRIs ought to attenuate the effects of CBT. We turn now to the treatment outcome literature to examine empirical support for these theoretical positions.

EMPIRICAL CONSIDERATIONS: WHAT DO THE OUTCOME DATA TELL US?

Despite the obvious importance of questions related to the efficacy of combined treatments, and the fact that SRI and CBT monotherapies for OCD have been in use since the 1960s, there are only a few methodologically sound randomized studies that have specifically examined the combined efficacy of SRIs and CBT. In a section on combined treatment from a recent textbook on anxiety disorders, Greist and Baer (2002) state that, "several studies . . . support the commonsense conclusion that a combination of these two independently effective treatment modalities is more effective than either treatment alone." However, the studies cited include several open trials of combined treatment with no direct comparison to either monotherapy (eg, March, Mulle, & Herbel, 1994; Neziroglu et al., 2000), a randomized trial in which significant differences were not found on the Y-BOCS (Goodman et al., 1989a, 1989b), which was the primary outcome measure (Hohagen et al., 1998), and one study in which patients were randomized to either CBT or clomipramine (CMI), with no combined treatment arm (de Haan, Hoogduin, Buitelaar, & Keijsers, 1998). Whereas these studies might indicate that combined treatments could be effective, they are not sufficient to support the claim that combined treatment is superior to either of the monotherapies.

In order to draw conclusions from the strongest available evidence, discussion of the empirical literature below will be limited to those studies that include the following elements: (*a*) patients had an established diagnosis of OCD; (*b*) the study included at least two treatment groups, one of which received pharmacotherapy or CBT monotherapy (CBT with or without pill placebo [PBO]) and the other, treatment combining CBT and medication; and (*c*) the study employed the following methodology: random assignment, sufficient statistical power, adequate treatment quality and dosage, and blind independent evaluation of OCD symptoms by a trained assessor. Six studies that met these criteria and are reviewed below.

Marks and Colleagues (1980)

The first study that directly compared EX/RP to medication, and also allowed for an examination of the efficacy of combined treatment, was conducted by Marks, Stern, Mawson, Cobb, and McDonald (1980). Using a complex experimental design, 40 patients were randomly assigned to receive either CMI or PBO for 4 weeks. Six weeks of inpatient psychological treatment (daily 45 min sessions) followed. During the first 3 weeks of this phase, 10 patients from each medication condition received EX/RP while the other 10 received relaxation. At week 7, those patients who had received relaxation were switched to EX/RP and the remaining continued to receive EX/RP; thus a direct comparison of the effects of CMI + EX/RP, EX/RP + PBO, and CMI + relaxation could only be made at week 7. At the end of the 6-week psychosocial treatment period, patients were discharged from the hospital but remained on medication until week 36, when a 4-week taper period commenced. Patients were followed for another year upon drug discontinuation. Results suggested that, compared to placebo, CMI produced significant improvements in mood and rituals only in those patients who were initially depressed. Compared to relaxation at week 7, EX/RP was associated with greater reductions in rituals, but not with improvements in mood. Combined treatment had a slight additive effect at week 7; the relative durability of gains could not be evaluated because of the subsequent shifts to other treatments after week 7.

This study meets several of the aforementioned criteria including random assignment to condition, use of blind raters, reasonable exclusion criteria, sufficient sample size ($n = 40$; 10 per experimental cell) to detect at least a moderate effect size, and statistical analyses were described clearly. Several methodological issues, most of which stem from the very complex experimental design, made interpretation of the findings difficult. Moreover, diagnostic methods were not described very well ("Patients were considered suitable ... if they had handicapping obsessional-compulsive rituals," p. 2), and the inpatient EX/RP condition, consisting of 45 min long daily sessions for 3–6 weeks (depending on treatment condition), may not have employed insufficiently strict response prevention instructions ("After exposure [in session], patients were asked not to carry out rituals for the rest of the session and to resist ritualizing for a specified time thereafter," p. 4). Length of the treatment session was neither specified, nor was sufficient information provided regarding what patients were instructed to do on the inpatient unit for 6 weeks when they were not in their OCD treatment sessions. Finally, in terms of evaluating the efficacy of CMI monotherapy, the drug-only period was too short (4 weeks) to allow optimal assessment of its efficacy.

Marks and Colleagues (1988)

In the next study of this issue, Marks et al. (1988) randomized 49 OCD patients to one of four treatment conditions, three of which included CMI for approximately 6 months and one PBO. One of the CMI groups received anti-exposure instructions for 23 weeks, the second group had self-controlled exposure for 23 weeks, and the third group received self-controlled exposure for 8 weeks followed by therapist-aided exposure from week 8 until week 23. The PBO group also received self-controlled exposure for 8 weeks followed by therapist-aided exposure from week 8 until week 23. Mean reduction after 8 weeks of self-exposure was 20% for rituals and 23% for OCD related discomfort; the mean reduction after an additional nine sessions of therapist-aided exposure was 71% and 68%, respectively. However, in the absence of a placebo group that received therapist-aided exposure first, an alternative explanation cannot be ruled out, namely that order effect mediated the superiority of therapist-aided exposure. Most importantly for our purposes here, CMI + EX/RP yielded superior outcome at week 8 compared to EX/RP + PBO on measures of rituals, depression, and social adjustment. At week 23, however, CMI + EX/RP did not separate from EX/RP + PBO. Thus, again, a small but transitory advantage was found for combined treatment over EX/RP monotherapy.

