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Obsessive-Compulsive and Related Disorders: A Critical Review of the New Diagnostic Class

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obsessive-compulsive disorder, obsessive-compulsive and related disorders, trichotillomania, excoriation, hair pulling, hoarding, body dysmorphic disorder

Abstract

The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* includes a new class of obsessive-compulsive and related disorders (OCDs) that includes obsessive-compulsive disorder (OCD) and a handful of other putatively related conditions. Although this new category promises to raise awareness of underrecognized and understudied problems, its empirical validity and practical utility are questionable. This article reviews the phenomenology of OCD and then presents a critical analysis of the arguments underlying the new OCD class. This analysis leads to a rejection of the OCD classification on both scientific and logical grounds. The article closes with a discussion of the treatment implications of the OCDs approach.

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INTRODUCTION

With the release of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) (Am. Psychiatr. Assoc. 2013), increased attention has been paid to obsessive-compulsive disorder (OCD) and a handful of other conditions that were pulled from various chapters of the previous DSM and grouped together in a novel diagnostic class: the obsessive-compulsive and related disorders (OCDs). This new category has raised awareness of serious but often understudied and underrecognized problems such as hoarding and body dysmorphic disorder (BDD), which are now classified as OCDs. Yet the empirical validity and practical utility of this new DSM category is questionable. In this article, we first provide a review of the essential phenomenology of OCD, including the most recent research findings, before turning to a critical analysis of the arguments underlying the new OCD class in DSM-5. We close with a discussion of the clinical and treatment implications of a diagnostic system that focuses on the superficial form of symptoms (i.e., repetitive thoughts and behaviors) as opposed to psychological processes and functions.

DSM-5: *Diagnostic and Statistical Manual of Mental Disorders*, 5th edition

OCD: obsessive-compulsive disorder

OCDs: obsessive-compulsive and related disorders

BDD: body dysmorphic disorder

PHENOMENOLOGY OF OBSESSIVE-COMPULSIVE DISORDERS

The two cardinal features of OCD as defined in DSM-5—obsessions and compulsions—are essentially unchanged since DSM-III. According to the diagnostic criteria, either symptom may be present to obtain the diagnosis.

Obsessions

Obsessions are defined as persistent intrusive thoughts, ideas, images, impulses, or doubts that the individual experiences as unwanted, unacceptable, or senseless. These thoughts evoke subjective distress (e.g., anxiety, fear, doubt) concerning the possibility or uncertainty of negative

Table 1 Common obsessions

Category	Example
Contamination	Fear of germs from saliva or genitals What if the public toilet I used had the AIDS virus on it?
Responsibility for harm	If I don't warn people that I might have dropped my medication, a child will eat it and die, and it will be my fault What if I hit a pedestrian with my car by mistake?
Sex and morality	Unwanted thoughts of incestuous relationships Unwanted impulses to grab women's buttocks
Violence	Thought of stabbing someone with the knife one is using to eat Thoughts of loved ones losing their lives in terrible accidents
Religion	What if I don't really believe in God? Images of Jesus with an erection on the cross
Symmetry and order	The sense that odd numbers are "bad" The feeling that books have to be arranged "just right" on the shelf

consequences (e.g., going to hell, becoming ill) and are not simply everyday worries about topics such as work, health, relationships, or finances. Obsessions generally relate to a finite number of themes, including: (a) germs, contamination, and illness; (b) responsibility for causing or failing to prevent harm, disasters, mistakes, or bad luck; (c) sex and morality; (d) violence; (e) religion; and (f) symmetry, order, and exactness. Their specific content, however, is rather heterogeneous and typically relates to values, interests, vulnerabilities, and uncertainties that the person holds as greatly important in his or her life (e.g., responsibility for fires, unwanted thoughts of harming loved ones, fears of punishment from God, "What if I molest my infant by mistake?").

Recent research indicates that obsessions can be broadly divided into two classes—autogenous and reactive—on the basis of how they are triggered and experienced (Lee & Kwon 2003, Lee & Telch 2010, Lee et al. 2005). Autogenous obsessions are thoughts and images that intrude into consciousness with or without identifiable triggers. They are experienced as highly repugnant and distressing, and the person resists them strongly. Autogenous obsessions typically take the form of unacceptable sexual, aggressive, or immoral ideas, doubts, images, or impulses. Reactive obsessions, on the other hand, are evoked by identifiable situations and stimuli (e.g., driving, bathrooms) and might be perceived as rational enough to provoke compensatory behaviors (e.g., checking, washing compulsions). These types of obsessions typically concern contamination, illness, mistakes, accidents, and symmetry. Most people with OCD display multiple types of obsessions; **Table 1** shows common examples.

Compulsive Rituals, Avoidance, and Neutralizing

Although topographically distinct from one another, compulsive rituals, avoidance behavior, and neutralizing strategies belong to the same functional class of behaviors. That is, they are all responses to obsessions that serve to reduce anxiety, control or resist unwanted thoughts, or reduce fears that the consequences featured in the obsessions will occur. Compulsive rituals, avoidance, and neutralizing all function as anxiety-reduction strategies.

Rituals. To reduce or otherwise control the anxiety and distress provoked by obsessional thoughts, individuals with OCD might perform overt or covert compulsive rituals—defined in DSM-5 as behavioral or mental acts that are completed according to a set of idiosyncratic personal

Table 2 Common compulsive rituals

Category	Case example
Decontamination	Washing hands for 30 minutes at a time after touching one's shoes Wiping down all mail and groceries brought into the house for fear of germs from the letter carrier and store clerks
Checking	Driving back to check that no accidents were caused at the intersection Checking locks, appliances, electrical outlets, and windows
Repeating routine activities	Getting up and down out of a chair until the obsessional thought has been dismissed
Ordering/arranging	Saying the word "correct" whenever one hears the word "right" Fixing pictures on the wall until they are hung "just right"
Mental rituals	Replacing a "bad" thought by thinking of a "good" thought Repeating a prayer until it is said "just right"

rules. For the most part, rituals are deliberate, purposeful, and goal-directed; yet they are clearly senseless or excessive in relation to the obsession they are designed to neutralize (e.g., checking the window locks 10 times before leaving the house; asking repeatedly for assurance that one hasn't murdered one's children). Rituals in OCD are not contentless motoric behaviors such as tics, nor are they pointless actions or perseverative behaviors as are sometimes observed in individuals with psychosis or developmental disorders (e.g., flapping in children with autism spectrum disorders). In OCD, the rituals are deliberate behaviors performed with the aim of reducing obsessional anxiety. Common overt rituals include excessive decontamination (e.g., cleaning, washing), counting, repeating routine actions (e.g., walking through a doorway several times until an obsession is dismissed), checking (e.g., windows, appliances), and repeatedly asking questions for reassurance. Some examples of covert or mental rituals include internally repeating "good" numbers or phrases (e.g., "I love God") to neutralize obsessional thoughts and trying to mentally review one's actions to reassure oneself that nothing bad has happened. **Table 2** presents examples of common rituals.

Avoidance. Although not listed among the DSM criteria, avoidance behavior is present to some degree in most people with OCD. Specifically, avoidance is intended to prevent exposure to situations that would provoke obsessional anxiety and compulsive rituals. For example, one man avoided going to swimming pools because seeing children in bathing suits evoked unwanted thoughts of child molestation. Other patients engage in avoidance so that they do not have to carry out time-consuming or embarrassing rituals. For instance, a woman with obsessional fears of mistakenly hitting pedestrians avoided all driving in order to not have to compulsively stop her car to explore the roadside for injured or dead bodies.

Neutralizing. Many people with OCD also deploy strategies in response to obsessional fear that technically do not meet DSM-5 criteria for compulsive rituals. That is, these behaviors are not rule-bound or repeated to excess. Examples include purposeful distraction, thought suppression, warning others of potential danger, and briefly wiping one's hands on one's pants. Such in-situation neutralizing behaviors can take infinitely diverse forms, some of which may be remarkably subtle.

Insight

Individuals with OCD vary widely with regard to their insight into the senselessness of their obsessions and rituals. Some readily acknowledge the irrationality of their obsessive thoughts and

compulsive behaviors (e.g., “I realize the probability of getting sick from touching my shoe is very low, but I just can’t take the chance.”), whereas others are firmly convinced that these are entirely rational behaviors (e.g., “I will definitely lose my eyesight if I shake hands with someone who is blind.”). An individual’s degree of insight may change over time and can vary among his or her different obsessions. For example, a woman might recognize her obsessive thoughts about bad luck from the number 6 as senseless and yet have poor insight into the irrationality of her contamination obsessions. The DSM-5 criteria for OCD include the specifiers (*a*) good or fair insight, (*b*) poor insight, and (*c*) absent insight/delusional beliefs to denote the degree to which the person views his or her OCD symptoms as logical.

