WHAT IS OCD AND WHAT IS NOT?
Problems with the OCD Spectrum Concept

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A "spectrum" of disorders thought to be related to obsessive-compulsive disorder (OCD) and representing a wide range of psychiatric conditions has been identified. Inclusion of disorders in the proposed "OCD spectrum" appears to be based chiefly on the presence of repetitive thoughts or behaviors. Advocates of the OCD spectrum concept argue that each of these disorders results from similar neurobiological deficits, possesses similar associated features, and responds preferentially to antipsychotic medication and behavior therapy. In this article we critically review the arguments for a spectrum of OCD-related disorders. Despite superficial topographical similarities (e.g., repetitive behaviors), behavioral analyses and research findings suggest that very few of the putative spectrum disorders are related to OCD. In addition, arguments that OCD spectrum disorders each respond preferentially to similar medications and behavior therapies are (a) unsupported by research data or clinical experience and (b) indicative of confusion concerning behavioral therapy procedures. We conclude that the OCD spectrum concept is not particularly useful, and may even undermine efforts to understand and treat OCD.

A general theme in biological taxonomy concerns the relative emphasis on lumping versus splitting. This distinction harks back to the folk observation that there are two kinds of people in the world: those who believe there are two kinds of people in the world and those who do not. Like biological taxonomy, psychiatric nosology has evidenced periods of both lumping and splitting. With its emphasis on psychoanalytic theory as an organizing framework, DSM–II lumped a number of conditions under the general concept of neurosis. With the advent of DSM–III and the ensuing revisions, we have witnessed a general trend of splitting within official psychiatric diagnoses over the past 20 years. For example, the DSM–II diagnosis of anxiety neurosis was abandoned in favor of several more specific diagnoses such as panic disorder, specific phobia, and generalized anxiety disorder. Numerous other examples of the splitting trend are noted when one compares DSM–II with DSM–IV.

Whether one prefers lumping to splitting may be a matter of mere personal taste, but at least some critics of the last three DSMs have argued that the splitting trend is problematic, with finer-grained distinctions bearing little evidence of organizing principles of classification. Therefore, when lumpers come along with empirically driven proposals to aggregate existing diagnoses into larger categories, it is worth attending to their proposals. For example, two recent factor analytic studies of symptom co-occurrence within the context of epidemiological studies, one in the United States and one in the Netherlands, have reported that one needs only three underlying dimensions to describe nine different DSM–III–R diagnoses. These rather remarkable findings suggest that at the symptom level, one needs only three broad constructs to account for the manner in which symptoms co-occur in large, population-wide samples. Such empirical lumping helps to explain the comorbidity of mood disorders and anxiety disorders, and also raises questions about the wisdom of continuing to conduct research as if, for example, generalized anxiety disorder is uniquely different from major depression. A less empirically based approach to lumping of disorders has been proposed by those who advocate for a spectrum of obsessive-compulsive disorders (OCD spectrum).

Advocates of the "OCD spectrum" concept have identified a number of psychiatric conditions that, because they appear to share some characteristics with OCD (e.g., repetitive behaviors), seem to belong to a
family of OCD-related disorders (Hollander & Wong, 1995a). The most salient feature linking the spectrum disorders is the presence of repetitive thoughts, behaviors, or both. These disorders are also hypothesized by advocates of the OCD spectrum to possess high comorbidity with OCD, common associated features (i.e., demographics), related neurobiological etiologies, and response to specific behavioral and pharmacological treatments (Hollander & Wong, 2000).

In their delineation of the OCD spectrum, Hollander and Wong (2000) included a wide range of disorders representing numerous forms of psychopathology. Among this list are impulse-control disorders, in which people repeat certain behaviors such as hair-pulling (trichotillomania), skin picking, nail-biting, gambling, shopping, and promiscuous sexual activity. A second cluster includes neurological disorders with involuntary motoric movements or tics, such as Tourette’s syndrome, Asperger’s syndrome, Sydenham’s chorea, and infantile autism. A third class includes disorders featuring preoccupation with one’s own body, such as body dysmorphic disorder, anorexia nervosa, and hypochondriasis.