Marks et al. (1988) provided a clear description of inclusion/exclusion criteria, employed random assignment, and utilized psychometrically acceptable outcome measures. However, the design adopted by the investigators did not allow for an unambiguous test of the relative efficacy of CMI, EX/RP, and combined treatment; nor of the relative efficacy of self-exposure versus therapist-aided exposure. Several other problems complicate interpretation of findings: diagnostic methods were not described, treatment descriptions were sparse, and time spent on exposure homework was not reported. The latter point is especially important because of the authors' interest in the efficacy of self-exposure programs.

Cottraux and Colleagues (1990)

Burgeoning interest in more selective SRIs and increased awareness of CMI's unfavorable side effect profile and cardiotoxicity risks prompted additional studies using medications other than CMI for OCD. Cottraux et al. (1990) compared the efficacy of fluvoxamine (FLV), EX/RP, and combined treatment. Patients were assigned to one of three conditions: FLV with anti-exposure instructions, FLV + EX/RP, and PBO with EX/RP. In the anti-exposure condition, patients were specifically instructed to avoid feared situations or stimuli. Treatment continued for 24 weeks, after which EX/RP was stopped and medication was tapered over 4 weeks. Exposure and response prevention treatment was provided in weekly sessions and consisted of two distinct treatment phases: self-controlled exposure between sessions and imaginal exposure during sessions for the first 8 weeks, followed by 16 weeks of therapist-guided EX/RP. Other psychosocial interventions (eg, couples' therapy, cognitive restructuring, assertiveness training) were also provided "as deemed necessary."Assessment included ratings by blind evaluators and self-report measures.

At posttreatment (week 24), the percent reduction in assessor-rated duration of rituals per day were: FLV + anti-exposure = 42%, FLV + EX/RP = 46%, and PBO + EX/RP = 25%. At 6 month follow-up, reduction in assessor-rated duration

of rituals per day were: FLV + anti-exposure = 42%, FLV + EX/RP = 45%, and PBO + EX/RP = 35%. While FLV + EX/RP produced slightly greater improvement in depression at posttreatment compared to PBO + EX/RP, the superiority of the combined treatment for depression was not evident at follow-up. The means suggest an advantage for combined treatment at the week 24 assessment, but the differences on OCD measures failed to reach statistical significance; insufficient statistical power is invoked as a possible explanation for why the null hypothesis was not rejected. Interestingly, patients in the FLV + anti-exposure group complied only minimally with therapy instructions: most reported doing exposure on their own, thus invalidating the comparison between exposure and anti-exposure with FLV.

Cottraux et al. (1990) randomly assigned patients to treatment conditions, used blind assessors, conducted appropriate nonparametric statistical analyses because of skewed distributions, provided estimates of power to detect differences on OC symptom measures, and used adequate inclusion/exclusion criteria. They did not, however, describe diagnostic methods adequately or provide treatment adherence ratings. The primary problem with this study lies in the implementation of EX/RP. The description of this treatment was inadequate; it was portrayed only as "flexible," included the use of a myriad of other techniques (eg, couples therapy, cognitive restructuring, assertion training) on an "as needed" basis, and ritual prevention instructions were not provided. Moreover, treatment sessions were conducted just once per week for an unspecified length of time, which means that it is possible that the EX/RP "dose" may have been substantially less than is typically recommended (Foa & Franklin, 2001; Kozak & Foa, 1997) or probably efficacious (Abramowitz, 1996).

van Balkom and Colleagues (1998)

In the fourth study, van Balkom et al. (1998) randomized 117 OCD patients to receive: (*a*) cognitive therapy (CT); (*b*) EX/RP; (*c*) FLV + CT; (*d*) FLV + EX/RP; or (*e*) wait-list. Psychotherapy was conducted over sixteen 45-min sessions (6 in the first 8 weeks, and 10, on the remaining weeks). In the two combined treatments, FLV was administered alone for 8 weeks, after which medication was stabilized and 10 sessions of therapy were added for additional 8 weeks. The wait-list condition also lasted for 8 weeks. Assessment was conducted at pre-, mid-, and posttreatment, and the main outcome measure was the independent assessor's ratings of the Y-BOCS. Results indicated that at midtreatment ($n = 100$), all four active treatments were superior to wait-list, with no significant differences among active treatments. At posttreatment ($n = 86$), the active treatments did not differ from one another, suggesting that the addition of FLV to either EX/RP or CT did not enhance the efficacy of psychotherapy.