Symptom Dimensions

As we have discussed, obsessions and compulsions are functionally related, which is another way of saying that rituals (and avoidance and neutralizing behaviors) are attempts to reduce obsessional distress (e.g., Rachman et al. 1976). Unfortunately, this functional relationship is not reflected well in the DSM-5’s definition of OCD. This is because the disorder can be diagnosed by the presence of either obsessions or compulsions, which leads to the inaccurate perception that obsessions and compulsions are independent of one another. Not only does research confirm that obsessions and compulsions are functionally related (Foa et al. 1995), but there are also empirically replicable OCD symptom dimensions consisting of both obsessions and compulsions (e.g., Abramowitz et al. 2010, Deacon & Abramowitz 2005, McKay et al. 2004). Specifically, obsessions about responsibility for harm and mistakes are associated with checking rituals that serve to provide reassurance; obsessions about incompleteness and the need for symmetry and exactness occur along with ordering, arranging, and counting rituals; contamination obsessions that feature the sense of tactile or mental contamination or disgust co-occur with washing and cleaning rituals; and religious, sexual, and violent obsessions often trigger mental rituals, efforts to neutralize and suppress unwanted thoughts, and other forms of checking and reassurance-seeking behaviors.

Hoarding, which has sometimes been included as a dimension or presentation of OCD, is no longer considered as such on the basis of important differences between hoarding and other OCD symptom dimensions (e.g., Wheaton et al. 2011). For example, hoarding is more strongly related to other sorts of psychopathology (e.g., personality disorders) than to OCD symptoms (e.g., Frost & Tolin 2008). Hoarding is also associated with an earlier age of onset and distinct biological phenomena relative to OCD symptoms (Wheaton et al. 2008). Finally, hoarding shows a weaker response to pharmacological and psychological treatments with demonstrated efficacy for OCD (e.g., Abramowitz et al. 2003). These differences were compelling enough to lead to the creation of hoarding disorder as a new diagnostic entity (included within the OCRD chapter) in DSM-5.

OBSESSIVE-COMPULSIVE AND RELATED DISORDERS IN DSM-5

Through DSM-IV-TR, OCD was included among the anxiety disorders along with conditions such as specific and social phobias, panic disorder, posttraumatic stress disorder (PTSD), and generalized anxiety disorder. This grouping made sense on two levels. First, at a purely descriptive level, OCD symptoms appear to overlap to a great degree with the main features of anxiety disorders: excessive and irrational fear, anxious apprehension, and avoidance behavior. The second level on which OCD overlaps with the other anxiety disorders is of even greater interest because it transcends mere descriptive psychopathology and also has treatment implications: OCD and anxiety disorders are maintained by the same psychological mechanisms involving cognitive distortions and negative reinforcement. Moreover, all of these conditions respond to a specific

intervention that promotes fear extinction—exposure therapy (Abramowitz et al. 2012, Barlow 2004). We return to overlaps between OCD and anxiety disorders below.

Some authors, however, assert that OCD was incorrectly classified as an anxiety disorder and belongs instead to a group of conditions (the OCRDs) that “share compulsive behavior and failures in behavioral inhibition” (Fineberg et al. 2011, p. 21). Although the OCRD notion was initially conceived on the basis of overlaps in overt symptom presentation among the disorders proposed for inclusion (e.g., repetitive thinking and behavior patterns; Hollander et al. 1996), proponents of this approach also assert that the OCRDs overlap in terms of their etiology, associated features (e.g., age of onset), patterns of comorbidity, and treatment response profile (e.g., Hollander et al. 2005). In DSM-5, OCD is the flagship diagnosis of this new category, which also includes: (a) two new disorders, hoarding disorder and skin picking (excoriation) disorder; (b) a former somatoform disorder, BDD; and (c) a former impulse control disorder, trichotillomania (now also known as hair-pulling disorder).

Many scientists and practitioners with expertise in OCD question (a) the removal of OCD from the anxiety disorders, (b) the creation of the new OCRD diagnostic category, and (c) decisions regarding which conditions have been included as part of this new category. Mataix-Cols et al. (2007), for example, surveyed 187 mental health professionals and researchers who specialize in OCD and found that 40% disagreed with moving OCD out of the anxiety disorders category, whereas the vast majority of those who agreed with creating a separate OCRD category believed that it should include OCD, BDD, trichotillomania, and possibly tic disorders and hypochondriasis (the latter two are not OCRDs).

Those who made decisions about the DSM-5 (e.g., Fineberg et al. 2011, Hollander et al. 2005) provided the following arguments for shifting OCD out of the anxiety disorders and creating the new OCRD classification:

1. The cardinal symptoms of OCD and the OCRDs are repetitive thoughts and behaviors and a failure of behavior inhibition;
2. OCD and the OCRDs overlap in their age of onset, comorbidity, and family loading;
3. OCD and the OCRDs share brain circuitry and neurotransmitter/peptide abnormalities; and
4. OCD and the OCRDs share similar treatment response profiles.

Over the past decade, proponents of the OCRD approach have widely disseminated their arguments in academic and clinical publications (Fineberg et al. 2011, Hollander & Evers 2004, Hollander et al. 2007, Lochner & Stein 2006, Stein & Lochner 2006). These articles use research to support each of the four arguments listed above. When these arguments are closely examined, however, they clearly show flaws in their underlying logic and a lack of empirical support. We now turn to a critical analysis of each.

CRITICAL REVIEW OF THE BASIS FOR THE OBSESSIVE-COMPULSIVE AND RELATED DISORDER DIAGNOSTIC CLASS

Repetitive Thoughts and Behaviors in Obsessive-Compulsive Disorder and Obsessive-Compulsive and Related Disorders

The DSM-5 provides only a vague overview of how OCD and the other OCRDs are related and why they have been grouped together. After a brief description highlighting the repetitiveness of obsessional thinking and compulsive behavior, the manual states that “some other obsessive-compulsive and related disorders are also characterized by preoccupations and by repetitive behaviors or mental acts in response to the preoccupations. Other obsessive-compulsive and related

disorders are characterized primarily by recurrent body-focused repetitive behaviors (e.g., hair pulling, skin picking) and repeated attempts to decrease or stop the behaviors” (Am. Psychiatr. Assoc. 2013, p. 235).

Proponents also assert that the repetitive thinking or behavior patterns fall along a spectrum of failure in behavior inhibition—the inability to cease one’s actions—with compulsive and impulsive behaviors at the opposite ends (Fineberg et al. 2011). In other words, at one end of the continuum are compulsive disorders such as OCD and BDD, which are characterized by repetitive behaviors that serve to reduce risk and harm. Impulse control problems (e.g., skin picking and hair pulling), in contrast, occupy the other end of the continuum, because these conditions are characterized by behaviors that often have a pleasurable consequence (even if the inability to resist performing these behaviors causes distress to the person). To this end, the unifying factor among the OCRDs is the presence of repetitive behaviors that the person seemingly cannot stop performing.

By emphasizing the presence of repetitive behaviors (compulsive, body-focused, impulsive, or otherwise) as a criterion for the OCRD class, this approach overlooks other essential (and arguably more fundamental) features of OCD and putatively related conditions. This oversight is, however, consistent with the DSM’s largely descriptive and atheoretical approach to taxonomy, wherein the disorders are defined merely by lists of signs and symptoms rather than by theoretically grounded conceptual models with empirical support. The definition of OCD as including either obsessions or compulsions is an emblem of this descriptive approach to diagnosis, which leads to drawing parallels between OCD symptoms and other disorders that involve repetitive thinking or behavior. However, the trouble with this method for grouping disorders is easy to recognize if we apply it to a different set of conditions. Consider, for example, the following reasoning:

- Vomiting is a symptom of bulimia nervosa.
- Vomiting is a symptom of salmonella poisoning.
- Therefore, bulimia and salmonella poisoning are part of the same family of disorders.

It is indeed easy to see how, from this perspective, repetitive hair pulling and skin picking might seem related to OCD and might be erroneously categorized as part of a same family of disorders. As we have discussed, however, in OCD the compulsive behavior is performed in response to specific cues such as particular situations or thoughts that evoke anxiety concerning feared outcomes. Moreover, anxiety and compulsive urges generally subside (at least somewhat, and only temporarily) after completion of the ritual. The evocation of compulsive urges by obsessional fear and the immediate reduction in anxiety following the compulsive behavior are the hallmarks of OCD. This problem is hardly a failure in behavioral inhibition, given that people with OCD are often able to delay or stop their ritualizing and many do so regularly—for example, if rituals will cause embarrassment or if instructed to do so as part of treatment by exposure and response prevention (ERP). Moreover, research shows that behavioral inhibition itself is a vulnerability factor for OCD and anxiety disorders (Coles et al. 2006, Rosenbaum et al. 1993). But is the functional pattern that characterizes OCD present in other disorders of the OCRD class, such as hair pulling and skin picking?

Trichotillomania (hair pulling). The DSM-5 includes five criteria for trichotillomania; the first two are: (a) recurrent hair pulling resulting in hair loss and (b) repeated attempts to stop the hair pulling. (The last three criteria specify that the problem causes distress or impairment, is not attributable to substance use or a medical condition, and is not better accounted for by another mental disorder.) If we employ these two criteria alone in understanding patients who repetitively pull their hair, they appear to share with people with OCD the presence of repetitive behaviors and the apparent failure in behavioral inhibition. Yet a more careful examination reveals that trichotillomania is phenomenologically distinct from OCD.

