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Research Article

WHERE DOES OBSESSIVE–COMPULSIVE DISORDER BELONG IN DSM-V?

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A reclassification of obsessive–compulsive disorder (OCD) into a new diagnostic category spectrum of "obsessive–compulsive spectrum disorders" (OCSDs) has recently been proposed, with considerable debate, for the forthcoming Diagnostic and Statistical Manual—Fifth Edition (DSM-V). This paper provides a critical analysis of the available empirical data regarding this conceptual and nosological shift. Specifically, we review research on shared commonalities and differences between OCD and the putative OCSDs in relation to their clinical presentation, phenotype, neurobiology, and treatment response. We conclude that a reclassification of OCD into a separate OCSD spectrum is premature and not supported by the currently available data. Depression and Anxiety 25:336–347, 2008. *© 2008 Wiley-Liss, Inc.*

Key words: ***obsessive–compulsive disorder; obsessive–compulsive spectrum; impulse control disorders; Diagnostic and Statistical Manual for Mental Disorders; Tourette's syndrome***

INTRODUCTION

A reclassification of obsessive–compulsive disorder (OCD) into a larger spectrum of disorders (termed as obsessive–compulsive spectrum disorders [OCSD]) has recently been proposed, with considerable debate [e.g., Mataix-Cols et al., 2007], for the forthcoming *Diagnostic and Statistical Manual—Fifth Edition* [DSM-V; Hollander, 2007; Hollander and Evers, 2004]. The OCSD model asserts that applicable disorders are located on a compulsivity–impulsivity dimension with the compulsive anchor characterized by harm avoidance and anxiety reduction, and the impulsive anchor characterized by pleasure-seeking and gratification behaviors [Hollander and Zohar, 2004]. The following disorders would be incorporated as OCSDs: OCD, body dysmorphic disorder (BDD), hypochondriasis, chronic tic disorders (e.g., Tourette's syndrome), numerous impulse control disorders (ICDs; e.g., trichotillomania, pathological gambling, compulsive shopping, pyromania), eating disorders, addictions, and autism [Hollander and Zohar, 2004]. Although these disorders were initially included in the spectrum on the basis of overlaps in overt symptom presentation [e.g., repetitive thinking and behavior; Hollander, 1993], OCSD proponents currently assert that the model is fundamentally etiological in that it defines OCD and related disorders based on endophenotypes and purported commonalities in etiologically relevant factors such as heritability, brain circuitry, neurotransmitter abnormalities, and phenotypic similarities with other disorders [Hollander et al., 2007].

The proposed OCSD model and diagnostic shift is widely contested by clinicians and researchers alike on both conceptual and empirical grounds. Illustrating this, Mataix-Cols et al. [2007] found that 40% of 187 mental health professionals who specialize in OCD treatment and research disagreed with moving

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OCD out of the anxiety disorders category, whereas the vast majority of those who agreed with creating a separate OCSD category believed that it should be narrow in scope, including OCD, BDD, trichotillomania, and possibility tic disorders and hypochondriasis. The inclusion of other disorders such as ICDs was not supported by the majority of respondents. Consensus opinion aside, the primary arguments put forth for considering shifting OCD from the anxiety disorders into a new OCSD classification are as follows [Bartz and Hollander, 2006; Hollander and Zohar, 2004; Stein and Lochner, 2006]:

(a) The symptoms of OCD and OCSDs share a core feature, namely repetitive thoughts and behaviors.
(b) Phenotypic similarities between OCD and OCSDs including age of onset, comorbidity, and family loading.
(c) OCD and the proposed OCSD share brain circuitry abnormalities, familial/genetic factors, and neurotransmitter/peptide abnormalities.
(d) OCD and the proposed OCSDs share similar treatment response profiles, specifically to pharmacological interventions.

Over the past several years, proponents of the OCSD approach have disseminated their arguments in a variety of academic and clinical publications [e.g., Hollander and Evers, 2004; Hollander et al., 2007; Lochner and Stein, 2006; Stein and Lochner, 2006]. These articles appear to provide evidence in support of each of the four arguments listed above. When each of these arguments is scrutinized, however, flaws in the underlying logic of the OCSD approach come to light. Given the recent promotion of the OCSD approach in the literature, without regard to opposing perspectives [for an exception, see the volume edited by Abramowitz and Houts, 2005], the purpose of this article is to present a critical review of the relevant data and thereby add another voice to the discussion and debate over the positioning of OCD in DSM-V. The scientific legitimacy of the OCSD approach can only be demonstrated when the model is exposed to scrutiny and its proponents have successfully defended its empirical foundations. We hope this discussion will spawn additional research to empirically address this debate.

DO OCD AND THE PROPOSED OCSDS SHARE REPETITIVE THOUGHTS AND BEHAVIORS AS A CORE SYMPTOM DOMAIN?

According to its proponents, the OCSDs are linked on a "core repetitive behavior domain" that is characterized by the inability to delay or inhibit repetitive behaviors. This assertion is modeled on the assumption that uncontrolled behaviors fall along a continuum from risk aversion (compulsions) to risk-taking (impulsivity). Stated differently, at one end of the continuum are located compulsive disorders such as OCD, hypochondriasis, BDD, and anorexia nervosa, which are characterized by repetitive behaviors that reduce or avoid risk and harm. ICDs (e.g., sexual compulsions, addictions, etc.), in contrast, represent the other anchor as these conditions are characterized by pleasure-seeking, often risky behaviors. To this end, the unifying factor among the proposed OCSDs is the presence of repetitive behaviors. Yet, the proposal that the presence of repetitive behaviors link disorders along a spectrum encounters significant conceptual difficulties that are discussed next.

PHENOMENOLOGY OF OCD AND ITS DISCONNECT WITH THE OCSD

By emphasizing the presence of repetitive behaviors as criteria for the spectrum, the OCSD approach overlooks other essential (and arguably more fundamental) features of the phenotypic presentation of OCD. Perhaps some "blame" for focusing on repetitive behaviors in the OCSD approach can be attributed to the current DSM's atheoretical approach to taxonomy wherein psychological disorders are defined merely by lists of signs and symptoms rather than theoretically derived models that incorporate empirically validated psychological constructs (e.g., motivation and cognition). This is especially the case in OCD where, due to its emphasis on symptom form as opposed to symptom function, the DSM-IV/DSM-IV-TR [American Psychiatric Association, 1994, 2000] definition and diagnostic criteria overlook important empirically established aspects of the phenomenology of obsessions and compulsions. Most critical to the current discussion is that, according to the DSM, either obsessions *or* compulsions are necessary and sufficient for a diagnosis of OCD. The difficulty here is that this leaves the impression that obsessions and compulsions are independent repetitive phenomena. Although this conceptualization of OCD is unsupported by the literature (as discussed below), it could lead to drawing parallels between OCD symptoms and other disorders that involve repetitious behavior. Examples include "compulsive" gambling, "compulsive" stealing, "compulsive" use of pornography, "obsessive" jealousy, and even excessive nail biting, which clinicians frequently subsume under the OCD umbrella.