Random assignment to treatments, reliable and valid diagnostic procedures, clear inclusion and exclusion criteria, use of treatment manuals, adequate sample size, and sophisticated statistical analyses all constitute strengths of van Balkom et al. (1998) study. The authors also used state-of-the-art indices of reliable change to examine the clinical relevance of their data. However, several methodological shortcomings should be noted. No mention is made of assessor blindness or training. More importantly, the version of EX/RP employed in this study may have been substandard. First, session length was relatively brief (45-min) and sessions were held once per week instead of the more frequent schedule that is often recommended (Foa & Franklin, 2001; Kozak & Foa, 1997). Second, it is unclear how much homework the therapist assigned to

patients, and to what extent patients complied with homework instructions. Third, because the investigators were interested in an interim evaluation of the ‘purer’ versions of EX/RP and CT, discussion of negative consequences in the first six sessions of EX/RP was prohibited. However, these discussions are an important component of EX/RP as it is generally conducted (Franklin & Foa, 2002), and thus banning them, while justifiable because of the desire to pursue the theoretical question regarding the difference between EX/RP and CT, may have further diluted the treatment (for a discussion of the cognitive mechanisms of EX/RP see Foa & Kozak, 1986). Accordingly, the results of this truncated treatment were lower than what is typical (for a review see Foa & Kozak, 1996), with a mean OCD symptom reduction of only 32% after 16 weeks of EX/RP.

Hohagen and Colleagues (1998)

In the fifth study, conducted by Hohagen et al. (1998), 58 patients with OCD were randomly assigned to one of two treatment conditions: EX/RP + FLV or EX/RP + PBO. The authors found an advantage for EX/RP + FLV on the Y-BOCS obsession scale, but not on the compulsions subscale or on the Y-BOCS total score (a composite of the two subscales). In both conditions, EX/RP involved a 3-week assessment period followed by a 4 week regimen of lengthy (3 h minimum) thrice-weekly EX/RP. Analyses were conducted on a subset of patients ($n = 49$), with nine statistical outliers dropped from the analyses in order to equate the two groups on baseline OCD severity using the Y-BOCS. Results indicated that both groups improved significantly and comparably on compulsions, but that the patients who received FLV in combination with EX/RP were significantly better at posttreatment on obsessions than those who received EX/RP plus placebo. The percent reductions on the Y-BOCS total scores were 44% for EX/RP plus PBO and 55% for EX/RP plus FLV, which are well within the expected range for EX/RP; however, the observed difference on the Y-BOCS total failed to reach statistical significance. Consistent with clinical observations, subanalyses from this dataset indicated that patients who suffered from secondary depression also fared better if they were receiving active medication along with EX/RP.

Hohagen et al. (1998) employed random assignment, clearly described their inclusion and exclusion criteria, used reliable and valid assessment instruments administered by trained evaluators, manualized EX/RP treatment, and had a sufficient sample size to test their primary hypotheses. Faced with a statistical conundrum, however, the authors chose to exclude outliers in order to eliminate baseline differences in severity between the groups. They then conducted all subsequent statistical analyses on the remaining subset of patients. Results using statistical methods designed for this purpose (eg, ANCOVA) were not presented, nor did the authors indicate that they were conducted and equivalent to the results that were presented in the paper. Nevertheless, these findings provide empirical support for the use of concomitant pharmacotherapy, especially with those patients for whom obsessions predominate the clinical picture and for those with significant secondary depression.

Foa and Colleagues (2005)

Finally, a recently completed multicenter study conducted at the University of Pennsylvania and Columbia University aimed to provide an unequivocal comparison

of the efficacy of CMI, intensive EX/RP, and their combination (Foa et al., 2005). In this study, an EX/RP program that included an intensive phase (fifteen 2-h sessions conducted over 3 weeks) and follow-up phase (six brief sessions delivered over 8 weeks) was compared to CMI, EX/RP + CMI, and a pill PBO condition. Consistent with study hypotheses, results indicated that all active treatments were superior to PBO and that EX/RP was superior to CMI. With respect to the issue of combined treatment versus the monotherapies, results indicated that EX/RP + CMI was superior to CMI but did not differ from EX/RP alone (Foa et al., 2005). Percent reductions on the Y-BOCS for study completers were as follows: PBO = 11%, CMI = 32%, EX/RP = 55%, and EX/RP + CMI = 58%. However, the design adopted in this study (and in several others) may not have been optimal for promoting an additive effect because the EX/RP program was largely completed before the effects of CMI could be realized.

Diagnostic status was determined by structured clinical interview, patients were randomly assigned to treatments, and assessments were conducted by trained blind evaluators. Treatments were manualized, measures of treatment adherence were included, and OCD severity was assessed using reliable and valid measures (eg, Y-BOCS). The sample size for this study (122 entrants) was also substantially larger than for most of the other studies of combined treatment, and results clearly suggest that the failure to find a difference between EX/RP + CMI and EX/RP alone was not the result of inadequate power.