ERP: exposure and response prevention

Although OCD and trichotillomania both involve some form of repetitive or recurrent behavior, the intrusive anxiety-evoking obsessional thoughts that occur in OCD are not present in trichotillomania. In explaining the inclusion of trichotillomania as a form of OCD, however, Stein et al. (1995) minimized the importance of obsessions, writing that the differences in emphasis between obsessions in OCD and compulsive behavior in trichotillomania are “subtle” (p. 29). Yet this is not a subtle difference but rather a very clear and important distinction, because obsessional fear motivates the repetitive behavior (i.e., compulsive rituals) in OCD. In contrast, research indicates that urges to pull hair in trichotillomania are precipitated not by obsessional fears, but rather by feelings of general tension, depression, anger, boredom, frustration, indecision, or fatigue (Christenson et al. 1993). Studies also suggest that hair pulling in trichotillomania leads to pleasurable feelings (Grant & Potenza 2004, Schreiber et al. 2011), a phenomenon not observed with rituals in OCD (Stanley et al. 1992).

Skin picking (excoriation). The diagnostic criteria for excoriation are identical to those for trichotillomania, substituting skin picking for hair pulling. Yet, although skin picking has not been as intensively studied as hair pulling, its distinctions from OCD are just as clear; that is, excoriation does not involve obsessional thoughts, and repetitive skin picking performs different functions than do compulsive rituals in OCD. Specifically, pathological skin picking may be triggered by an array of antecedents, including general stress, apprehension, time away from scheduled activities (e.g., sitting on the couch, watching television, reading), boredom, and feelings of tiredness or anger (Arnold et al. 2001, Neziroglu et al. 2008). Moreover, unlike in OCD, emotion regulation difficulties and emotional reactivity have been shown to predict skin picking even after controlling for depression, anxiety, and worry (Snorrason et al. 2010). Skin picking can also be triggered by the feel (e.g., a bump or unevenness) or look (e.g., a blemish or discoloration) of the skin. Additionally, episodes of skin picking (and hair pulling) often begin without the person’s awareness (i.e., they are unfocused) but become more conscious (i.e., focused) after a period of time (Keuthen et al. 2000, Lochner et al. 2002). This is in contrast to the deliberate anxiety-reducing compulsive behavior in OCD.

Hoarding disorder. The DSM-5 describes hoarding disorder as characterized by excessive acquisition and difficulty discarding or parting with possessions—even those of limited value—due to an intense perceived need to save such items and distress associated with discarding them. As a result, large numbers of possessions accumulate and clutter the person’s living areas to the extent that it may become difficult to use the living space for its intended purposes. Hoarding is actually observed in a number of DSM conditions, including depression, anorexia nervosa, schizophrenia, and dementia (e.g., Abramowitz et al. 2008, Frankenburg 1984, Luchins et al. 1992), and the text of the DSM-5 does not make clear why it is included as an OCD. For several decades, hoarding was considered to be a symptom of OCD (e.g., Coles et al. 2003); yet hoarding symptoms differ markedly from those of OCD. First, although hoarding involves recurring thoughts of acquiring and maintaining possessions, these thoughts are not experienced as fear provoking in the same way that OCD obsessions are, and they are not particularly intrusive or unwanted (Rachman et al. 2009, Wheaton et al. 2011). Moreover, excessive acquiring and saving does not result in an escape from obsessional anxiety in the way that washing or checking in OCD does, and thus cannot be conceptualized as compulsive or ritualistic in the OCD sense.

Body dysmorphic disorder. There are two main diagnostic criteria for BDD in DSM-5. The first is a preoccupation with one or more perceived defects or flaws in one’s physical appearance (i.e., a belief that one looks ugly or deformed) that are only slightly or not observable to others.

The second is excessive repetitive behaviors (e.g., checking one's appearance) or mental acts (e.g., comparing oneself to others) that are performed in response to the preoccupation. Research and clinical observations indicate that in addition to the superficial similarities between the symptoms of BDD and OCD—namely, both involve repetitive thoughts and behaviors—there are also more genuine similarities in how these repetitive symptoms are experienced and how they function to maintain their respective conditions. In particular, as with obsessions in OCD, the appearance-related preoccupations in BDD are experienced as intrusive, unwanted, and anxiety provoking, and as with compulsive rituals in OCD, the repetitive behaviors have an anxiety-reduction function and are not experienced as pleasurable (Phillips et al. 2010). Moreover, research shows that the excessive checking and comparing behaviors contribute to maintaining the appearance-related preoccupations in much the same way that compulsive rituals maintain obsessional fears in OCD (Phillips et al. 2010, Veale & Riley 2001, Windheim et al. 2011). Accordingly, the symptoms in BDD appear to overlap in important ways with those of OCD.

Conclusions. Apparent overlaps between the symptoms of OCD and those of other mental disorders may be found on two levels. The first and more trivial level is the form, or topography, of symptoms. It is undeniable that OCD and the other OCRDs share repetitive thinking or behavior as a symptom. Yet only some of these repetitive behaviors resemble OCD on the second and more critical level: symptom function or motivation. Under careful examination these phenomena reveal rich patterns of thought and behavior that go beyond repetitiveness and that are unique to OCD and BDD among the OCRDs. Careful examination of behaviors such as skin picking and hair pulling leads to a rich understanding of these impulse control problems as well, but the mechanisms of thinking and behaving involved here are quite distinct from those of OCD and BDD.

Do compulsivity and impulsivity exist on a spectrum, as advanced by OCRD proponents? Do we have a firm basis for considering the OCRDs as characterized by failures in behavioral inhibition? Although this notion may have some commonsensical appeal, the meanings of compulsivity, impulsivity, and spectrum have not been well defined by the proponents of this categorization. What is known is that the compulsive behavior in OCD and BDD has an escape function (i.e., from obsessional fear and preoccupation) and is negatively reinforced by the (albeit temporary) reduction in distress that it engenders, and that the impulsive behavior that characterizes trichotillomania and excoriation is gratifying in that the patient feels a rush of excitement when the behavior is performed that is intrinsically positively reinforcing (Grant & Potenza 2004). Yet the extant research suggests no specific relationship between these types of behaviors. Indeed, as we discuss below, DSM-IV impulse control disorders occur at low rates among patients with OCD (Bienvenu et al. 2000), and individuals with OCD do not exhibit greater levels of impulsivity than those with other mental disorders (Summerfeldt et al. 2004). Accordingly, if we consider overlap in symptom function as a criterion for relatedness to OCD, only BDD should legitimately be included as an OCRD.

Overlaps in Age of Onset, Comorbidity, and Family Loading

Proponents of the OCRD category argue that OCD and the putative OCRDs overlap in terms of their onset age, comorbidity patterns, and familial patterns; and that such overlaps indicate relationships among these conditions. In this section we address the science and logic of these claims.

Age of onset and course. Consistent findings across numerous studies suggest that although OCD may begin at any time from childhood through old age, its mean age of onset is in the late teenage years into the mid-twenties (e.g., Anholt et al. 2014, Antony et al. 1998). OCD is

also a chronic condition that, absent effective treatment, waxes and wanes throughout its course. Likewise, the other OCRDs typically onset in adolescence through early adulthood and follow similar courses (e.g., Bjornsson et al. 2013, Flessner et al. 2010, Grisham et al. 2006, Odlaug & Grant 2012, Wilhelm et al. 1999). Similarity in age of onset and course, however, is not a persuasive argument for the OCRD diagnostic class, because these demographic features are not at all specific to OCD and the other OCRDs. A perusal through the DSM-5 shows that many depressive, bipolar, anxiety, somatic symptom, dissociative, sexual, sleep, personality, substance-related, psychotic, and eating disorders also begin during this time of life and exhibit a chronic course if effective treatment is not sought. Thus, the fact that the OCRDs share these characteristics does not indicate anything specific about these conditions, much less that they are related to one another or to OCD.

Patterns of comorbidity. Proponents of the OCRD approach have argued that another basis for grouping these conditions together is their comorbidity with one another and with OCD. Several large studies, however, do not support this assertion. For example, Bienvenu et al. (2000) found that among 80 individuals with OCD, the comorbidity rate with BDD was 15%, whereas only 4% of these patients had comorbid trichotillomania. This supports our contention that BDD is more closely related to OCD than is trichotillomania. Other studies have reported largely similar results (Jaisoorya et al. 2003, Lovato et al. 2012), suggesting that other than BDD, the OCRDs are rather uncommon among individuals with OCD.

It is somewhat astounding that the proponents of moving OCD out of the anxiety disorders would appeal to the comorbidity argument to make their case for the OCRDs, because research shows that OCD is more consistently comorbid with anxiety disorders than with OCRDs (e.g., Jaisoorya et al. 2003). For example, in one study 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, and 30.7% for specific phobias (Nestadt et al. 2001). Thus, the existing empirical research suggests that OCD is more closely related (i.e., 5 to 10 times more closely associated) to other anxiety disorders than to some of the OCRDs.

A logical, as opposed to empirical, problem with using comorbidity patterns to group disorders into categories or classes is that comorbidity signifies merely some association among disorders—not necessarily an etiologically or nosologically meaningful one. That is, comorbidity is commonplace among most major mental disorders, and there are numerous explanations for this phenomenon that do not require co-occurring conditions to be related to one another, as reasoned by OCRD proponents. As an example, consider that alcohol dependence and posttraumatic stress disorder have a high rate of comorbidity that goes beyond a chance association (Kramer et al. 2014). Although there might be several reasons for the co-occurrence of these disorders (e.g., both may develop when reintegrating into life following military deployment), we would not suggest they are part of the same diagnostic category. Similarly, at least half of OCD sufferers also meet criteria for a depressive disorder, yet this doesn't suggest that OCD and depression are part of the same diagnostic class. Thus, comorbidity patterns are of limited value in understanding the uniqueness of OCD and its links to OCRDs.