Spectrum enthusiasts also conceptualize OCD along several dimensions. Along the compulsive-impulsive dimension lay compulsive, harm-avoidant rituals (i.e., washing to prevent illness) at one end, and such impulsive behaviors as repetitive gambling, nose picking, masturbation, and shopping at the other (Hollander, 1993a, 1993b). Hollander and Wong (2000) also proposed a motoric and obsessive dimension, with involuntary behaviors involving little or no obsessive thinking (e.g., motor tics) at one end and obsessive thinking involving little or no compulsive behavior at the other. Finally, the insight dimension includes delusional disorders with poor insight into the irrationality of obsessive thoughts and beliefs, versus conditions in which the senselessness of these cognitions is recognized.

Unfortunately, the OCD spectrum concept has many serious flaws. In the present article, we begin by highlighting overlooked phenomenological distinctions between OCD and proposed spectrum disorders. Indeed, research on important differences that clearly set OCD apart from these other conditions seems to have been largely ignored in favor of a superficial and largely arbitrary approach of grouping disorders based on the mere presence of repetitive thoughts or behaviors in the DSM criteria. We next discuss misinterpretations of neuroimaging research results that have led to conclusions that OCD and the spectrum disorders possess similar neurobiological etiologies. Indeed, there is currently little convincing evidence that OCD itself is caused by specific neurobiological factors. Third, we discuss problems with arguments that spectrum disorders have overlaps in associated features and are highly comorbid with OCD.

Fourth, we describe difficulties with assertions that these disorders all show a similar treatment response. Proposed spectrum disorders do not all respond preferentially to a specific form of pharmacotherapy; and similar claims about response to behavioral treatment reveal critical misunderstandings of the principles of behavior therapy. Fifth and finally, we discuss the differences between compulsivity and impulsivity, and the difficulties involved with placing these constructs on a single bipolar dimension. We assert that a careful phenomenological analysis of the proposed spectrum disorders, and a conscientious reading of the research, suggest that only a precious few of the more than 20 conditions that are proposed to be in the OCD spectrum may actually be related to OCD (see Table 1).

**YOU CAN’T JUDGE A BOOK . . .**

Early descriptions of obsessive-compulsive patients emphasized attempts to understand the phenomenology of their problem (Janet, 1903). Behavioral psychology propagated this tradition by highlighting the antecedents and consequences of obsessions and compulsions (e.g., Mower, 1960; Rachman & Hodgson, 1980). Recent theorists have identified cognitive biases that may also play a role in the development and maintenance of these symptoms (Obsessive-Compulsive Cognitions Working Group, 1997; Salkovskis, 1985, 1989). Hypotheses about OCD symptoms and their interrelationships were developed from clinical observations and subsequently tested in the laboratory (Roper & Rachman, 1975; Roper, Rachman, & Hodgson, 1973; for a review, see Rachman & Hodgson, 1980), adding to our understanding of the persistence and treatment of this problem. However, the precision afforded by this approach seems to have faded with the growing enthusiasm over diagnostic criteria such as those of the DSM, which emphasize disorders that are composed of lists of signs and symptoms. As a result of this shift, the conceptual understanding of individual signs and symptoms is sometimes overlooked in favor of a superficial observational approach that merely collects behaviors according to form or topography as opposed to function. Such an approach, inaugurated with the DSM-III operational criteria, made a good deal of sense in terms of resolving the major problem of DSM-II, namely, diagnostic unreliability. To be sure, one way to enhance reliability is to generate a set of specific
 TABLE 1. DISORDERS OF THE PROPOSED OCD SPECTRUM

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<td>Impulse control</td>
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<td>Sexual compulsions</td>
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<td>Impulsive Personality Disorders</td>
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<td>Antisocial personality disorder</td>
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What Is OCD?