As mentioned above, this superficial conceptual approach to OCD is 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% 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 [e.g., Leckman et al., 1997], as well as on measures of symptom severity [Amir et al., 1997; Deacon and Abramowitz, 2005]. 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.

Rather, OCD is characterized by an internally consistent link between obsessions and compulsions. Initially, individuals experience involuntary thoughts and doubts (obsessions) that they find unacceptable and anxiety evoking. Often, but not always, the obsessions are triggered by environmental stimuli. To deal with the obsessional distress, patients resort to purposeful behavioral or mental activity that, at least temporarily, reduces distress. OCD is therefore best conceptualized as a problem in which an individual is fearful of certain situations and stimuli, which are the focus of the obsessions. Compulsive behavior is performed with the purpose of alleviating obsessional distress, a phenomenon that unifies OCD with other anxiety disorders (as discussed further below). We next turn to empirical research supporting this approach to understanding the symptoms of OCD.

A series of simple yet elegant laboratory studies has closely examined the relationship between obsessions and compulsions 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, whereas 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 and 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 and Rachman, 1976; Roper et al., 1973] and in a study of patients with mental compulsions evoked by intrusive unacceptable thoughts [de Silva et al., 2003].

The above 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 after compulsive behavior, and the eventual reduction of anxiety and compulsive urges (even if the ritual is not performed) are the hallmarks of OCD, but not necessarily other disorders in the proposed OCSD (e.g., compulsive gambling, paraphilias, etc.).

An often overlooked group of symptoms in OCD are mental rituals and other "mini rituals" often referred to collectively as "neutralizing behaviors". Although such behaviors do not necessarily meet DSM diagnostic criteria for compulsions, they possess identical *functional* properties as compulsive rituals such as washing and checking [Rachman and Shafran, 1998]. That is, although not necessarily stereotyped or repetitive, they are intended to control obsessional thoughts, reduce the perceived probability of feared consequences, and reduce anxiety [e.g., van den Hout et al., 2001]. Thus, these "covert" rituals are conceptually the same as the "classic" repetitive, overt compulsive rituals. Neutralizing strategies include brief reassurance-seeking efforts, overanalyzing and rational self-talk (i.e., 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., 2000]. 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 and Ladouceur, 1997; Ladouceur et al., 2000]. The theoretical importance of mental rituals and covert neutralizing behaviors (i.e., mini rituals) is that they indicate that repetitive and compulsive behavior, per se, is not the key defining feature of OCD. Rather, repetition is simply one of the several means by which patients with OCD neutralize or "deal with" obsessional distress. "Compulsivity" is simply a way of describing this behavior.

PROBLEMS WITH THE OCSD APPROACH

PHENOMENOLOGICAL DIFFERENCES BETWEEN OCD AND OCSDS

Are compulsivity and impulsivity opposite ends of a spectrum?. Although the idea has some intuitive appeal (i.e., classifying multiple disorders based on the commonality of repetitive behaviors), what exactly is meant by "compulsivity," "impulsivity," and "spectrum" has not been well defined in this context. Like the other anxiety disorders, OCD typically involves the use of maladaptive strategies (e.g., avoidance, compulsive rituals) for coping with perceived threat or discomfort (i.e., obsessional thoughts). Compulsive behavior in OCD is therefore best conceptualized as an escape behavior (i.e., from obsessional distress) that is similar in nature to avoidance or safety behaviors in other anxiety disorders. Impulsive behavior, such as

that which characterize ICDs (i.e., pathological gambling or kleptomania), is pleasure-seeking rather than motivated by distress reduction. The patient feels a rush of excitement when the behavior is performed that is intrinsically reinforcing [Grant and Potenza, 2004]. Yet, where are the data to suggest that these two classes of behavior exist on a spectrum? In fact, it is unclear exactly what kind of data would demonstrate whether such a spectrum is present or not, and it may be that impulsivity and compulsivity are best viewed as dimensions that can co-occur in any given person regardless of whether they have a diagnosis of OCD. In support of this, the extant evidence suggests the lack of any specific relationship between impulsivity and compulsivity for a number of reasons. First, ICDs occur at fairly low rates among patients with OCD [Bienvenu et al., 2000]. Second, patients with OCD do not necessarily evidence greater levels of impulsivity than do individuals with other sorts of psychiatric disorders [for an exception in a comparison with panic disorder and social phobia, see Richter et al., 2003; Summerfeldt et al., 2004]. Third, very different treatment approaches are successful with these two sorts of clinical behavior [e.g., Abramowitz and Houts, 2002], whereas the treatment of OCD and other anxiety disorders, particularly through psychotherapeutic techniques, is remarkably similar.

Where are cognitions?. Another phenomenological difference between OCD and the OCSDs includes the role of cognition in clinical presentation and symptom function. In general, the OCSD model ignores the contribution of cognitions that underlie the motivation to perform the repetitive behaviors. Cognitive-behavioral models of OCD symptoms highlight the role of dysfunctional beliefs and interpretations of situations and normally occurring intrusive thoughts in explaining the persistence of obsessions and compulsions [Rachman, 1997; Salkovskis, 1985, 1989]. When a person appraises an otherwise normally occurring mental intrusion (e.g., a thought about harming one's child) as highly meaningful (e.g., "This thought means I am a terrible, dangerous parent"), distress ensues together with attempts to remove the intrusion and prevent the feared consequences vis-à-vis rituals or avoidance. Paradoxically, the frequency of intrusions and the associated anxiety intensity is increased and they escalate into persistent and distressing clinical obsessions (see Fig. 1). Compulsions maintain the intrusions through anxiety reduction and also prevent the self-correction of mistaken cognitive appraisals, which sets into motion a vicious cycle of intrusion → misinterpretation → anxiety → rituals → intrusion.

Empirical evidence for this theoretical approach is considerable [e.g., Abramowitz et al., 2001; Barrett and Healy, 2003; OCCWG, 2003; Rachman, 1998] and should not be overlooked in any attempt to classify (or reclassify) OCD. Moreover, treatment based on this model—cognitive-behavioral therapy (CBT)—is the most effective individual treatment available for OCD

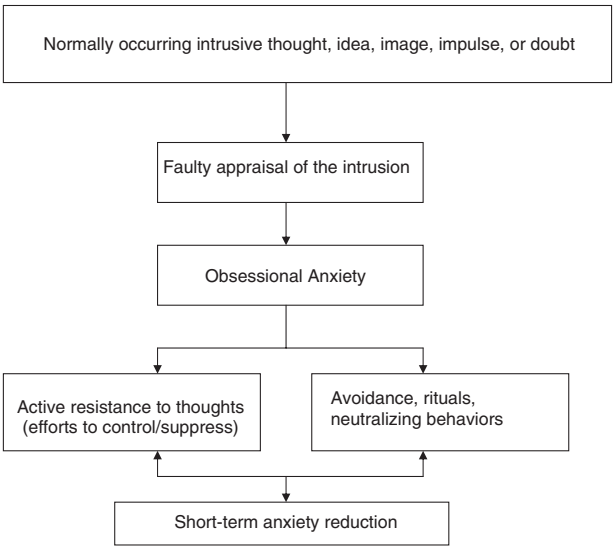

Figure 1. Cognitive-behavioral model of obsessive–compulsive disorder.

