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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Received for publication 22 February 2008; Revised 22 February 2008; Accepted 25 February 2008

DOI 10.1002/da.20488
Published online in Wiley InterScience (www.interscience.wiley.com).
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 DSMs 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; 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, phobias, 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. Neutalizing 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 [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 neural circuitry 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, Summefeldt 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., DeVeau and Giss 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 (Chris- tensen 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 psychotic 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

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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,

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<th>Diagnosis</th>
<th>Fear-evoking stimuli</th>
<th>Underlying beliefs</th>
<th>Safety behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td>OCD</td>
<td>Intrusive thoughts and related triggers</td>
<td>Thoughts are equivalent to actions, inflated responsibility for preventing harm [e.g., OCCWG, 1997]</td>
<td>Avoidance, checking, hoarding, washing, ordering/arranging, covert neutralizing</td>
</tr>
<tr>
<td>Specific phobia</td>
<td>Snakes, heights, injections, storms, etc.</td>
<td>Overestimation of the likelihood or severity of danger from the feared stimulus [Beck and Emery, 1985]</td>
<td>Avoidance, drinking alcohol before flying, distraction during injections</td>
</tr>
<tr>
<td>Social phobia</td>
<td>Social situations, embarrassment, etc.</td>
<td>Other people are highly judgmental, negative evaluation is intolerable [Clark and Wells, 1995]</td>
<td>Avoidance of social situations, speaking softly, using alcohol</td>
</tr>
<tr>
<td>Panic disorder and agoraphobia</td>
<td>Arousal-related body sensations</td>
<td>Heart palpitations signify a heart attack, dizziness leads to fainting [Clark, 1986]</td>
<td>Agoraphobic avoidance, sitting down, going to emergency room, drinking water, calling a safe person</td>
</tr>
<tr>
<td>PTSD</td>
<td>Memories of traumatic events</td>
<td>Nowhere is safe, I could have prevented the trauma [Foa and Rothbaum, 1998]</td>
<td>Avoidance of triggers, distraction, relying on others for safety, carrying a flashlight</td>
</tr>
<tr>
<td>GAD</td>
<td>Images of low-probability catastrophes</td>
<td>Intolerance of uncertainty, the world is a dangerous place [Wells, 2000]</td>
<td>Calling loved ones to verify safety, asking for reassurance</td>
</tr>
</tbody>
</table>

OCD, obsessive–compulsive disorder; PTSD, posttraumatic stress disorder; GAD, generalized anxiety disorder.
symptoms (e.g., contamination fears, fears of unwanted OCD as it is currently conceptualized. Some OCD likely a consequence of the extreme heterogeneity of disorders or OCSDs. This perception, however, is does not appear to fit well with either the anxiety ‘classic’ OCD symptoms [Lochner et al., 2005]. Other characteristics that distinguish it from more often ego-syntonic nature of hoarding symptoms among OCD subtype [e.g., Grisham et al., 2005], given the 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 multi- determined 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


Depression and Anxiety