Family patterns. Proponents of the OCRD approach have also asserted that if OCRDs occur frequently in relatives of people with OCD, then such problems are related and share a common genetic underpinning. The available research, however, does not support this contention. For example, Bienvenu et al. (2000) found that the lifetime prevalence of trichotillomania in first-degree relatives of adults with OCD was only 1%. In contrast, the rates of anxiety disorders among first-degree relatives of people with OCD are far higher than the rates of OCRDs among

relatives of OCD sufferers (e.g., Bienvenu et al. 2000, Nestadt et al. 2001). Thus, the assertion that familial pattern represents a valid basis for grouping together the OCRDs once again more strongly supports the notion that OCD should be grouped with the anxiety disorders. In other words, the available data are more consistent with the notion of shared genetic vulnerability among OCD and anxiety disorders as opposed to genetic links between OCD and the putative OCRDs.

Overlaps in Neurobiological Factors

Proponents of the OCRD classification assert that OCD and the other conditions in this group share underlying neurobiological abnormalities—specifically, brain circuitry and neurotransmitter irregularities (Fineberg et al. 2011, Hollander et al. 2005). Fineberg and colleagues (2011, p. 1), for example, argue that “the focus has shifted from learning models in which anxiety-driven obsessions entrain neutralizing compulsions to an emphasis on the primacy of obsessional thoughts and compulsive behaviors as disorders of basal ganglia dysregulation.” There are, however, a number of crucial empirical and logical difficulties with strong contentions such as this one. In this section we discuss how data from brain imaging, pharmacotherapy outcome studies, and studies of neurotransmitter systems have been misrepresented as suggesting a common neurobiological etiology of the OCRDs.

Brain structure and function. A number of studies—primarily brain imaging investigations—have found that individuals with and without OCD show differences in variables related to brain structure and function (for a review, see Whiteside et al. 2004). Much less brain imaging research has been conducted with the other OCRDs, yet the few comparisons to healthy control groups also show some differences (e.g., Buchanan et al. 2013, Chamberlain et al. 2008, Grant et al. 2013, Mataix-Cols et al. 2011). Proponents of the OCRD model have exuberantly interpreted such data as verifying the presence of a common causal brain abnormality, imbalance, or defect across the OCRDs (e.g., Fineberg et al. 2011). A more careful examination of the evidence, however, indicates no basis for causal inferences or for using brain imaging studies as a basis for grouping the OCRDs.

One set of caveats concerns the methodology of most brain scan studies of OCRDs. First, sample sizes in most of these studies are prohibitively small [e.g., only 13 patients with skin picking in Grant et al. (2013) and only 18 with trichotillomania in Chamberlain et al. (2008)] and therefore preclude drawing strong inferences from the data. Another issue is the proclivity for type I error in such studies. That is, authors of brain imaging studies typically amass very large data sets with numerous brain-related dependent variables (e.g., numerous voxels or readings from different regions of the brain). They are therefore able to conduct numerous between-group comparisons in the search for significant differences between OCRD patients and control groups. Yet in many instances authors do not statistically correct for multiple tests. Such correction is important because of the high potential for false positives in brain imaging research [as has been sardonically illustrated by Bennett et al. (2009)]. Absent such correction, brain scan study data sets are almost guaranteed to yield the ambitious statistician with at least one desired result, even if by chance (Vul et al. 2009). But such findings might be artifacts. Even worse, authors sometimes highlight and interpret only the variables showing significant differences between groups and explain away (or even ignore) no-difference findings. These are serious problems, and they raise questions about the authenticity of brain imaging study conclusions as well as the suitability of such studies to serve as a basis for a new diagnostic category.

Another limitation of the brain imaging literature is the lack of direct comparison studies among the various OCRDs to test whether these disorders are similar with respect to brain-related

variables. There is also a dearth of comparisons between patients with OCRDs and with other mental disorders (e.g., eating disorders, impulse control disorders) to determine whether the brain differences in OCRDs are specific to these disorders or associated with mental illness more generally. Similarities among the OCRDs (and specificity to these conditions) have been assumed chiefly on the basis of independent studies rather than direct comparisons despite inconsistencies across these studies. Whiteside et al. (2004), for example, found that most brain imaging results with OCD patients could not be replicated. There also appear to be differences across the OCRDs. For example, using morphometric functional magnetic resonance imaging (fMRI), O'Sullivan et al. (1997) reported reduced basal ganglia volumes in trichotillomania that were inconsistent with studies in OCD in which reduced caudate and white matter volumes were found (e.g., Jenike et al. 1996).

To compound matters, even seemingly minor differences in how data are collected (e.g., how research participants are treated, the setting and context where the scans are completed, and slight differences in the scanners themselves) can influence brain imaging results. Thus, direct comparison studies in which identical procedures in the same lab are used to compare two or more OCRDs, or OCRDs and non-OCRDs, are imperative to provide convincing data regarding whether OCRDs (and only OCRDs) overlap with respect to putative brain abnormalities. Perhaps at some point such studies will be conducted and will provide consistent results that can form a potential basis for grouping together OCD and other disorders. Presently, however, such data do not exist.

The aforementioned issues aside, causal inferences such as those by Fineberg et al. (2011) still reach far beyond the available data because they overlook basic conceptual and logical limitations of the correlational studies on which they are based. Brain imaging studies are cross-sectional, and they involve no experimental manipulation. At best, such correlational designs can merely detect associations between variables—in this case between an OCRD diagnosis and brain structure or function. One cannot logically infer the presence of brain abnormalities or dysregulation from correlational studies any more than one could infer that studies showing an association between anorexia nervosa and being female indicate that being female is an abnormality or a dysregulation. The correlation of a certain variable with the symptoms of a mental disorder does not imply that this variable signals the presence of an abnormality or dysregulation with etiological significance. That is, it is logically improper to infer causation from correlation. In the absence of true experimental data (i.e., manipulation of an independent variable under controlled settings to observe its effects on a dependent variable), conclusions regarding OCD and brain imaging findings must be restricted to those allowed by correlational data. It is equally plausible that the observed differences in brain structure and function between OCRDs and controls are the result of having an OCRD or are caused by one or more extraneous variables not measured in brain scan studies.

Finally, although brain imaging is an important tool in the study of brain-behavior relationships, proponents of the OCRD approach (and most of the lay public) appear to fall victim to the fallacy of naïve realism (the commonsense misconception that seeing is believing) and uncritically take brain scans at face value. Brain scans, however, are not raw snapshots of the brain's real-time functioning (Roskies 2007). Rather, they are highly processed representations of the brain's activities. Moreover, there is a considerable difference between the conclusion that people with OCRDs show enhanced activation in the basal ganglia—the accurate interpretation—and the less cautious conclusion that the activity in the basal ganglia represents an abnormality that causes OCRDs. Despite the accumulation of more than two decades' worth of brain imaging studies, there have been no major advances into the causes of mental illness (Insel 2009). Considering the empirical and logical limitations of brain imaging studies, the appeal to overlaps in brain structural and functional abnormalities as a basis for creating the OCRDs classification is unpersuasive.

Neurotransmission. Probably the most consistent (and yet still overstated) finding in the biological literature on OCD is that pharmacotherapy by selective serotonin reuptake inhibitors (SSRIs; e.g., fluoxetine, sertraline) can be effective (Greist et al. 1995). This, together with a small literature comparing serotonergic and nonserotonergic processes in OCD patients (Insel et al. 1985), led to the serotonin hypothesis that OCD is caused by a chemical imbalance—abnormalities in the serotonergic system (Barr et al. 1993, Zohar et al. 2004). Given the oversimplicity and lack of empirical support (as we discuss below in this section) for the serotonin hypothesis, as well as the fact that a typical response to SSRIs is a mere 20% to 40% reduction in OCD symptoms (and half of OCD patients show no response to these agents), some authors have searched for alternative chemical imbalance models. They have more recently argued that dopamine plays a role in OCD, largely on the basis of the significant role that dopamine mechanisms play in the effects of SSRIs (Denys et al. 2008, Zurofski et al. 2008). That is, SSRIs might be less selective than was once thought.

Here as well, OCD proponents draw unwarranted causal conclusions inferring that the effectiveness of SSRIs proves that the OCDs overlap in terms of abnormally functioning serotonin and dopamine systems (e.g., Fineberg et al. 2011). Yet specific models of etiology cannot logically be derived from knowledge of treatment response. This is a commonsense error known as *ex juvantibus* reasoning (i.e., reasoning backward from what helps) and a variation of the *post hoc, ergo propter hoc* (i.e., after this, therefore because of this) fallacy. Such tenuous reasoning is pandemic in the mental health field and has been used repeatedly over the past half century (Valenstein 2002, Whitaker 2011). The logical fallacy is clear if one considers, for example, the following: (a) By applying steroid cream, the skin rash goes away; (b) Therefore, the rash was caused by an abnormally low level of steroids. Similarly, there may be many possible mechanisms—in addition to serotonin and dopamine—by which SSRIs decrease OCD symptoms. Therefore, from an epistemological standpoint, successful response to a treatment cannot lead to definitive conclusions regarding the etiology of a disorder. This is a particular problem with the SSRIs because they appear to improve (albeit inconsistently) a multitude of mental conditions yet are also associated with a substantial placebo effect (e.g., Kirsch 2011).