[e.g., Foa et al., 2005; Nakatani et al., 2005]. Not only does this further suggest the validity of the theory, but if one argues that more effective prevention and treatment ought to be the goal of any new formulation of OCD and potentially related disorders, it follows that cognition, which provides a basis for CBT, should be at the forefront of any such formulation.

Overall, the cognitive-behavioral model of OCD provides a clearly articulated, comprehensive, logically sound, and empirically verifiable theory for the nature of obsessions, compulsions, and related features—but is neglected from the OCSD approach. The cognitive-behavioral model implicates normal human learning principles (i.e., conditioning) and normal cognitive processes in accounting for the nature and etiology of OCD with greater parsimony than do models that appeal to the presence of chemical imbalances, disease states, or general deficits in attempting to explain the nature and etiology of OCD. The proposed OCSD theory fails to satisfactorily account for the role of cognition in OCD despite its considerable role.

Is the presence of repetitive behavior specific to the OCSDs?. It is unclear how the presence of repetitive behaviors has any real sensitivity and specificity to the OCSDs given that repetitive behaviors are characteristic of multiple psychiatric disorders. For example, there are no fundamental differences in repetitive behaviors that reduce or avoid risk and harm for proposed OCSD disorders (e.g., OCD, hypochondriasis, BDD) and anxiety disorders excluded from the OCSD spectrum (e.g., social phobia, generalized anxiety disorder, and panic disorder). In addition, although theoretical underpinnings are provided that purportedly link disorders along the proposed spectrum, operational definitions of what constitutes a repetitive behavior are lacking specificity

and sensitivity, and establishing such definitions has numerous pragmatic issues. For example, how can one determine the topography of behavior across multiple illness presentations, yet equate them? A similar question exists with the issue of symptom frequency, given that some disorders are characterized by the very frequent occurrence of repetitive behavior (e.g., OCD, trichotillomania), whereas the primary symptoms of others may occur less frequently (e.g., certain sexual paraphilias). Finally, the notion that the OCSDs can be viewed as occurring along a compulsivity–impulsivity continuum [e.g., Bartz and Hollander, 2006] is also not unique to the proposed OCSDs. Aside from the debatable nature of this continuum (reviewed above), non-OCSDs may also be characterized by these constructs. For example, few would argue that people who are manic have a problem delaying or inhibiting certain behaviors, whereas people with generalized social phobia, in contrast, are characterized by a high degree of inhibition [Degnan and Fox, 2007]. Given that the OCSD concept is based on large part on the presence of repetitive behaviors/symptoms, the issue that the cardinal phenotypic characteristic of OCSDs is neither sensitive nor specific to OCSDs represents a significant problem.

BRAIN CIRCUITRY AND NEUROTRANSMITTER/PEPTIDE SYSTEMS

Proponents of the OCSD model concede that existing data on the brain circuitry of OCD and OCSDs remain somewhat equivocal [Bartz and Hollander, 2004]. Nevertheless, differences or commonalities in brain circuitry are hypothesized between OCD and other anxiety disorders, and OCD and ICDs. For example, some findings implicate frontal–striatal circuitry in the pathophysiology of OCD, whereas some appear to suggest that the amygdala is central in the pathophysiology of many anxiety disorders [Saxena and Rauch, 2000]. Proponents of the OCSD model imply that understanding such differences can help shift the DSM from diagnoses that are based on the identification of symptom clusters toward a manual that also incorporates information about brain structure and functionality.

Although this proposition may have merit for future editions of the DSM [Hyman, 2007], it is at best premature for the present revision (i.e., DSM-V). A primary difficulty with neuroimaging findings is that results across studies have not been highly consistent. In a recent meta-analysis of neuroimaging studies, for example, Whiteside et al. [2004] found that differences between OCD patients and controls were not replicated across studies for the orbitofrontal cortex, caudate nucleus, frontal cortex, parietal, left temporal, right temporal, anterior cingulate, or thalamus. This review included only 13 methodologically rigorous studies indicating the preliminary nature of this literature. Although recent authors have suggested that it is too early to base diagnostic reclassifications on neurobiology findings [e.g., Hyman, 2007; Mataix-Cols et al., 2007], it is reasonable to use these data to develop well-controlled studies that can more conclusively answer the question of shared commonalities/differences among disorders. However, in the absence of such studies, it is "premature to bring neurobiology into the formal classification of mental disorders that will form the core of the DSM-V" [Hyman, 2007, p 731].

In addition, the *specificity* of brain structures and functions to OCD has not been studied. That is, there are few (if any) controlled studies in which people with OCD and those with other anxiety disorders have been directly compared on neuropsychiatric (imaging) variables. This problem of lack of evidence for specificity is perhaps the most limiting factor in our ability to infer that OCD is characterized by differences in neurocircuitry from other anxiety disorders. Similarly, very little data exist comparing patients with OCD relative to those with proposed OCSDs. In the absence of such data, it is premature to make a decision to restructure the DSM-V based on biological variables.

COMORBIDITY

Proponents of the OCSD approach also appeal to comorbidity in linking OCD to the putative OCSDs, and argue that the high comorbidity among OCD and the OCSDs supports a new category of OCSDs in DSM-V. Results of several large-scale comorbidity studies, however, largely stand in contrast to this proposition. For example, in the Johns Hopkins OCD family study, Bienvenu et al. [2000] reported the following rates of OCSDs among 80 individuals with OCD: hypochondriasis = 16%, BDD = 15%, anorexia nervosa = 9%, bulimia = 4%, trichotillomania = 4%, kleptomania = 3%, pathologic gambling = 0%, and pyromania = 0%. In a study of 98 adults with OCD, Richter et al. [2003] found comorbidity rates of 13% for a tic disorder, 9% for trichotillomania, 5% for an eating disorder, and 15% for clinical skin picking. Similar results have been found by others [Barsky et al., 1992; Savron et al., 1996].

The findings presented above indicate that OCSDs are quite *uncommon* among patients with OCD [with the exception of BDD and hypochondriasis, which are considered by many researchers to be anxiety-motivated disorders; Abramowitz, 2005; Abramowitz and Braddock, 2006; Noyes, 1999; Phillips et al., 1995]. This is perhaps dramatically illustrated by noting that among OCD patients in the Bienvenu et al. [2000] study, 96% *did not* have trichotillomania, whereas in the Richter et al. [2003] study, 87% did not have a tic disorder. However, even if it is granted that OCSD comorbidity rates are higher than chance in OCD, and even if comorbidity is a useful basis for lumping disorders in DSM-V (an issue we consider further below), the bigger problem for this argument is that comorbidity rates between OCD and other anxiety and

mood disorders are considerably higher than the rates for putative OCSDs. For example, data from the John 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 OCSD proponents [e.g., Hollander et al., 2005] use to support their contention that OCSDs are 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 two- to three-fold closer!).