Summary of the Relevant RCTs

Several key points from the review above warrant emphasis. First, because authors of the few well-conducted studies addressing combination treatment have placed an emphasis on *extending* previous findings, as opposed to *replicating* previous work, there are many procedural, methodological, and treatment protocol variations that make it extremely difficult to draw broad conclusions with confidence. With respect to pharmacotherapy, for example, three of the studies used CMI (Foa et al., 2005; Marks et al., 1980, 1988), while the other three used FLV (Cottraux et al., 1990; Hohagen et al., 1998; van Balkom et al., 1998); meta-analytic findings reported by Greist, Jefferson, Kobak, Katzelnick, and Serlin (1995) suggest that CMI is superior to FLV. The duration of the pharmacotherapy regimens also varied across the studies, as did the dosages used within the CMI and FLV studies. The EX/RP procedures and visit schedules also varied widely from study to study. For example, van Balkom et al. (1998) and Cottraux et al. (1990) used a weekly regimen, Foa et al. (in press) used an intensive outpatient regimen, Hohagen et al. (1998) used thrice weekly, Marks et al. (1980) used an intensive inpatient program, and Marks et al. (1988) used self-controlled exposure. Variations in such EX/RP procedures have been found in meta-analytic studies to be associated with outcome (eg, Abramowitz, 1996), and thus weaken the evidence base from which broader conclusions must be drawn. Certainly, practical factors contribute to the tendency in clinical science to devise innovative studies at the expense of careful replication. For example, one could readily imagine a grant review panel's less than enthusiastic impression of an application's innovation if the proposed study were a simple replication of earlier work. Nevertheless, our conclusions are obfuscated in the absence of replication of at least some fundamental components of previous studies (eg, sampling frame, EX/RP protocols). Therefore, caution must be heeded lest we

collectively extract clearer and more definitive statements from the literature than can be supported by the relevant data.

Second, results from the available studies clearly do not support the theoretical argument that concomitant SRI treatment compromises EX/RP by preventing adequate emotional engagement during exposure. Of the four studies reviewed above that included unequivocal comparisons between combined treatment and EX/RP monotherapy (Cottraux et al., 1990; Foa et al., in press; Hohagen et al., 1998; van Balkom et al., 1998), none found EX/RP to be superior to combination treatment (nor were there any nonsignificant trends in that direction). Open studies of combined treatment also support the notion that at the very least, SRIs and CBT are compatible (eg, Franklin, Abramowitz, Bux, Zoellner, & Feeny, 2002; Franklin et al., 1998; March et al., 1994; Piacentini, Bergman, Jacobs, McCracken, & Kretchman, 2002; Simpson, Gorfinkle, & Liebowitz, 1999). Data on long-term follow-up in OCD also fail to suggest that combined treatment compromises maintenance of gains (eg, Simpson et al., in press), and thus OCD may differ from panic disorder in this regard. The divergence between OCD and panic disorder data with respect to an interference effect following treatment discontinuation for combined treatment may occur because OCD patients do not generally fear the very physical sensations that would be suppressed by medication, and thus medication does not specifically interfere with testing of disorder-related cognitions (Foa et al., 2002). Taken together, the data are quite clear in failing to support the hypothesis that combined treatment is less efficacious than monotherapy.

Third, with respect to the more widely held view that combined treatment is superior to EX/RP monotherapy, two studies failed to detect any additive effect (Cottraux et al., 1990; Foa et al., in press), two found a small but transitory advantage (Marks et al., 1980, 1988), one found a difference on measures of depression and a trend on measures of OCD favoring combined treatment at posttreatment, but these were no longer evident at follow-up (Cottraux et al., 1990), and one (Hohagen et al., 1998) found an advantage for combined treatment over EX/RP alone on Y-BOCS related obsessions but neither on compulsions nor on the composite total score. Of the two studies that allowed for a clear comparison of combined treatment versus SRI monotherapy, one found an advantage for combined treatment (Foa et al., in press) and one did not (Cottraux et al., 1990). Collectively, the data may be summarized as suggestive of an advantage for combined treatment, but the picture is by no means clear nor is the broad statement of its superiority supported. Notably, the two largest studies (Foa et al., in press; van Balkom et al., 1998) did not detect any effect for combined treatment over EX/RP alone, which further qualifies the claim that combined treatment is necessarily superior to both of the monotherapies, at least using the simultaneous start designs that have been used thus far.

CONCLUSIONS

Taken together, results from the most rigorously conducted studies comparing EX/RP monotherapy to combined treatment suggest that while EX/RP is not impeded by SRI pharmacotherapy, the addition of SRIs does not strongly enhance the effect of EX/RP monotherapy. Because the designs tested thus far have generally involved starting the SRI and EX/RP simultaneously, there are insufficient data to inform us of

whether the addition of EX/RP after SRI treatment has already been initiated would allow for detection of a combined treatment effect. Two reasonable next steps in examining the utility of combined treatment approaches would be: (*a*) to determine whether allowing more opportunity for a response to SRIs at an adequate dose and duration (by premedicating patients prior to EX/RP) will afford a better test of the relative efficacy of combined treatment over the monotherapies; and (*b*) to examine the utility of combined approaches in a different pool of OCD patients, namely those who have failed to respond adequately to either of the monotherapies. This latter question regarding the clinical management of SRI partial response is of paramount importance given the wide availability of SRIs and that residual symptoms following SRI monotherapy are the norm, and is discussed in detail elsewhere (Foa et al., 2002; Franklin, Foa, & March, 2003).