Neurotransmitter models of OCDs could be supported by evidence from controlled studies showing differences in serotonergic or dopaminergic functioning between individuals with and without OCD, or better yet by studies in which these neurotransmitters are manipulated and lead to increased symptom expression. Yet, as with brain imaging studies, in the last few decades a considerable amount of energy has been devoted in OCD research to biological marker studies (in which biological variables are compared between groups) and biological challenge studies (in which an agent is given to induce symptoms) (e.g., Barr et al. 1993). Collectively, however, the findings from these investigations have been remarkably inconsistent (Barr et al. 1993, Koo et al. 2010, Zohar et al. 2004). A further problem is that virtually no marker or challenge study of the serotonin system has been conducted on OCDs other than OCD. Correlational studies have also found associations between OCD and dopamine functioning (e.g., Denys et al. 2004, 2006); yet, as discussed previously, because of how they are designed such correlational studies cannot address etiology.

There is no doubt that obsessive-compulsive behavior and the behaviors involved in the other OCDs involve the serotonin and dopamine systems at some level; yet there is no convincing evidence that these conditions are caused by abnormally functioning neurotransmitter systems. One problem with lumping together a group of disorders, such as the OCDs, on the basis of their relationship with serotonin and dopamine is that these neurotransmitters are involved with countless aspects of human behavior and cognition (e.g., depression). Thus, the argument that all OCDs are related to serotonin and dopamine functioning does not reveal anything specific about these disorders.

Conclusions. The idea, as asserted by Fineberg et al. (2011), that OCD and the other OCRDs are best viewed as disorders of basal ganglia dysfunction rather than as problems of learned behavioral responses to fear-provoking stimuli suggests that biological phenomena are more fundamental than psychological phenomena. Yet, although a brain and neurotransmitters are required to have an OCRD, biological processes are not more fundamental than psychological processes, and it does not follow that the best way to understand OCRDs is by reducing them to the level of biological functions. To draw a parallel, the best way to understand architecture is not to reduce it to the raw building materials. Although the nature of the building materials puts constraints on the types of structures that can be built, it does not characterize the structure's design or function.

Correspondingly, although brain imaging data provide information not obtainable through self-report or behavioral means, these data are not inherently more accurate, more meaningful, or more objective than psychological data (Miller & Keller 2000). Rather, biological and psychological approaches offer different types of data of potentially equal relevance for understanding OCRDs—not different levels of analysis. Neither underlies the other and neither explains away the other; they are simply different domains of information. Accordingly, the appeal to a common neurobiological etiology as a basis for grouping the OCRDs seems to be more a post hoc attempt to justify the OCRD classification than a cautious, scientifically- or logically-grounded argument.

Overlaps in Treatment Response

Treatment response is perhaps the most important litmus test for the validity of the OCRD class of disorders, because successful treatment is the ultimate goal of identification, classification, and research on potential etiological factors. Yet, once more, the OCRD conceptualization faces considerable conceptual and empirical difficulties. The DSM-5 notes the “clinical utility” (Am. Psychiatr. Assoc. 2013, p. 235) of grouping OCRDs together in the same diagnostic class, and OCRD proponents have specifically argued that these conditions overlap in terms of their response to pharmacological interventions—namely, preferential response to serotonergic medications (i.e., SSRIs; Fineberg et al. 2011). The appeal to this preferential response, however, is only clinically useful in delineating a class of OCRDs if three conditions are met: (a) A preferential response to SSRIs is observed uniformly among the OCRDs; (b) The preferential response to SSRIs is specific to the OCRDs (i.e., it is observed only among the OCRDs); and (c) SSRIs are the best treatment available for the OCRDs. Careful examination of the most prudently conducted research [i.e., randomized controlled trials (RCTs)] indicates that none of these conditions is met.

Assumption A: uniform response to SSRIs. Meta-analysis of the numerous RCTs with thousands of OCD patients indicates the efficacy of SSRIs (as well as clomipramine, which is a less selective SRI) relative to placebo (Eddy et al. 2004, Greist et al. 1995). The only available placebo-controlled trial in BDD also indicates that fluoxetine (an SSRI that is effective for OCD) is efficacious for this OCRD (Phillips et al. 2002). Yet SSRI response in the other OCRDs is inconsistent. One meta-analytic study by Bloch et al. (2007) found that, with the exception of clomipramine, SSRIs were no more effective than placebo in the treatment of trichotillomania. A second meta-analysis by Bloch et al. (2014) revealed that patients with hoarding symptoms were approximately 50% less likely than patients with OCD to respond to SSRIs. Only two placebo-controlled studies have been published for skin picking disorder, one showing significant reduction with fluoxetine on only one of three outcome measures (Simeon et al. 1997) and the other finding that citalopram was no more effective than placebo (Arbabi et al. 2008). The only conclusion that can be drawn from these data is that the OCRDs do not show a uniform response to SSRIs.

Assumption B: Preferential response to SSRIs is specific to OCRDs. Numerous RCTs demonstrate that SSRIs are efficacious in the treatment of problems other than the OCRDs, including unipolar depressive disorders (e.g., Fournier et al. 2010, Schatzberg & Nemeroff 2013) and anxiety disorders such as social anxiety disorder (Hedges et al. 2007). Thus, the OCRD conceptualization again runs into a lack of specificity problem: Because SSRIs help numerous conditions, the observation that a group of problems respond preferentially to these agents cannot reveal anything unique about these disorders.

CBT:
cognitive-behavioral
therapy

Assumption C: SSRIs are the best treatment for OCRDs. A recent meta-analysis of 13 RCTs directly comparing SSRIs and cognitive-behavioral therapy (CBT; mainly ERP) in the treatment of OCD revealed that CBT is at least as effective (if not more so) than SSRIs for OCD (Romanelli et al. 2014). Indeed, it is widely acknowledged that CBT is the first-line treatment for OCD (before SSRIs in most cases; Koran et al. 2007, NICE 2005). Many of these studies included patients with hoarding behaviors, yet no RCTs to date have compared SSRIs with other forms of treatment (e.g., CBT) in an exclusively hoarding disorder sample. In BDD, although larger effect sizes and more consistently positive findings are reported in studies of CBT (usually ERP) as compared to those evaluating SSRIs, there are no direct-comparison RCTs (Ipser et al. 2009, Williams et al. 2006). Similarly, data from RCTs for skin picking disorder indicate more consistency in response to behavioral interventions (e.g., habit reversal training) than to SSRIs (e.g., Grant et al. 2012), although there are as yet no direct comparison studies. In trichotillomania, Bloch et al.'s (2007) meta-analysis revealed that habit reversal training had a larger effect size than did SSRIs. Thus, research suggests that SSRIs are not the most effective treatment for any of the OCRDs.

Rather, the empirical evidence suggests that psychological interventions (i.e., behavioral and cognitive-behavioral) are at least as effective as the SSRIs across the OCRDs. A noteworthy aspect of cognitive and behavioral interventions often overlooked by OCRD proponents is that they are derived from specific models of psychopathology (not necessarily etiology) that have a sound empirical basis. The use of ERP for OCD and BDD, for example, is guided by a literature of experimental research demonstrating that these conditions are characterized by irrational (obsessional) fears that are maintained by avoidance, compulsive rituals, and other neutralizing responses that reduce fear in the short term but impede longer-term fear extinction (e.g., Rachman & Hodgson 1980, Veale & Riley 2001). This is in contrast to antidepressant medications, which were discovered serendipitously and gave rise to general (post hoc) theories about serotonin primarily on the basis of treatment response (Whitaker 2011). Moreover, as we have also discussed, chemical imbalance models have never been well articulated and are not well supported by other types of (nontreatment) research designs. Thus, whereas these limitations render pharmacotherapy response virtually useless in helping to understand how mental disorders can be classified, response to cognitive and behavioral interventions that target specific and empirically established processes has a better chance of identifying natural and useful boundaries and classes of disorders. It is not clear why this was overlooked by the architects of the OCRD chapter in DSM-5.

Accordingly, it is worth considering CBT as a litmus test of the OCRD approach and evaluating the first two assumptions discussed above with respect to these interventions. Regarding the first parameter of preferential response, whereas OCD and BDD respond preferentially to ERP, as discussed previously (Romanelli et al. 2014, Williams et al. 2006), ERP is not used in the treatment of other OCRDs such as trichotillomania and skin picking because these problems do not involve obsessional fears and are not maintained by avoidance and rituals. Instead, these impulse control problems are addressed using habit reversal training, which involves three types of techniques: awareness training (identifying high-risk situations), development of a competing response until the urge to pull or pick passes (e.g., wearing gloves, holding an object), and stimulus control

(eliminating cues for pulling and picking) (Azrin & Nunn 1973, Odlaug & Grant 2012). CBT for hoarding involves a mix of some exposure-based techniques, some strategies similar to habit reversal, and some cognitive therapy strategies to help patients modify exaggerated beliefs about their own possessions (e.g., “I couldn’t live without this”) (Frost & Tolin 2008).