Is comorbidity a useful indicator of taxometric or etiologic relationships between disorders? It is our position that this is not a sound basis for grouping disorders into DSM spectra (OCD or otherwise). Indeed, comorbidity is common in most major psychiatric disorders, and there are numerous explanations for this phenomenon. For instance, alcohol dependence, depression, and posttraumatic stress disorder (PTSD) are all more highly comorbid than what would be expected by chance. Although it is easy to recognize several potential reasons for the co-occurrence of these disorders, few would suggest that alcohol dependence, depression, and PTSD are part of the same spectrum. Not surprisingly, Summerfeldt et al. [2004] found that although OCD was associated with elevated levels of impulsivity compared to nonclinical controls, this relationship was not unique to OCD as all of the anxiety disorders showed increased impulsivity relative to the control group. All of these data suggest that comorbidity is of limited value in understanding the uniqueness of OCD and its links to OCSDs.

FAMILY HISTORY

A similar argument applies to OCSD proponents' claim that OCD has a familial or genetic association with OCSDs. Not only does this claim remain to be demonstrated for many of the proposed OCSDs, but the available data show that 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 [e.g., Bienvenu et al., 2000; Nestadt et al., 2001]. For example, Nestadt et al. [2001] reported the following rates of anxiety disorders in first-degree relatives of adults with OCD: 16.3% OCD, 15.6% generalized anxiety disorder, 9.0% panic disorder, 22.9% social phobia, 25.0% specific phobia, and 12.6% separation anxiety disorder. In the same data set, however, lifetime prevalence rates of ICDs in first-degree relatives of adults with OCD were 1% trichotillomania, 0% kleptomania, 9% pathologic gambling, 0% pyromania, 4% "any" eating disorder, and 17% any "grooming" disorder [Bienvenu et al., 2000]. Therefore, the assertion that familial pattern is a good evidence for a relationship between OCD and other disorders again ends up supporting the view that OCD is more strongly related to other anxiety disorders than to the 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.

TREATMENT RESPONSE

One aspect of the OCSD position is that OCD has a similar response to pharmacological treatment as do the various OCSDs. This line of reasoning has been primarily focused on pharmacological interventions, unfortunately minimizing the role of effective psychological treatments [for reviews, see Abramowitz et al., 2005; Storch and Merlo, 2006]. Nonetheless, a number of rigorous randomized controlled studies have demonstrated the effectiveness of serotonin reuptake inhibitors (SRIs), which include clomipramine and selective serotonin reuptake inhibitors (SSRIs), in adults and children with OCD [e.g., DeVeaugh-Geiss et al., 1992; Foa et al., 2005; Geller et al., 2003; Goodman et al., 1989; Greist et al., 1995; Tollefson et al., 1994]. Response rates have generally been good, with about 50–60% of patients showing a positive outcome.

In contrast to data in OCD patients, SRI response in OCSDs is more modest and varied in terms of efficacy and the amount of available data. For example, several studies have shown no beneficial effect of SSRIs in adults with trichotillomania relative to placebo [Christenson et al., 1991; Ninan et al., 2000; Streichenwein and Thronby, 1995; Van Minnen et al., 2003], whereas others have found efficacy [Dougherty et al., 2006]. In a methodologically rigorous multimodal trial, Van Minnen et al. [2003] found that both behavioral psychotherapy and a waitlist control were superior to fluoxetine. Data, however, are seriously limited regarding other disorders that would be included in the proposed OCSD (e.g., paraphilias, sexual addictions). A small number of uncontrolled trials have shown a moderate response to SRI therapy in patients with nonparaphilic sexual addictions [Kafka, 1994; Stein et al., 1992]. However, as noted by Mataix-Cols et al. [2007], there are insufficient data at present to support any linkage among putative OCSDs as a function of treatment response.

Treatment response ought to be the litmus test for any OCSD proposals as it is ultimately the successful treatment that is sought by dealing with matters of phenomenology and etiology. Here again, the OCSD proposal encounters significant problems and may even lead people with OCD *away from* the best treatment for their condition. OCSD proponents [e.g., Hollander et al., 2005; Stein and Lochner, 2006] 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 proposed OCSDs. This means that the assertion of preferential treatment response in most OCSDs is based on open-trial study results that are not adequate for answering the question of relative efficacy of medication. It is troubling that such broad speculations about treatment response are made given the lack of convincing data and implications for clinical management. Evidence even suggests that non-SRIs are helpful for many of the proposed OCSDs such as kleptomania [McElroy et al., 1989], compulsive shopping [McElroy et al., 1991], and pathological gambling [Moskowitz, 1980] to name a few. Also, neuroleptic medications (e.g., haldol) that are ineffective as monotherapies for OCD are often used in the treatment of Tourette's syndrome [Leckman et al., 1991].

Second, in addition to OCD, the SRIs are efficacious in the treatment of depressive disorders [e.g., Nemeroff and Schatzberg, 1999] and other anxiety disorders including panic disorder [e.g., Boyer, 1995] that are excluded from the OCSD theory. Again, the spectrum argument runs into the lack of specificity problem: because SRIs help so many disorders, the observation that a group of problems respond preferentially to these drugs does not tell us anything special about these disorders. Just because *preliminary* data support their use in OCSDs does not mean this is evidence of a common linkage. If that were true, a vast number of psychiatric disorders (e.g., depression, bipolar disorder, personality disorders, non-OCD anxiety disorders) not considered within the OCSD theory would be implicated for their linkage. Further, many patients with ICDs who benefit from SSRIs have comorbid anxiety and/or depressive disorders, which may explain treatment-associated benefits [Grossman et al., 1999].

Third, ignored within the OCSD conceptualization is the highly effective psychological treatment for OCD, namely CBT. It is now well acknowledged that CBT involving the procedures of exposure and response prevention (ERP) is the most effective treatment for OCD [Abramowitz et al., 2005; Foa et al., 2005; Jenike, 2004]. 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 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. This similarity of theoretically derived and specific effective treatment techniques/programs for OCD and other anxiety disorders provides evidence that OCD is closely related to those anxiety disorders. For example, CBT for OCD is quite similar to that used for other anxiety disorders (e.g., social phobia, PTSD) in that structured treatment protocols include: (1) exposure (placing the patient in situations that elicit anxiety related to their fears); (2) response prevention (deterring the ritualistic or avoidance behaviors that may serve to reduce or avoid anxiety); and (3) teaching objective thinking strategies (e.g., training the patient to identify and correct anxiety-provoking cognitions).