Although the data do not generally support unqualified preference of combined treatment over either of the monotherapies, there may very well be some patients who would fare better clinically if they received both treatments. For example, severe depression has been found to interfere with EX/RP (eg, Abramowitz, Franklin, Street, Kozak, & Foa, 2000), although the mechanism by which depression exerts this influence has yet to be clearly identified. For a patient who presents for EX/RP who has substantial depressive symptoms, a trial of SRI at an adequate dose and duration may reduce these depressive symptoms and thereby increase the likelihood of treatment response. The same case can be made for patients with other comorbid symptoms that are likely to respond to SRIs (if the broad effects of the medication alleviate the comorbid symptoms and produce the expected partial response on OCD symptoms, EX/RP may prove easier and more tolerable for the patient). Similarly, combined treatment may be useful when patient evidences highly fixed OCD beliefs. In such cases, an SRI trial may be a useful alternative, and even a medication combination strategy might be considered in this circumstance, since some data (eg, Foa, Abramowitz, Franklin, & Kozak, 1999) and general clinical impression suggest that EX/RP outcome is compromised with such patients, presumably mediated by homework noncompliance and inability to profit from exposure exercises.

Patient preference must also be considered when offering treatment to OCD patients in clinical settings. A subset of OCD patients refuse EX/RP because the treatment inherently involves direct confrontation with feared thoughts and situations and simultaneous elimination of rituals. For those who refuse EX/RP on these grounds, SRIs seem to be a reasonable choice, but combined treatment may also allay concerns that the treatment will be too overwhelming for the patient to handle. Conversely, patients who are reluctant to initiate pharmacotherapy may be more willing to try this treatment if it were delivered along with CBT. The rationale here is that since relapse rates following medication discontinuation appear to be lower for patients who receive combined treatment than for those who receive SRI pharmacotherapy alone (eg, Simpson et al., in press), this hope to those patients who fear being on medication indefinitely. Patient preference has not been studied yet in OCD, although a recent analogue investigation examining choice in PTSD treatments suggest that individuals may choose cognitive-behavioral treatments and pharmacotherapy for different reasons (Zoellner, Feeny, Cochran, & Pruitt, in press). It may be that at least some patients prefer combined treatment over monotherapy, and this preference needs to be taken into consideration in the clinical context. Given the inherent demands of EX/RP, it may also be that some patients would prefer a different psychotherapy

choice altogether, and the efficacy data on the newer forms CT that are tailored to the putative OCD-relevant beliefs offer hope that patient choice can be further expanded as this form of treatment also becomes more widely available.

The study of treatment choice, as well as improvement in the state of clinical care for most OCD patients, is hampered of course by the lack of availability of CBT monotherapy and, by extension, combined treatment. Significantly more attention and resources need to be allocated to studying ways to improve access to EX/RP. This might include studies of how best to disseminate CBT into the clinical settings where OCD patients are most likely to seek services at present, or developing regional centers of expertise to reduce the travel and expenses associated with receiving care in the few centers of expertise that currently provide this form of treatment. The outcome data do tell us that we have several treatments that work for this often disabling condition, and yet the options available for most patients will be limited until we begin to confront the market and clinical realities that prevent patients from accessing EX/RP and combined treatment. Some important work on the problem of access has already begun (eg, Marks et al., 1998), but much remains to be accomplished.

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Reply to Franklin:

USING COMBINATION TREATMENTS FOR OCD: A REPLY TO FRANKLIN

H. Blair Simpson and Michael R. Liebowitz

In reviewing the OCD treatment literature, we and Dr Franklin arrive at many of the same conclusions. First, we agree that ERP and pharmacotherapy with SRIs are both efficacious treatments for adults with OCD. Second, we concur that ERP monotherapy can produce better outcome than SRI monotherapy when ERP is delivered by skilled therapists and patients are motivated for treatment. Third, we agree that important limitations of the six relevant studies prevent definitive conclusions regarding whether combination treatment (SRI + ERP) is necessary for all treatment-seeking patients. Finally, we agree that only two of the six available studies provide strong evidence for the superiority of combination treatment over monotherapy. Specifically, Hohagen et al. (1998) conclude that SRI + ERP is superior to ERP + PBO when OCD patients have comorbid depression or when obsessions dominate the clinical picture. Foa et al. (in press) demonstrate that CMI + ERP is equivalent to EX/RP and that both are superior to CMI in OCD patients without comorbid depression.

Thus, Dr Franklin and ourselves both conclude that the available data do not support the use of combination treatment as a first-line intervention for all OCD patients. On the other hand, we both agree that there are particular clinical situations in which combination therapy should be considered over ERP monotherapy. Specifically, these include (*a*) when OCD is complicated by substantial depression or another comorbid condition that is responsive to SRIs; (*b*) when the patient refuses ERP because they doubt their ability to tolerate exposure to feared situations; and (*c*) when the patient evidences fixed obsessional beliefs, making them unlikely to adhere to ERP. Likewise, there are clinical situations where combination treatment should be considered over SRI monotherapy: when an OCD patient has a partial response to SRI monotherapy, when SRI exposure needs to be minimized because of troubling side effects, and/or when a patient wants to discontinue their SRI treatment (eg, to become pregnant) but fears relapse.

We and Dr Franklin also disagree on certain details. For example, Dr Franklin emphasizes that Hohagen et al. (1998) found no significant differences in total Y-BOCS scores between patients receiving FLV + ERP and patients receiving ERP + PBO treatment. However, there were significantly more responders (defined as those with a Y-BOCS reduction of at least 35%) in the FLV + ERP group (87.5%) than in

the ERP + PBO group (60%). Thus, we conclude that this study provides partial evidence for the superiority of combination treatment over ERP monotherapy even in the absence of the secondary analyses performed by the authors. The secondary analyses indicated that OCD patients with severe obsessions and those with comorbid depression would benefit most from the addition of FLV to ERP.