With respect to the second assumption regarding specificity, both exposure-based ERP and habit reversal training are used successfully in the treatment of disorders outside the OCRD classification. On one hand, numerous RCTs indicate that exposure therapy is the most effective intervention in CBT for anxiety disorders, all of which involve some form of irrational fear that is maintained by avoidance behavior or safety-seeking strategies that are functionally similar to the compulsive rituals and neutralizing behaviors in OCD (e.g., Abramowitz et al. 2012). Numerous RCTs also indicate that habit reversal training, on the other hand, is effective in the treatment of tic disorders along with other undesirable habits, such as thumb sucking and nose picking, which are maintained by different processes than OCD, BDD, and the anxiety disorders (e.g., Azrin & Nunn 1973, Piacentini & Chang 2006).

From the standpoint of clinical practice, treatment response is probably the most important gauge of the validity of the OCRD class of disorders. Not only is successful treatment (and prevention) the ultimate goal of identification, classification, and research on potential etiological factors, but there are no reliable or valid biological or laboratory tests for OCD or the other OCRDs (Hyman 2007). Even if such tests were available, how feasible would it be for most practicing clinicians to conduct examinations of genetics, brain circuitry, or neurotransmitter functioning to decide on a treatment regimen, considering costs and insurance reimbursement practices? Taken together, the treatment response data on pharmacological and psychological interventions have two broad implications for the OCRD classification. First, they suggest that although OCD and BDD overlap and could be considered OCRDs, hoarding, trichotillomania, and skin picking disorders would not fit into the same category. Second, they suggest that OCD and BDD bear substantial overlaps with the other anxiety disorders.

CONCLUSIONS: WHAT IS OBSESSIVE-COMPULSIVE DISORDER, AND WHERE DOES IT BELONG IN THE DSM?

We have critically evaluated the basis for DSM-5’s repositioning of OCD from the anxiety disorders into a new category of OCRDs that includes BDD, trichotillomania, hoarding disorder, and skin picking disorder. With the exception of BDD, which appears to have substantial phenomenological and treatment response overlaps with OCD, the pertinent empirical literature largely suggests that this marked conceptual and nosological shift lacks scientific merit. The case for the OCRD classification is undermined by the lack of a fine-grained conceptualization of OCD and OCRDs, reliance on high base rates of overlapping features with poor sensitivity and specificity (e.g., comorbidity, response to SRI medications), misinterpretation of data from brain imaging and treatment outcome research, and omission of data that largely fail to support the diagnostic shift (e.g., differential response to ERP, comorbidity data). Although we would never espouse majority opinion as a substitute for scientific data, it is worth noting that many expert clinicians (Mataix-Cols et al. 2007) and even prominent psychiatric researchers (Hyman 2007) unmistakably recognize that the basis for the OCRD chapter in DSM-5 is dubious.

The DSM classification system, which focuses on the superficial form of symptoms as opposed to the psychological processes involved in these symptoms, has led to a cursory conceptualization of OCD as a problem characterized simply by repetitive thoughts and behavioral patterns. This has given rise to the illusion that OCD overlaps with other disorders that involve repetitive thinking or behavior. Attempts to incorporate neuroscience into this diagnostic system—although vastly

premature—would move this focus further away from understanding psychological processes and closer to reducing problems like OCD to putative dysfunctions in brain structure and functioning associated with repetitive thinking and behavior. We argue here that the essence of OCD is not to be found in the repetitive nature of obsessions and compulsions, but rather in functional aspects of these signs and symptoms and the relationships between them.

The evidence is compelling that people with OCD experience thoughts that they misperceive as highly significant and foreboding of danger and then engage in attempts to reduce the chances of danger or to control the thought itself with tactics such as compulsive rituals, avoidance, or other neutralizing strategies. These strategies become habitual because they often reduce obsessional fear immediately, yet they maintain the problem in the long run by interfering with the natural extinction of obsessional fear. We understand that for those who focus exclusively on symptom form (as in the DSM), and for those invested in a neurobiological explanation for such symptoms, the illusion that OCD is a disorder of behavioral inhibition would be very seductive. Yet research does not support this position. It does, however, show that: (a) Obsessional thoughts and anxiety arise largely from mistaken perceptions of normal cognitive intrusions (the distal etiology of which is unclear); (b) Compulsive rituals are deliberate acts; and (c) The processes of classical conditioning and negative reinforcement maintain obsessional fears and compulsive urges (Abramowitz et al. 2009).

These key phenomenological processes are internally valid and accessible via proper assessment. They are also the same processes involved in the maintenance of anxiety disorders such as social anxiety disorder, panic disorder, agoraphobia, and PTSD (Abramowitz et al. 2012, Barlow 2004). Also maintained in this fashion is BDD, which traditionally has been categorized in DSM as a somatoform disorder because the content of its obsessions concerns the body (another example of DSM's superficial focus). In all of these conditions there is (a) the perception that some unlikely feared outcome will occur and be catastrophic and (b) the use of maladaptive strategies to deal with the perceived threat. Although the focus of the fear and maladaptive safety behaviors vary across these problems (e.g., in OCD it is intrusive thoughts, symmetry, or contamination; in BDD it is the concern with physical appearance; in social phobia it is social situations; in panic disorder it is arousal-related body sensations; etc.), the key psychological processes overlap. Each anxiety disorder involves overestimates of threat and subsequent responses that prevent realization that the fear is groundless.

Moreover, treatment based on this conceptualization, which aims to extinguish irrational fear using exposure to conditioned fear stimuli in the absence of safety maneuvers and cues, is highly effective for OCD, BDD, and other anxiety disorders. Trichotillomania, skin picking, and hoarding disorder, however, have demonstrated mixed responses to exposure-based treatments. Instead, a first-line treatment for hair pulling and skin picking is habit reversal training (which includes techniques such as eliminating cues of picking and pulling as opposed to systematically confronting them as is done in exposure). Similarly, strategies similar to habit reversal as well as cognitive therapy techniques for challenging exaggerated beliefs about possessions supplement exposure-based techniques for hoarding. Grouping all of these problems in the same diagnostic class because they are considered to be OCD-related, however, gives the clinician the false impression that all OCRDs should be conceptualized and treated in a similar fashion. Thus, the OCRD concept and its superficial focus on form of symptoms rather than their functional relationship may ultimately lead patients away from receiving evidence-based treatment of their problem.

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LITERATURE CITED

- Abramowitz JS, Deacon BJ, Olatunji BO, Wheaton MG, Berman NC, et al. 2010. Assessment of obsessive-compulsive symptom dimensions: development and evaluation of the Dimensional Obsessive-Compulsive Scale. *Psychol. Assess.* 22(1):180–98
- Abramowitz JS, Deacon BJ, Whiteside SPH. 2012. *Exposure Therapy for Anxiety: Principles and Practice*. New York: Guilford
- Abramowitz JS, Franklin ME, Schwartz SA, Furr JM. 2003. Symptom presentation and outcome of cognitive-behavioral therapy for obsessive-compulsive disorder. *J. Consult. Clin. Psychol.* 71(6):1049–57
- Abramowitz JS, Taylor S, McKay D. 2009. Obsessive-compulsive disorder. *Lancet* 374(9688):491–99
- Abramowitz JS, Wheaton MG, Storch EA. 2008. The status of hoarding as a symptom of obsessive-compulsive disorder. *Behav. Res. Ther.* 46(9):1026–33
- Am. Psychiatr. Assoc. 2013. *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC: Am. Psychiatr. Publ. 5th ed.
- Anholt GE, Aderka IM, van Balkom AJLM, Smit JH, Schruers K, et al. 2014. Age of onset in obsessive-compulsive disorder: admixture analysis with a large sample. *Psychol. Med.* 44(1):185–94
- Antony MM, Downie F, Swinson RP. 1998. Diagnostic issues and epidemiology in obsessive-compulsive disorder. In *Obsessive-Compulsive Disorder: Theory, Research, and Treatment*, ed. RP Swinson, MM Antony, S Rachman, MA Richter, pp. 3–32. New York: Guilford
- Arbab M, Farnia V, Balighi K, Mohammadi MR, Nejati-Safa AA, et al. 2008. Efficacy of citalopram in treatment of pathological skin picking, a randomized double blind placebo controlled trial. *Acta Med. Iran.* 46(5):367–72
- Arnold LM, Auchenbach MB, McElroy SL. 2001. Psychogenic excoriation: clinical features, proposed diagnostic criteria, epidemiology and approaches to treatment. *CNS Drugs* 15(5):351–59
- Azrin NH, Nunn RG. 1973. Habit-reversal: a method of eliminating nervous habits and tics. *Behav. Res. Ther.* 11(4):619–28
- Barlow DH. 2004. *Anxiety and Its Disorders: The Nature and Treatment of Anxiety and Panic*. New York: Guilford
- Barr LC, Goodman WK, Price LH. 1993. The serotonin hypothesis of obsessive compulsive disorder. *Int. Clin. Psychopharmacol.* 8(Suppl. 2):79–82
- Bennett CM, Miller MB, Wolford GL. 2009. Neural correlates of interspecies perspective taking in the post-mortem Atlantic Salmon: an argument for multiple comparisons correction. *NeuroImage* 47(Suppl. 1):S125
- Bienvenu OJ, Samuels JF, Riddle MA, Hoehn-Saric R, Liang KY, et al. 2000. The relationship of obsessive-compulsive disorder to possible spectrum disorders: results from a family study. *Biol. Psychiatry* 48(4):287–93
- Bjornsson AS, Didie ER, Grant JE, Menard W, Stalker E, Phillips KA. 2013. Age at onset and clinical correlates in body dysmorphic disorder. *Compr. Psychiatry* 54(7):893–903
- Bloch MH, Bartley CA, Zipperer L, Jakubowski E, Landeros-Weisenberger A, et al. 2014. Meta-analysis: hoarding symptoms associated with poor treatment outcome in obsessive-compulsive disorder. *Mol. Psychiatry* 19(9):1025–30
- Bloch MH, Landeros-Weisenberger A, Dombrowski P, Kelmendi B, Wegner R, et al. 2007. Systematic review: pharmacological and behavioral treatment for trichotillomania. *Biol. Psychiatry* 62(8):839–46
- Buchanan BG, Rossell SL, Maller JJ, Toh WL, Brennan S, Castle DJ. 2013. Brain connectivity in body dysmorphic disorder compared with controls: a diffusion tensor imaging study. *Psychol. Med.* 43(12):2513–21
- Chamberlain SR, Menzies LA, Fineberg NA, Del Campo N, Suckling J, et al. 2008. Grey matter abnormalities in trichotillomania: morphometric magnetic resonance imaging study. *Br. J. Psychiatry* 193(3):216–21
- Christenson GA, Ristvedt SL, Mackenzie TB. 1993. Identification of trichotillomania cue profiles. *Behav. Res. Ther.* 31(3):315–20
- Coles ME, Frost RO, Heimberg RG, Steketee GS. 2003. Hoarding behaviors in a large college sample. *Behav. Res. Ther.* 41(2):179–94
- Coles ME, Schofield CA, Pietrefesa AS. 2006. Behavioral inhibition and obsessive-compulsive disorder. *J. Anxiety Disord.* 20(8):1118–32