When one considers the outcome of exposure-based CBT, a pattern emerges that links OCD with the other anxiety disorders. Outcomes for CBT in varied anxiety disorders across the age range are quite consistent. For example, between 54 and 84% of adults with social phobia are considered responders to CBT [Clark et al., 2003, 2006; Taylor, 1996], a figure comparable to OCD outcome data [e.g., Abramowitz et al., 2005; Foa et al., 2005; Storch et al., 2007]. Notably, this is in stark contrast to the outcomes of CBT protocols for ICDs. Currently, very few randomized trials have been conducted that examine CBT for OCSDs. Although some positive results have been found [e.g., Mitchell et al., 2006], many studies report on small samples and are vulnerable to the bias that positive results are more likely to be published than negative results. Other studies are limited to subjects who comply with treatment, which is a significant factor when considering the pleasure-seeking nature of ICDs (e.g., only the very motivated seek treatment). Given the large dropout rates of many treatments, this practice exerts a sampling bias in favor of positive results.

Because CBT is based on the specific relationship between obsessional fear and compulsive behavior, this treatment is irrelevant for most of the putative OCSDs. For example, because trichotillomania involves neither obsessional fears nor urges to perform compulsive rituals designed to escape or neutralize anxiety, there would be no logic in using exposure or response prevention in the treatment of this disorder. Hair pulling in trichotillomania is not evoked by obsessional anxiety, but rather by general tension, fatigue, or boredom. Hair pulling in trichotillomania is also not performed to reduce the probability of danger, as is observed with compulsions in OCD.

As can be seen, trichotillomania 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 [Himle et al., 2006]. 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 (e.g., 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, pathological sexual behavior, and substance abuse disorders.

IS OCD AN ANXIETY DISORDER?

One aim of diagnostic classification is to organize clinical problems into groups sharing common characteristics, including treatment response. Although most experts agree that OCD is correctly classified as an anxiety disorder, a few authors have raised this issue as a matter of debate by asserting that OCD is *more* closely linked to OCSDs due to the presence of repetitive behavior as the primary symptom, as well as similarities in clinical presentation, neurobiology, and treatment response. Authors who envision OCD as distinct from other anxiety disorders also point to the presence of strange, intrusive, and repetitious obsessions and compulsions that although present in OCD are not apparent in other anxiety disorders. Further, the DSM-IV/DSM-IV-TR includes a specifier for "poor insight" in OCD, but not for the other anxiety conditions. Below, it is asserted that OCD *is* an anxiety disorder on the basis of shared fundamental phenomenology and similar responses to specific treatments for anxiety and fear.

Although an examination of the current DSM criteria might lead one to see topographical differences between OCD and other anxiety disorders, the DSM criteria are merely meant to be descriptive; it is an atheoretical document [American Psychiatric Association, 2000]. In contrast, empirical research on the psychological experiences of those with OCD indicates clearly that there are strong similarities with other anxiety states. An in-depth review of this research is beyond the scope of this article [indeed, entire books have been written on the subject; e.g., Barlow, 2002]; yet, the main conclusions of this work are summarized in Table 1. As can be seen in the table, OCD, specific and social phobia, panic, PTSD, and generalized anxiety disorder all involve fear that occurs in the context of more or less 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 given in Table 1).

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 feared situations. In a broad sense, when danger is perceived, taking action to avoid or escape from the potential threat is normal and adaptive (i.e., the "fight or flight" instinct). Indeed, this kind of avoidance and "safety-seeking" behavior is observed across the anxiety disorders [e.g., Clark, 1999]. Examples include resting to prevent heart attacks during panic episodes, the agoraphobic's use of a "safety person" to avoid losing control, over-rehearsal of a speech by someone with social anxiety who fears that any mistake would result in public humiliation, and (of course) phobic avoidance of disorder-specific feared stimuli.

Likewise, in OCD the same type of phobic avoidance of situations that trigger obsessional fear is apparent (e.g., public bathrooms, knives, "unlucky" numbers,

TABLE 1. Empirically demonstrated phenomenological aspects of OCD and other anxiety disorders

Diagnosis	Fear-evoking stimuli	Underlying beliefs	Safety behaviors
OCD	Intrusive thoughts and related triggers	Thoughts are equivalent to actions, inflated responsibility for preventing harm [e.g., OCCWG, 1997]	Avoidance, checking, hoarding, washing, ordering/arranging, covert neutralizing
Specific phobia	Snakes, heights, injections, storms, etc.	Overestimation of the likelihood or severity of danger from the feared stimulus [Beck and Emery, 1985]	Avoidance, drinking alcohol before flying, distraction during injections
Social phobia	Social situations, embarrassment, etc.	Other people are highly judgmental, negative evaluation is intolerable [Clark and Wells, 1995]	Avoidance of social situations, speaking softly, using alcohol
Panic disorder and agoraphobia	Arousal-related body sensations	Heart palpitations signify a heart attack, dizziness leads to fainting [Clark, 1986]	Agoraphobic avoidance, 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 and Rothbaum, 1998]	Avoidance of triggers, distraction, relying on others for safety, carrying a flashlight
GAD	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, obsessive–compulsive disorder; PTSD, posttraumatic stress disorder; GAD, generalized anxiety disorder.

cemeteries, etc.). Yet because many obsessional fears are ubiquitous or unavoidable (e.g., using the bathroom, intrusive violent thoughts), the patient resorts to compulsive rituals to escape from anxiety—e.g., washing behavior because of feared contact with "urine germs," repeated checking motivated by thoughts of having hit a pedestrian with one's car." These rituals, however, are essentially the same as the behavior of a person with social phobia who finds herself in a social situation and so consumes alcohol to reduce distress; or a person with panic who goes to the emergency room because he feels a panic attack beginning.

Although the excessive avoidance and escape behaviors in OCD and in the other anxiety disorders might appear topographically diverse, each is phenomenologically linked to a feared stimulus and a belief (i.e., overestimation of threat) that are characteristic of each disorder. To the patient, the safety response functions to reduce the perceived probability of feared consequences. Put another way, there is an internal logic to the types of compulsive rituals performed by people with OCD; and this is the same "logic" that is observed across the anxiety disorders (but not in many of the putative OCSD conditions such as ICDs or Tourette's disorder). The far right column in Table 1 displays examples of the so-called "safety behaviors" observed in the various anxiety disorders. Although it may be tempting to view compulsive rituals as unique from the safety behaviors in other anxiety disorders, any differences in repetitiveness, strangeness, or degree of functional impairment they engender are merely topographical differences that are less important than the fundamental psychological purpose of these behaviors, which are the same. All of this is to point out that the fundamental phenomenology of OCD is threat detection-based, and the same as that in the other anxiety disorders.

Although the above conceptualization applies to the majority of patients with OCD, it is balanced to note that recent research has shown that symptoms of OCD may be motivated by factors other than fear. Several examples are relevant to this end and may represent distinct disorder subtypes. First, some people exhibit contamination symptoms that are primarily driven by disgust rather than feared outcomes [Stein et al., 2001]. Second, some patients with OCD experience feelings of incompleteness or "just right" feelings rather than a specific fear [e.g., Leckman et al., 1995]. Finally, a considerable literature has debated the inclusion of hoarding as an OCD subtype [e.g., Grisham et al., 2005], given the often ego-syntonic nature of hoarding symptoms among other characteristics that distinguish it from more "classic" OCD symptoms [Lochner et al., 2005].