Second, Dr Franklin dismisses the findings of Marks, Stern, Mawson, Cobb, and McDonald (1980), Marks et al. (1988), and of Cottraux et al. (1990) that suggest a small early advantage of combination treatment over ERP monotherapy, even though no advantage for combination treatment was observed with longer follow-up. However, since the follow-up data were confounded by shifts in subsequent treatment, I discount the follow-up data and conclude that these studies also provide some (albeit weak) evidence that combination treatment is superior to ERP monotherapy.

Third, Dr Franklin argues that the two largest studies (Foa et al., in press; van Balkom et al., 1998) did not detect significant effects for combination treatment over ERP alone. However, these studies appear to have found similar results for different reasons. In the study by Foa and colleagues, ERP was intensive (fifteen 2-h sessions over 3 weeks) and delivered by skilled therapists. Patients completing ERP monotherapy evidenced a mean 54% reduction in Y-BOCS scores, whereas those completing CMI + ERP had a 56% reduction. Most patients in the CMI + ERP group (unlike those in the CMI monotherapy group) did not achieve the maximal permitted dose of CMI. van Balkom and colleagues also found that the effects of ERP monotherapy were not significantly different from FLV + ERP. However, in that study, ERP consisted of 16 weekly exposure sessions (each 45 min long) and led to only a 32% reduction in Y-BOCS scores. FLV + ERP treatment produced a 49% Y-BOCS reduction despite the fact that the FLV dose was less than optimal (ie, average dose less than 200 mg per day) and the ERP condition consisted of only 10 sessions. Together, these data suggest that ERP monotherapy is only equivalent to combination treatment when ERP is delivered by skilled therapists in an intensive format, and/or when combination treatment is delivered less than optimally. In routine clinical practice, where therapy is usually delivered weekly and ERP experts are rare, ERP monotherapy may not fare so well, and combination treatment might be required to produce a good outcome. Of course, if ERP is delivered poorly, combination treatment may show no significant benefit over SRI monotherapy. In summary, to decide whether combination treatment is preferable to monotherapy for an individual patient, one must consider not only data from these randomized controlled trials, but also clinical characteristics of the patient (eg, motivation for ERP, severe depression), the skill of the treating clinician (eg, are they experienced in providing ERP?), and the clinical setting in which treatment is provided (eg, can ERP be delivered more than once per week?).

In summary, SRI and ERP are both effective treatments for OCD. In short-term outcome and in long-term maintenance of gains, ERP has the potential to be more effective than SRIs in OCD patients without comorbid depression. On the other hand, SRI treatment is currently more widely available, and many OCD patients have comorbid depression. Thus, SRI monotherapy is often used as the first line treatment for practical reasons. On the other hand, partial response to SRI monotherapy is common in OCD (Pigott & Seay, 1999). Thus, when SRI monotherapy is initiated first, augmentation is usually needed. The only pharmacological strategy currently proven in randomized controlled trials to augment SRI pharmacotherapy in OCD is the addition of antipsychotic agents (eg, McDougle, Epperson, Pelton, Wasylink, & Price,

2000). However, antipsychotics carry with them several significant risks (eg, tardive dyskinesia and neuroleptic malignant syndrome) and thus are not benign agents. Preliminary data indicate that ERP augmentation of SRI pharmacotherapy is at least as effective as augmentation with antipsychotic medications, and a much safer alternative (Simpson, Gorfinkle, & Liebowitz, 1999).

Therefore, we concur strongly with Dr Franklin that significantly more attention and resources should be devoted to improving patient access to ERP, since ERP can be used to augment the benefit of SRI medication, reduce medication for those who suffer side effects, or treat those who do not want (or cannot tolerate) medication. The combination of ERP and SRI pharmacotherapy may not be required as a first-line treatment for all OCD patients, but it should be available to those with a partial response to monotherapy since it is likely to be more successful and safer than many other pharmacological augmentation strategies currently used in clinical practice for OCD.

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Reply to Simpson and Liebowitz:

MEETING IN THE MIDDLE, THEN MOVING FORWARD TOGETHER

Martin E. Franklin

In keeping with the high quality of their previous work (eg, Liebowitz et al., 1992; Simpson, Gorfinkle, & Liebowitz, 1999), Simpson and Liebowitz' chapter in this volume is comprehensive, authoritative, and rooted firmly in the empirical literature. The similarities between the conclusions drawn in the two chapters about the relative efficacy of combined treatment versus the monotherapies were notable, as were the collective conveyance of the sense that the empirical literature on this topic is far less developed than it ought to be given the importance of the question. Moreover, their chapter went beyond my own in spelling out a number of useful avenues to pursue with respect to determining how best to deliver combined treatment and to whom. Specifically, a major emphasis was placed in their chapter on the possible utility of targeting combined treatment to certain patient populations (eg, severely depressed) and for those with specific clinical difficulties (eg, noncompliance with exposure and response prevention [EX/RP]) in order to improve outcome. The purpose of this response is to identify common ground, places where the chapters' emphases differed and, perhaps most importantly, to outline ways that interdisciplinary research efforts can further advance the science informing the clinical management of OCD patients. I will pursue these goals using subheadings provided in my colleagues' chapter, discussing each of the issues in the order in which they were raised.