- Deacon BJ, Abramowitz JS. 2005. The Yale-Brown Obsessive Compulsive Scale: factor analysis, construct validity, and suggestions for refinement. *J. Anxiety Disord.* 19(5):573–85
- Denys D, Fineberg N, Carey PC, Stein DJ. 2008. Reply. *Biol. Psychiatry* 63(1):e7
- Denys D, van der Wee N, Janssen J, De Geus F, Westenberg HGM. 2004. Low level of dopaminergic D₂ receptor binding in obsessive-compulsive disorder. *Biol. Psychiatry* 55(10):1041–45
- Denys D, Van Nieuwerburgh F, Deforce D, Westenberg H. 2006. Association between the dopamine D₂ receptor TaqI A2 allele and low activity COMT allele with obsessive-compulsive disorder in males. *Eur. Neuropsychopharmacol.* 16(6):446–50
- Eddy KT, Dutra L, Bradley R, Westen D. 2004. A multidimensional meta-analysis of psychotherapy and pharmacotherapy for obsessive-compulsive disorder. *Clin. Psychol. Rev.* 24(8):1011–30
- Fineberg NA, Saxena S, Zohar J, Craig KJ. 2011. Obsessive-compulsive disorder: boundary issues. In *Obsessive-Compulsive Spectrum Disorders: Refining the Research Agenda for DSM-V*, ed. E Hollander, J Zohar, PJ Sirovatka, DA Regier, pp. 1–32. Washington, DC: Am. Psychiatr. Assoc.
- Flessner CA, Lochner C, Stein DJ, Woods DW, Franklin ME, Keuthen NJ. 2010. Age of onset of trichotillomania symptoms: investigating clinical correlates. *J. Nerv. Ment. Dis.* 198(12):896–900
- Foa EB, Kozak MJ, Goodman WK, Hollander E, Jenike MA, Rasmussen SA. 1995. DSM-IV field trial: obsessive-compulsive disorder. *Am. J. Psychiatry* 152(1):90–96
- Fournier JC, DeRubeis RJ, Hollon SD, Dimidjian S, Amsterdam JD, et al. 2010. Antidepressant drug effects and depression severity: a patient-level meta-analysis. *J. Am. Med. Assoc.* 303(1):47–53
- Frankenburg FR. 1984. Hoarding in anorexia nervosa. *Br. J. Med. Psychol.* 57(1):57–60
- Frost RO, Tolin DF. 2008. Compulsive hoarding. In *Clinical Handbook of Obsessive-Compulsive Disorder and Related Problems*, ed. JS Abramowitz, D McKay, S Taylor, pp. 76–94. Baltimore, MD: Johns Hopkins Univ. Press
- Grant JE, Odlaug BL, Chamberlain SR, Keuthen NJ, Lochner C, Stein DJ. 2012. Skin picking disorder. *Am. J. Psychiatry* 169(11):1143–49
- Grant JE, Odlaug BL, Hampshire A, Schreiber LRN, Chamberlain SR. 2013. White matter abnormalities in skin picking disorder: a diffusion tensor imaging study. *Neuropsychopharmacology* 38(5):763–69
- Grant JE, Potenza MN. 2004. Impulse control disorders: clinical characteristics and pharmacological management. *Ann. Clin. Psychiatry* 16(1):27–34
- Greist JH, Jefferson JW, Kobak KA, Katzelnick DJ, Serlin RC. 1995. Efficacy and tolerability of serotonin transport inhibitors in obsessive-compulsive disorder: a meta-analysis. *Arch. Gen. Psychiatry* 52(1):53–60
- Grisham JR, Frost RO, Steketee G, Kim H-J, Hood S. 2006. Age of onset of compulsive hoarding. *J. Anxiety Disord.* 20(5):675–86
- Hedges DW, Brown BL, Shwalb DA, Godfrey K, Larcher AM. 2007. The efficacy of selective serotonin reuptake inhibitors in adult social anxiety disorder: a meta-analysis of double-blind, placebo-controlled trials. *J. Psychopharmacol.* 21(1):102–11
- Hollander E, Evers M. 2004. Review of obsessive-compulsive spectrum disorders: What do we know? Where are we going? *Clin. Neuropsychiatry* 1(1):32–51
- Hollander E, Friedberg JP, Wasserman S, Yeh C-C, Iyengar R. 2005. The case for the OCD spectrum. In *Concepts and Controversies in Obsessive-Compulsive Disorder*, ed. JS Abramowitz, AC Houts, pp. 95–118. New York: Springer
- Hollander E, Kim S, Khanna S, Pallanti S. 2007. Obsessive-compulsive disorder and obsessive-compulsive spectrum disorders: diagnostic and dimensional issues. *CNS Spectr.* 12(2 Suppl. 3):5–13
- Hollander E, Kwon JH, Stein DJ, Broatch J. 1996. Obsessive-compulsive and spectrum disorders: overview and quality of life issues. *J. Clin. Psychiatry* 57:3–6
- Hyman SE. 2007. Can neuroscience be integrated into the DSM-V? *Nat. Rev. Neurosci.* 8(9):725–32
- Insel TR. 2009. Translating scientific opportunity into public health impact: a strategic plan for research on mental illness. *Arch. Gen. Psychiatry* 66(2):128–33
- Insel TR, Mueller EA, Alterman I, Linnoila M, Murphy DL. 1985. Obsessive-compulsive disorder and serotonin: Is there a connection? *Biol. Psychiatry* 20(11):1174–88
- Ipsen JC, Sander C, Stein DJ. 2009. Pharmacotherapy and psychotherapy for body dysmorphic disorder. *Cochrane Database Syst. Rev.* 2009:CD005332