Taken together, it may be that on the whole, OCD does not appear to fit well with either the anxiety disorders or OCSDs. This perception, however, is likely a consequence of the extreme heterogeneity of OCD as it is currently conceptualized. Some OCD symptoms (e.g., contamination fears, fears of unwanted impulses) share many features with anxiety disorders such as phobias and panic disorder. On the other hand, OCD symptoms involving symmetry, ordering, and hoarding can sometimes share features with other sorts of disorders proposed as OCSDs (e.g., ICDs). Perhaps a resolution to the present debate over where OCD belongs in future editions of the DSM would be to parse OCD into more homogeneous categories, some of which overlap with other anxiety disorders, and some of which are more similar to ICDs, tic disorders, and neurological conditions with repetitive behaviors. We hope researchers will take up this issue of trying to better "carve OCD at its joints" rather than continuing to study and treat the disorders as if it were a homogeneous entity.

CONCLUSION

This article provides a critical review of the research related to proposals that OCD be removed from the anxiety disorders and into a new OCSD category in the DSM-V. As noted, there is inadequate empirical support for such a marked conceptual and nosological shift. Furthermore, incorrect conclusions have been drawn from existing data and cited to support the OCSD shift. Pragmatically, the OCSD model has limited heuristic and practical value when compared to a phenotypic or functional model of diagnostic classification. For example, diagnosing an OCSD on the basis of common etiological factors is problematic given the limited number of and dissemination of reliable and valid laboratory tests [Hyman, 2007]. Is it feasible, or even possible, for the practicing clinician to conduct tests of genetics, brain circuitry, and neurotransmitter functioning to arrive at a diagnosis, particularly when one considers cost and current insurance reimbursement practices? Stated differently, if a patient's symptoms appear phenotypically and functionally equivalent to prototypical OCD, does it matter what caused them when it comes to assigning a diagnosis? In addition, is it not likely that a particular disorder will be multidetermined rather than due to a single etiological factor? Given this, it only seems reasonable that an etiologically based system of diagnostic classification must be accompanied by sensitive and specific measures of etiology for it to have any real clinical utility—unfortunately, these measures either do not exist, or are not available to the practicing clinician who sees the majority of patients.

Evidently, many expert clinicians and researchers recognize issues such as these [e.g., Hyman, 2007; Mataix-Cols et al., 2007], which explains why the vast majority of those surveyed in the study by Mataix-Cols et al. [2007] disagreed with a diagnostic shift for DSM-V that places OCD with ICDs. We suggest here that it is simply premature to make the proposed OCSD shift given the absence of supporting empirical data, a lack of consensus among experts in the field, and further conceptual issues that must be integrated. It is hoped

that this paper will open up a more balanced and objective discussion of the potential merits and limitations of the OCSD conceptualization.

REFERENCES

Abramowitz JS. 2005. Hypochondriasis: conceptualization, treatment, and relationship to obsessive–compulsive disorder. Ann Clin Psychiatry 17:211–217.

Abramowitz JS, Braddock AE. 2006. Hypochondriasis: conceptualization, treatment, and relationship to obsessive–compulsive disorder. Psychiatr Clin North Am 29:503–519.

Abramowitz JS, Houts AC. 2002. What is OCD and what is not? problems with the OCD spectrum concept. The Scientific Review of Mental Health Practice 1:139–156.

Abramowitz JS, Houts AC. 2005. Concepts and controversies in obsessive–compulsive disorder. New York, NY, USA: Springer Science. 437 p.

Abramowitz JS, Tolin DF, Street GP. 2001. Paradoxical effects of thought suppression: a meta-analysis of controlled studies. Clin Psychol Rev 21:683–703.

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

American Psychiatric Association. 1994. Diagnostic and statistical manual of mental disorders (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. 2000. Diagnostic and statistical manual of mental disorders (4th ed., text revision). Washington, DC: American Psychiatric Association.

Amir N, Foa E, Coles M. 1997. Factor structure of the Yale–Brown Obsessive Compulsive Scale. Psychol Assess 9:312–316.

Barlow DH. 2002. Anxiety and its disorders: the nature and treatment of anxiety and panic (2nd ed.). New York, NY, USA: Guilford Press.

Barrett P, Healy L. 2003. An examination of the cognitive processes involved in childhood obsessive–compulsive disorder. Behav Res Ther 41:285–299.

Barsky AJ, Wyshak G, Klerman GL. 1992. Psychiatric comorbidity in DSM-III-R hypochondriasis. Arch Gen Psychiatry 49:101–108.

Bartz JA, Hollander F. 2006. Is obsessive–compulsive disorder an anxiety disorder? Prog Neuropsychopharmacol Biol Psychiatry 30:338–352.

Beck AT, Emery G. 1985. Anxiety disorders and phobias: a cognitive perspective. New York: Basic Books.

Barsky AJ, Wyshak G, Klerman GL. 1992. Psychiatric comorbidity in DSM-III-R hypochondriasis. Arch Gen Psychiatry 49:101–108.

Bartz JA, Hollander E. 2006. Is obsessive–compulsive disorder an anxiety disorder? Prog Neuropsychopharmacol Biol Psychiatry 30:338–352.

Bienvenu OJ, Samuels JF, Riddle MA, Hoehn-Saric R, Liang KY, Cullen BA, Grados MA, Nestadt G. 2000. The relationship of obsessive–compulsive disorder to possible spectrum disorders: results from a family study. Biol Psychiatry 48:287–293.

Boyer W. 1995. Serotonin uptake inhibitors are superior to imipramine and alprazolam in alleviating panic attacks: a meta-analysis. Int Clin Psychopharmacol 10:45–49.

Christenson GA, Mackenzie TB, Mitchell JE, Callies AL. 1991. A placebo controlled, double-blind crossover study of fluoxetine in trichotillomania. Am J Psychiatry 148:1566–1571.

Clark DM. 1986. A cognitive approach to panic. Behav Res Ther 24:461–470.

Clark DM. 1999. Anxiety disorders: why they persist and how to treat them. Behav Res Ther 37:S5–S27.

Clark DM, Wells A. 1995. A cognitive model of social phobia. In: Heimberg R, Liebowitz M, Hope DA, Schneier FR, editors. Social phobia: diagnosis, assessment and treatment New York: Guilford Press. p 69–93.

Clark DM, Ehlers A, Hackmann A, McManus F, Fennell M, Grey N, Waddington L, Wild J. 2006. Cognitive therapy versus exposure and applied relaxation in social phobia: a randomized controlled trial. J Consult Clin Psychol 74:568–578.

Clark DM, Ehlers A, McManus F, Hackmann A, Fennell M, Campbell H, Flower T, Davenport C, Louis B. 2003. Cognitive therapy versus fluoxetine in generalized social phobia: a randomized placebo-controlled trial. J Consult Clin Psychol 71:1058–1067.