SUMMARY OF THE RANDOMIZED CONTROLLED TRIALS

We agreed that the literature examining the relative efficacy of combined treatment versus either of the monotherapies was sparse, with only six randomized studies meeting both of our respective methodological requirements. Moreover, design issues and procedural variation among the studies limit conclusions that can be drawn from them. We also agreed that there was no evidence suggesting that combined treatment impeded monotherapy, although Simpson and Liebowitz did go on to discuss clinical circumstances in which monotherapy might be considered advantageous for certain patients.

One point that was raised, but not highlighted in either chapter, was that all six of these studies directly compared combined treatment to EX/RP alone, whereas only two (Cottraux et al., 1990; Foa et al., in press) allowed for a direct comparison of combined treatment to an adequate trial of anti-obsessional medication. Further, one of the latter two studies (Cottraux et al., 1990) involved anti-exposure instructions within the pharmacotherapy condition; yet patients reported that they were doing exposure on their own, which means that the comparison of FLV to FLV + EX/RP may have been compromised. Thus, only one study allowed for an *unambiguous* comparison; and that study found a significant difference between combined treatment and CMI alone (Foa et al., in press). Further, that study's design involved a simultaneous start of EX/RP and CMI, which could have led to an underestimation of the possible benefit of adding EX/RP after patients have reached therapeutic doses and have had sufficient time to achieve medication benefits. In short, even after 25 years of research on the topic, we really have very little to go on regarding the direct comparison of combined treatment and SRI monotherapy, and our conclusions must be appropriately cautious.

With respect to the comparison of combined treatment and EX/RP, the two chapters were largely in agreement with one another in interpreting all but one of the six studies they reviewed in detail, the study conducted by Hohagen et al. (1988). Simpson and Liebowitz note that Hohagen et al. (1998) found significantly more responders (defined as ≥35% improvement in Y-BOCS total score) in the FLV + ERP group (87.5%) than in the ERP + PBO group (60%), then go on to report that Hohagen et al. (1998) secondary analyses of the continuous Y-BOCS data revealed that, "both groups improved significantly and comparably on compulsions, but the FLV + ERP groups significantly more improved on obsessions; and (b) patients with comorbid depression fared better if they received FLV + ERP." Importantly, the analyses of the continuous Y-BOCS data in Hohagen et al. (1998) indicated that the treatments did *not* differ significantly on Y-BOCS total or on the Y-BOCS compulsion subscale, although the two did separate on obsessions. Studies that use the Y-BOCS typically present analyses of continuous Y-BOCS total score as the primary measure of outcome(eg, Geller et al., 2001), but the emphasis in the Hohagen paper is placed more on the obsession subscale and on the categorical analyses where the significant differences was found. The trends for most of these analyses consistently favor combined treatment over EX/RP + PBO, but nevertheless the failure to detect a significant difference using inferential statistics on the primary measure of OCD symptom severity available in the field is meaningful, and ought not to be minimized.

We also differed on Hohagen et al. (1998) to the extent that we accepted their argument in the abstract (p. 71) and in the body of the paper that the findings support the statement that, "behavior therapy should be combined with FLV when obsessions dominate the clinical picture." This phrase is open to multiple interpretations, however. It could mean that combined treatment is preferred when a patient has many more obsessions than compulsions, but this does not fit the sample description well (eg, "all patients showing both obsessions and compulsions," p. 71), nor is there any analysis of a subgroup of such patients who appeared to respond best to EX/RP + FLV. Perhaps instead the authors were referring to patients suffering from very severe obsessions, regardless of the severity of their compulsions. Here again there are no analyses in the paper where patients high and low on obsessions were compared to one another within the two conditions, so the statement does not seem pertain to this subgroup either. Instead, the data do support the more cautious statement that

when EX/RP + FLV is provided one might expect a greater advantage (compared to EX/RP + PBO) at posttreatment in the reduction of obsessions but no significant advantage on compulsions or overall. Moreover, the absence of any follow-up data also do not allow us to determine whether this effect on obsessions was transitory, which is what several other studies have found at follow-up for combined treatment when a posttreatment advantage is found (Marks, Stern, Mawson, Cobb, & McDonald, 1980; Marks et al., 1988).

THE TREATMENT OF COMORBIDITY THAT MIGHT INTERFERE WITH EX/RP

We both raised the possibility that concomitant pharmacotherapy and EX/RP might afford advantages for those patients with OCD who were comorbid with conditions known to respond to the same medications, such as other anxiety disorders and depression. Hohagen et al. (1998) data on high- and low-depressed patients are consistent with this position. Future studies specifically recruiting patients with these comorbidities and then comparing premedication with SRIs prior to EX/RP to appropriate control conditions are urgently needed. I suggested in my chapter that one possible mechanism by which comorbidity might attenuate outcome is by reducing compliance with EX/RP procedures which, as Simpson and Liebowitz discuss, is supported by data from Marks et al. (1980). Simpson and Liebowitz extend this argument, suggesting that OCD patients with difficulty adhering to EX/RP might benefit from SRI regardless of their comorbidity status. This issue has not been examined carefully as yet, although the failure to find a general advantage in efficacy for combined treatment compared to EX/RP monotherapy suggests that targeting patients at risk for noncompliance (eg, those with high overvalued ideation) for randomization to combined treatment or EX/RP alone might prove more fruitful than examining the issue in a general sample of individuals with OCD who are seeking treatment.