- Jaisoorya TS, Janardhan Reddy YC, Srinath S. 2003. The relationship of obsessive-compulsive disorder to putative spectrum disorders: results from an Indian study. *Compr. Psychiatry* 44(4):317–23
- Jenike MA, Breiter HC, Baer L, Kennedy DN, Savage CR, et al. 1996. Cerebral structural abnormalities in obsessive-compulsive disorder: a quantitative morphometric magnetic resonance imaging study. *Arch. Gen. Psychiatry* 53(7):625–32
- Keuthen NJ, Deckersbach T, Wilhelm S, Hale E, Fraim C, et al. 2000. Repetitive skin-picking in a student population and comparison with a sample of self-injurious skin-pickers. *Psychosomatics* 41(3):210–15
- Kirsch I. 2011. *The Emperor's New Drugs: Exploding the Antidepressant Myth*. New York: Basic Books
- Koo M-S, Kim E-J, Roh D, Kim C-H. 2010. Role of dopamine in the pathophysiology and treatment of obsessive-compulsive disorder. *Expert Rev. Neurother.* 10(2):275–90
- Koran LM, Hanna GL, Hollander E, Nestadt G, Simpson HB (Am. Psychiatr. Assoc.). 2007. Practice guideline for the treatment of patients with obsessive-compulsive disorder. *Am. J. Psychiatry* 164(7 Suppl.):5–53
- Kramer MD, Polusny MA, Arbisi PA, Krueger RF. 2014. Comorbidity of PTSD and SUDs: toward an etiologic understanding. In *Trauma and Substance Abuse: Causes, Consequences, and Treatment of Comorbid Disorders*, ed. P Ouimette, JP Read, pp. 53–75. Washington, DC: Am. Psychol. Assoc. 2nd ed.
- Lee H-J, Kwon S-M. 2003. Two different types of obsession: autogenous obsessions and reactive obsessions. *Behav. Res. Ther.* 41(1):11–29
- Lee H-J, Kwon S-M, Kwon JS, Telch MJ. 2005. Testing the autogenous-reactive model of obsessions. *Depress. Anxiety* 21(3):118–29
- Lee H-J, Telch MJ. 2010. Differences in latent inhibition as a function of the autogenous-reactive OCD subtype. *Behav. Res. Ther.* 48(7):571–79
- Lochner C, Simeon D, Niehaus DJH, Stein DJ. 2002. Trichotillomania and skin-picking: a phenomenological comparison. *Depress. Anxiety* 15(2):83–86
- 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(3):353–61
- Lovato L, Ferrão YA, Stein DJ, Shavitt RG, Fontenelle LF, et al. 2012. Skin picking and trichotillomania in adults with obsessive-compulsive disorder. *Compr. Psychiatry* 53(5):562–68
- Luchins DJ, Goldman MB, Lieb M, Hanrahan P. 1992. Repetitive behaviors in chronically institutionalized schizophrenic patients. *Schizophr. Res.* 8(2):119–23
- 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(9):1313–14
- Mataix-Cols D, Pertusa A, Snowden J. 2011. Neuropsychological and neural correlates of hoarding: a practice-friendly review. *J. Clin. Psychol.* 67(5):467–76
- McKay D, Abramowitz JS, Calamari JE, Kyrios M, Radomsky A, et al. 2004. A critical evaluation of obsessive-compulsive disorder subtypes: symptoms versus mechanisms. *Clin. Psychol. Rev.* 24(3):283–313
- Miller GA, Keller J. 2000. Psychology and neuroscience making peace. *Curr. Dir. Psychol. Sci.* 9(6):212–15
- Nestadt G, Samuels J, Riddle MA, Liang KY, Bienvenu OJ, et al. 2001. The relationship between obsessive-compulsive disorder and anxiety and affective disorders: results from the Johns Hopkins OCD Family Study. *Psychol. Med.* 31(3):481–87
- Neziroglu F, Rabinowitz D, Breytman A, Jacofsky M. 2008. Skin picking phenomenology and severity comparison. *Prim. Care Companion J. Clin. Psychiatry* 10(4):306–12
- NICE (Nat. Inst. Health Care Excell.). 2005. *Obsessive-Compulsive Disorder: Core Interventions in the Treatment of Obsessive-Compulsive Disorder and Body Dysmorphic Disorder*. Manchester, UK: NICE. <http://nice.org.uk/guidance/cg31>
- O'Sullivan RL, Rauch SL, Breiter HC, Grachev ID, Baer L, et al. 1997. Reduced basal ganglia volumes in trichotillomania measured via morphometric magnetic resonance imaging. *Biol. Psychiatry* 42(1):39–45
- Odlaug BL, Grant JE. 2012. Pathological skin picking. In *Trichotillomania, Skin Picking, and Other Body-Focused Repetitive Behaviors*, ed. JE Grant, DJ Stein, DW Woods, NJ Keuthen, pp. 21–41. Arlington, VA: Am. Psychiatr. Publ.

- Phillips KA, Albertini RS, Rasmussen SA. 2002. A randomized placebo-controlled trial of fluoxetine in body dysmorphic disorder. *Arch. Gen. Psychiatry* 59(4):381–88
- Phillips KA, Wilhelm S, Koran LM, Didie ER, Fallon BA, et al. 2010. Body dysmorphic disorder: some key issues for DSM-V. *Depress. Anxiety* 27(6):573–91
- Piacentini JC, Chang SW. 2006. Behavioral treatments for tic suppression: habit reversal training. *Adv. Neurol.* 99:227–33
- Rachman S, de Silva P, Roper G. 1976. The spontaneous decay of compulsive urges. *Behav. Res. Ther.* 14(6):445–53
- Rachman S, Elliott CM, Shafran R, Radomsky AS. 2009. Separating hoarding from OCD. *Behav. Res. Ther.* 47(6):520–22
- Rachman S, Hodgson RJ. 1980. *Obsessions and Compulsions*. Englewood Cliffs, NJ: Prentice-Hall
- Romanelli RJ, Wu FM, Gamba R, Mojtatabi R, Segal JB. 2014. Behavioral therapy and serotonin reuptake inhibitor pharmacotherapy in the treatment of obsessive-compulsive disorder: a systematic review and meta-analysis of head-to-head randomized controlled trials. *Depress. Anxiety* 31(8):641–52
- Rosenbaum JF, Biederman J, Bolduc-Murphy EA, Faraone SV, Chaloff J, et al. 1993. Behavioral inhibition in childhood: a risk factor for anxiety disorders. *Harv. Rev. Psychiatry* 1(1):2–16
- Roskies AL. 2007. Are neuroimages like photographs of the brain? *Philos. Sci.* 74(5):860–72
- Schatzberg AF, Nemeroff CB. 2013. *Essentials of Clinical Psychopharmacology*. Arlington, VA: Am. Psychiatr. Publ.
- Schreiber L, Odlaug BL, Grant JE. 2011. Impulse control disorders: updated review of clinical characteristics and pharmacological management. *Front. Psychiatry* 2:1
- Simeon D, Stein DJ, Gross S, Islam N, Schmeidler J, Hollander E. 1997. A double-blind trial of fluoxetine in pathologic skin picking. *J. Clin. Psychiatry* 58(8):341–47
- Snorrason I, Smári J, Olafsson RP. 2010. Emotion regulation in pathological skin picking: findings from a non-treatment seeking sample. *J. Behav. Ther. Exp. Psychiatry* 41(3):238–45
- Stanley MA, Swann AC, Bowers TC, Davis ML, Taylor DJ. 1992. A comparison of clinical features in trichotillomania and obsessive-compulsive disorder. *Behav. Res. Ther.* 30(1):39–44
- Stein DJ, Lochner C. 2006. Obsessive-compulsive spectrum disorders: a multidimensional approach. *Psychiatr. Clin. North Am.* 29(2):343–51
- Stein DJ, Simeon D, Cohen LJ, Hollander E. 1995. Trichotillomania and obsessive-compulsive disorder. *J. Clin. Psychiatry* 56(Suppl. 4):28–34
- Summerfeldt LJ, Hood K, Antony MM, Richter MA, Swinson RP. 2004. Impulsivity in obsessive-compulsive disorder: comparisons with other anxiety disorders and within tic-related subgroups. *Personal. Individ. Differ.* 36(3):539–53
- Valenstein E. 2002. *Blaming the Brain: The Truth About Drugs and Mental Health*. New York: Simon & Schuster
- Veale D, Riley S. 2001. Mirror, mirror on the wall, who is the ugliest of them all? The psychopathology of mirror gazing in body dysmorphic disorder. *Behav. Res. Ther.* 39(12):1381–93
- Vul E, Harris C, Winkielman P, Pashler H. 2009. Puzzlingly high correlations in fMRI studies of emotion, personality, and social cognition. *Perspect. Psychol. Sci.* 4(3):274–90
- Wheaton MG, Abramowitz JS, Fabricant LE, Berman NC, Franklin JC. 2011. Is hoarding a symptom of obsessive-compulsive disorder? *Int. J. Cogn. Ther.* 4(3):225–38
- Wheaton MG, Timpano KR, LaSalle-Ricci VH, Murphy D. 2008. Characterizing the hoarding phenotype in individuals with OCD: associations with comorbidity, severity and gender. *J. Anxiety Disord.* 22(2):243–52
- Whitaker R. 2011. *Anatomy of an Epidemic: Magic Bullets, Psychiatric Drugs, and the Astonishing Rise of Mental Illness in America*. New York: Broadway Books
- Whiteside SP, Port JD, Abramowitz JS. 2004. A meta-analysis of functional neuroimaging in obsessive-compulsive disorder. *Psychiatry Res. Neuroimaging* 132(1):69–79
- Wilhelm S, Keuthen NJ, Deckersbach T, Engelhard IM, Forker AE, et al. 1999. Self-injurious skin picking: clinical characteristics and comorbidity. *J. Clin. Psychiatry* 60(7):454–59
- Williams J, Hadjistavropoulos T, Sharpe D. 2006. A meta-analysis of psychological and pharmacological treatments for body dysmorphic disorder. *Behav. Res. Ther.* 44(1):99–111

- Windheim K, Veale D, Anson M. 2011. Mirror gazing in body dysmorphic disorder and healthy controls: effects of duration of gazing. *Behav. Res. Ther.* 49(9):555–64
- Zohar J, Kennedy JL, Hollander E, Koran LM. 2004. Serotonin-1D hypothesis of obsessive-compulsive disorder: an update. *J. Clin. Psychiatry* 65(Suppl. 14):18–21
- Zurowski B, Kordon A, Wahl K, Hohagen F. 2008. Non-selective effects of selective serotonin reuptake inhibitors. *Biol. Psychiatry* 63(1):e5