Deacon BJ, Abramowitz JS. 2005. The Yale-Brown obsessive compulsive scale: factor analysis, construct validity, and suggestions for refinement. J Anxiety Disord 19:573–585.

Deacon B, Abramowitz J. 2006. Anxiety sensitivity and its dimensions across the anxiety disorders. J Anxiety Disord 20:837–857.

Degnan KA, Fox NA. 2007. Behavioral inhibition and anxiety disorders: multiple levels of a resilience process. Dev Psychopathol 19:729–746.

de Silva P, Menzies RG, Shafran R. 2003. Spontaneous decay of compulsive urges: the case of covert compulsions. Behav Res Ther 41:129–137.

DeVeaugh-Geiss J, Moroz G, Biederman J, Cantwell D, Fontaine R, Greist JH, Rcichler R, Katz R, Landau P. 1992. Clomipramine hydrochloride in childhood and adolescent obsessive–compulsive disorder: a multicenter trial. J Am Acad Child Adolesc Psychiatry 31:45–49.

Dougherty DD, Loh R, Jenike MA, Keuthen NJ. 2006. Single modality versus dual modality treatment for trichotillomania: sertraline, behavioral therapy, or both? J Clin Psychiatry 67:1086–1092.

Foa EB, Kozak MJ. 1995. DSM-IV field trial: obsessive–compulsive disorder. Am J Psychiatry 152:90–96.

Foa EB, Rothbaum BO. 1998. Treating the trauma of rape: Cognitive-behavioral therapy for PTSD. New York, NY, USA: Guilford Press.

Foa EB, Liebowitz MR, Kozak MJ, Davies S, Campeas R, Franklin ME, Huppert JD, Kjernisted K, Rowan V, Schmidt AB, Simpson HB, Tu X. 2005. Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive compulsive disorder. Am J Psychiatry 162:151–161.

Freeston MH, Ladouceur R. 1997. What do patients do with their obsessive thoughts? Behav Res Ther 35:335–348.

Geller DA, Biederman J, Stewart SE, Mullin B, Martin A, Spencer T, Faraone SV. 2003. Which SSRI? A meta-analysis of pharmacotherapy trials in pediatric obsessive–compulsive disorder. Am J Psychiatry 160:1919–1928.

Goodman WK, Price LH, Rasmussen SA, Delgado PL, Heninger GR, Charney DS. 1989. Efficacy of fluvoxamine in obsessive–compulsive disorder. A double-blind comparison with placebo. Arch Gen Psychiatry 46:36–44.

Grant JE, Potenza MN. 2004. Impulse control disorders: clinical characteristics and pharmacological management. Ann Clin Psychiatry 16:27–34.

Greist J, Chouinard G, DuBoff E, Halaris A, Kim SW, Koran L, Liebowitz M, Lydiard RB, Rasmussen S, White K. 1995. Double-blind parallel comparison of three dosages of sertraline and placebo in outpatients with obsessive–compulsive disorder. Arch Gen Psychiatry 52:289–295.

Grisham JR, Brown TA, Liverant GI, Campbell-Sills L. 2005. The distinctiveness of compulsive hoarding from obsessive–compulsive disorder. J Anxiety Disord 19:767–779.

Grossman LS, Martis B, Fichtner CG. 1999. Are sex offenders treatable? A research overview. Psychiatr Serv 50:349–361.

Himle MB, Woods DW, Piacentini JC, Walkup JT. 2006. Brief review of habit reversal training for Tourette syndrome. J Child Neurol 21:719–725.

Hodgson R, Rachman S. 1972. The effects of contamination and washing in obsessional patients. Behav Res Ther 10:111–117.

Hollander E. 1993. Obsessive–compulsive spectrum disorders: an overview. Psychiatr Ann 23:355–358.

Hollander E, Evers M. 2004. Review of obsessive–compulsive spectrum disorders: what do we know? Where are we going? Clin Neuropsychiatry 1:32–51.

Hollander E, Friedberg JP, Wasserman S. 2005. The case for the OCD spectrum. In: Abramowitz JS, Houts AC, editors. Concepts and controversies in obsessive–compulsive disorder. New York, NY, USA: Springer Science. p 95–118.

Hollander E, Kim S, Khanna S, Pallanti S. 2007. Obsessive–compulsive disorder and obsessive–compulsive spectrum disorders: diagnostic and dimensional issues. CNS Spectr 12:5–13.

Hollander E, Zohar J. 2004. Beyond refractory obsessions and anxiety states: toward remission. J Clin Psychiatry 14:3–5.

Hyman SE. 2007. Can neuroscience be integrated into the DSM-V? Nat Rev Neurosci 8:725–732.

Jenike M. 2004. Clinical practice. Obsessive-compulsive disorder. N Engl J Med 350:259–265.

Kafka MP. 1994. Sertraline pharmacotherapy for paraphilias and paraphilia-related disorders: an open trial. Ann Clin Psychiatry 6:189–195.

Ladouceur R, Freeston MH, Rheaume J, Dugas MJ, Gagnon F, Thibodeau N, Fournier S. 2000. Strategies used with intrusive thoughts: a comparison of OCD patients with anxious and community controls. J Abnorm Psychol 109:179–187.

Leckman JF, Grice DE, Boardman J, Zhang II, Vitale A, Bondi C, Alsobrook J, Peterson BS, Cohen DJ, Rasmussen AS, Goodman WK, McDougle CJ, Pauls DL. 1997. Symptoms of obsessive–compulsive disorder. Am J Psychiatry 154:911–917.

Leckman JF, Grice DE, Barr LC, de Vries ALC, Martin C, Cohen DJ, McDougle CJ, Goodman WK, Rasmussen SA. 1995. Tic-related vs. non-tic-related obsessive compulsive disorder. Anxiety 1:208–215.

Leckman JF, Hardin MT, Riddle MA, Stevenson J, Ort SI, Cohen DJ. 1991. Clonidine treatment of Gilles de la Tourette's syndrome. Arch Gen Psychiatry 48:324–328.

Lochner C, Kinnear CJ, Hemmings SM, Seller C, Niehaus DJ, Knowles JA, Daniels W, Moolman-Smook JC, Seedat S, Stein DJ. 2005. Hoarding in obsessive–compulsive disorder: clinical and genetic correlates. J Clin Psychiatry 66:1155–1160.

Lochner C, Stein DJ. 2006. Does work on obsessive–compulsive spectrum disorders contribute to understanding the heterogeneity of obsessive–compulsive disorder? Prog Neuropsychopharmacol Biol Psychiatry 30:353–361.

Mataix-Cols D, Pertusa A, Leckman JF. 2007. Issues for DSM-V: how should obsessive–compulsive and related disorders be classified? Am J Psychiatry 164:1313–1314.

McElroy SL, Hudson JI, Pope HG, Keck PE. 1991. Kleptomania: clinical characteristics and associated psychopathology. Psychol Med 21:93–108.