TREATMENT OF PARTIAL RESPONDERS TO MONOTHERAPY

Partial response to SRI is apparently the norm, and SRIs are clearly the most widely available of the empirically supported treatments for OCD. Accordingly, there is a growing population of patients who have experienced a partial response to SRIs, and, as has been argued elsewhere, an OCD patient on a third SSRI trial is likely to have as much as a threefold lower chance of responding than a treatment-naïve patient (March & Ollendick, 2004), so the development of combined treatment strategies involving EX/RP is imperative. Simpson and Liebowitz note that examination of the efficacy of such approaches is underway, although the dissemination problem at the back end of such studies is not discussed extensively in their chapter. Just as we have strong evidence that combined treatment and EX/RP alone are both efficacious initial treatments for OCD, even if we find that EX/RP is indeed more helpful than other augmentation strategies for SRI partial responders, the problem of disseminating EX/RP into the clinical settings of relevance (outpatient psychotherapy and pharmacotherapy clinics) has to be addressed if the treatment is to have a broader

impact. As it currently stands, a host of marketing and training issues render EX/RP a "boutique" treatment, available only to those who happen to live near the academic centers that specialize in its use or to those with the financial means to circumvent the many barriers encountered locally by the vast majority of individuals with OCD who seek treatment. Here, there are a number of promising opportunities for psychologists and psychiatrist to collaborate and, perhaps as suggested by comparison of findings from Cottraux et al. (1990) and Foa et al. (in press), the advantages of combined treatment may actually become more apparent in this context, since disseminated EX/RP programs are likely to be somewhat curtailed by market forces that at the very least will limit session frequency and expertise of the treatment provider.

THE TREATMENT OF SEVERE OCD

Simpson and Liebowitz noted that although published treatment guidelines for OCD (March, Frances, Carpenter, & Kahn, 1997) suggest an advantage for combined treatment when OCD is "more severe," severity has not consistently been found to be a predictor of outcome (Steketee & Shapiro, 1995). Why then the apparent inconsistency between expert consensus and empirical findings? Perhaps the identifiable source is the nature of the question asked of the experts who were surveyed: Kozak (1999) points out that panelists were asked to compare the relative efficacy of medications and *weekly* CBT for this more severe group, which might have resulted in a down-rating of CBT because more intensive regimens (eg, daily EX/RP) were not one of the choices given. Another point worth mentioning is that "more severe OCD" is defined quite liberally in the Expert Consensus Guidelines as a Y-BOCS score of at least 18, which is the usual minimum cutoff for RCTs that use the Y-BOCS as the primary measure of OCD symptom severity. Data on the efficacy of EX/RP monotherapy collected thus far do not support the statement that combined treatment is necessarily superior in the long run for such patients, and the studies that need to be done to address this issue, examination of treatment efficacy in a sample of OCD patients with truly severe symptoms (eg, Y-BOCS ≥ 30), has not been attempted. It is highly likely that comorbidity will confound any such study, and thus severity and clinical complexity are likely to have to be addressed together, with statistical methods used to parse out the impact of severity and comorbidity as best as possible.

TO MINIMIZE SRI EXPOSURE

Simpson and Liebowitz raise the intriguing possibility that physicians may be able to minimize SRI exposure if EX/RP were added to the treatment regimen, which may in turn reduce side effects and, as they also mention, increase the likelihood of successful medication discontinuation down the road. Here, it may make the most sense to test models of disseminating EX/RP directly to physicians in the context of medication management, where it appears that most patients with OCD who seek treatment are likely to wind up. Again, collaboration between psychologists and psychiatrists will be required to test and then disseminate combined treatment protocols that can be used by physicians in the clinical contexts in which they currently treat OCD patients. Work of this kind has already been attempted in adult social phobia

(eg, Blomhoff et al., 2001), and may serve as a reasonable model for such endeavors in OCD.

FINAL COMMENTS

Collectively our chapters suggest that some progress has been made in examining EX/RP, SRIs, and EX/RP + SRIs as initial treatments for OCD, and that the data hint at but do not overwhelmingly support a global combined treatment effect, at least from the studies conducted thus far. Much more needs to be done in this area, including the examination of different combined treatment models (eg, premedication prior to EX/RP), targeting of specific patient populations who may be at risk for nonresponse to monotherapy, and empirical evaluation of strategies to improve clinical management of SRI partial response. Perhaps most ambitiously and importantly, though, work is needed in the development and empirical evaluation of dissemination models that will improve patient access to EX/RP and to combined treatment, both of which have been found efficacious for OCD and hold great promise for the many OCD patients who at present cannot avail themselves of these potentially helpful alternatives.

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