McElroy SL, Keck Jr PE, Pope Jr HG, Hudson JI. 1989. Pharmacological treatment of kleptomania and bulimia nervosa. J Clin Psychopharmacol 9:358–360.

Mitchell JE, Burgard M, Faber R, Crosby RD, de Zwaan M. 2006. Cognitive behavioral therapy for compulsive buying disorder. Behav Res Ther 44:1859–1865.

Moskowitz JA. 1980. Lithium and lady luck; use of lithium carbonate in compulsive gambling. N Y State J Med 80:785–788.

Nakatani E, Nakagawa A, Nakao T, Yoshizato C, Nabeyama M, Kudo A, Isomura K, Kato N, Yoshioka K, Kawamoto M. 2005. A randomized controlled trial of Japanese patients with obsessive–compulsive disorder effectiveness of behavior therapy and fluvoxamine. Psychother Psychosom 74:269–276.

Nemeroff CB, Schatzberg AF. 1999. Recognition and treatment of psychiatric disorders: a psychopharmacology handbook for primary care. American Psychiatric Association: Washington, DC.

Nestadt G, Samuels J, Riddle MA, Liang KY, Bienvenu OJ, Hoehn-Saric R, Grados M, Cullen B. 2001. The relationship between obsessive–compulsive disorder and anxiety and affective disorders: results from the Johns Hopkins OCD Family Study. Psychol Med 31:481–487.

Ninan PT, Rothbaum BO, Marsteller FA, Knight BT, Eccard MB. 2000. A placebo-controlled trial of cognitive-behavioral therapy and clomipramine in trichotillomania. J Clin Psychiatry 61:47–50.

Noyes Jr R. 1999. The relationship of hypochondriasis to anxiety disorders. Gen Hosp Psychiatry 21:8–17.

Obsessive–Compulsive Cognitions Working Group. 2003. Psychometric validation of the Obsessive Beliefs Questionnaire and the Interpretation of Intrusions Inventory: part I. Behav Res Ther 41:863–878.

QCCWG. 1997. Cognitive assessment of obsessive–compulsive disorder. Obsessive Compulsive Cognitions Working Group. Behav Res Ther 35:667–681.

Phillips KA, Kim JM, Hudson JI. 1995. Body image disturbance in body dysmorphic disorder and eating disorders. Psychiatr Clin North Am 18:317–334.

Rachman S. 1997. A cognitive theory of obsessions. Behav Res Ther 35:793–802.

Rachman S. 1998. A cognitive theory of obsessions: elaborations. Behav Res Ther 36:385–401.

Rachman S, Shafran R. 1998. Cognitive and behavioral features of obsessive–compulsive disorder. In: Swinson RP, Antony MM, Rachman S, Richter MA, editors. Obsessive–compulsive disorder: theory, research, and treatment. New York, NY, USA: Guilford Press. p 51–78.

Richter MA, Summerfeldt LJ, Antony MM, Swinson RP. 2003. Obsessive–compulsive spectrum conditions in obsessive–compulsive disorder and other anxiety disorders. Depress Anxiety 18: 118–127.

Roper G, Rachman S. 1976. Obsessional-compulsive checking: experimental replication and development. Behav Res Ther 14:25–32.

Roper G, Rachman S, Hodgson R. 1973. An experiment on obsessional checking. Behav Res Ther 11:271–277.

Salkovskis PM. 1985. Obsessional-compulsive problems: a cognitive-behavioural analysis. Behav Res Ther 23:571–583.

Salkovskis PM. 1989. Obsessions and compulsions. In: Scott J, Williams JM, Beck AT, editors. Cognitive therapy in clinical practice: an illustrative casebook. Florence, KY, USA: Taylor & Francis/Routledge. p 50–77.

Savron G, Fava GA, Grandi S, Rafanelli C, Raffi AR, Belluardo P. 1996. Hypochondriacal fears and beliefs in obsessive–compulsive disorder. Acta Psychiatr Scand 93:345–348.

Saxena S, Rauch SL. 2000. Functional neuroimaging and the neuroanatomy of obsessive compulsive disorder. Psychiatr Clin North Am 23:563–586.

Stein DJ, Hollander E, Anthony DT, Schneier FR, Fallon BA, Liebowitz MR, Klein DF. 1992. Serotonergic medications for sexual obsessions, sexual addictions, and paraphilias. J Clin Psychiatry 53:267–271.

Stein DJ, Liu Y, Shapira NA, Goodman WK. 2001. The psychobiology of obsessive–compulsive disorder: how important is the role of disgust? Curr Psychiatry Rep 3:281–287.

Stein DJ, Lochner C. 2006. Obsessive–compulsive spectrum disorders: a multidimensional approach. Psychiatr Clin North Am 29:343–351.

Storch EA, Geffken GR, Merlo LJ, Mann G, Duke D, Munson M, Adkins J, Grabill KM, Murphy TK, Goodman WK. 2007. Family-based cognitive-behavioral therapy for pediatric obsessive–compulsive disorder: comparison of intensive and weekly approaches. J Am Acad Child Adolesc Psychiatry 46:469–478.

Storch EA, Merlo LJ. 2006. Treatment of the patient with obsessive–compulsive disorder. J Fam Prac 55:329–333.

Streichenwein SM, Thornby JI. 1995. A long-term, double-blind, placebo-controlled crossover trial of the efficacy of fluoxetine for trichotillomania. Am J Psychiatry 152:1192–1196.

Summerfeldt L, Hood K, Antony M, Richter M, Swinson R. 2004. Impulsivity in obsessive–compulsive disorder: comparisons with other anxiety disorders and within tic-related subgroups. Pers Individ Differences 36:539–553.

Taylor S. 1996. Meta-analysis of cognitive-behavioral treatments for social phobia. J Behav Ther Exp Psychiatry 27:1–9.

Tollefson GD, Rampey Jr AH, Potvin JH, Jenike MA, Rush AJ, Kominguez RA, Koran LM, Shear MK, Goodman W, Genduso LA. 1994. A multicenter investigation of fixed-dose fluoxetine in the treatment of obsessive compulsive disorder. Arch Gen Psychiatry 51:559–567.

van den Hout M, van Pol M, Peters M. 2001. On becoming neutral: effects of experimental neutralizing reconsidered. Behav Res Ther 39:1439–1448.

Van Minnen A, Hoogduin KA, Keijsers GP, Hellenbrand I, Hendriks GJ. 2003. Treatment of trichotillomania with behavioral therapy or fluoxetine: a randomized, waiting-list controlled study. Arch Gen Psychiatry 60:517–522.

Wells A. 2000. Emotional disorders and metacognition: Innovative cognitive therapy. Wells, Adrian; New York, NY, USA: John Wiley & Sons.

Whiteside SP, Port JD, Abramowitz JS. 2004. A meta-analysis of functional neuroimaging in obsessive–compulsive disorder. Psychiatry Res 32:69–79.