OCD is an anxiety disorder involving two well-defined phenomena. *Obsessions* are thoughts, ideas, images, or impulses that are experienced as intrusive, repugnant, inappropriate, and seemingly bizarre (ego-dystonic), and that give rise to significant anxiety or fear (e.g., thoughts of poisoning family members by mistake). *Compulsions* are urges to repeat behavioral or mental acts (rituals) to reduce anxiety or distress associated with obsessions (e.g., excessive handwashing, checking, praying). Obsessions and compulsions are related in that compulsive rituals are designed to neutralize specific obsessional thoughts by reducing feelings of anxiety or uncertainty over whether a specific disastrous consequence will occur (e.g., that one will mistakenly poison his or her family). Indeed, numerous laboratory investigations and clinical observations have established that compulsive rituals are purposeful and lead to immediate, but short-term, reduction in fear and uncertainty (e.g., Roper & Rachman, 1975; but see Rachman & Hodgson, 1980, for a review). Because rituals bring about an escape from distress, they are more likely to be repeated when an obsessional fear recurs (i.e., negative reinforcement). Therefore, repetitive behavior observed in OCD (i.e., compulsive rituals) is essentially escape or avoidance behavior that is performed in response to clear antecedents, namely obsessional thoughts and anxiety.

To briefly illustrate this condition, consider Mr. G, who suffers from obsessional thoughts of making mistakes when completing paperwork. On numerous occasions while paying his telephone bills he has experienced the thought that perhaps he wrote the incorrect dollar amount on the check, or that he copied his invoice number incorrectly from the statement. Such a thought evokes subsequent thoughts and images of losing his telephone service, harassment from collection agencies, social embarrassment, and a general sense of imperfection. To reduce his uncertainty and distress, Mr. G rereads his checks and compares the dollar amount and invoice number several times to make certain it is correct. Feeling more confident, he places the check into its envelope, only to once
again entertain the nagging doubt that perhaps he did not catch an important mistake. This time, he tries to remind himself that there were no errors found during the last check. Indeed, he has never found any errors when rechecking. However, the lack of absolute certainty is unbearable for Mr. G and he reluctantly checks again and again, sometimes rewriting checks to be sure he is paying the correct amount, and wasting up to 2 hours each time he has to pay bills. To avoid this distress, Mr. G now asks his wife to pay the bills.

This case is prototypical of OCD. The key features are the unwanted obsessive thoughts that produce unrealistic worry and anxiety, and the ritualistic checking, which reduces anxiety. Without these components and their functional relationship, OCD is not present. There is also the profound intolerance for uncertainty and sense that certain thoughts are equivalent to reality (i.e., “thought-action fusion”). Yet, although such cognitive biases exist in the context of particular obsessional situations, they typically do not interfere with general executive or intellectual functioning. In fact, individuals with OCD generally show normal IQ scores and memory ability (e.g., Tolin et al., 2001). These findings are critically important in evaluating the OCD spectrum concept because the disorders subsumed under the OCD spectrum concept do not all share these components in the particular functional manner described above.

**Is Trichotillomania a Form of OCD?**

An excellent example of how an emphasis on lists of signs and symptoms can be misleading in understanding OCD is found in the common misconstrual of trichotillomania (TTM) as a form of OCD. Consider first the chief DSM–IV diagnostic criteria for TTM: (a) recurrent pulling of one’s hair resulting in noticeable hair loss; (b) increase in tension immediately before pulling, or when attempting to resist pulling; and (c) pleasure, gratification, or relief when pulling out the hair. If we employ this list of features alone in understanding patients with TTM, it appears to share some characteristics with OCD; namely, both involve repetitive behaviors. This is exactly the primary rationale for including TTM in the OCD spectrum (Stein, Simeon, Cohen, & Hollander, 1995). The problem is that although some behaviors in OCD and TTM might seem similar, the intrusive, anxiety-evoking obsessional thoughts that occur in OCD are not present in TTM. In explaining the inclusion of TTM as a form of OCD, however, Stein et al. minimized the importance of obsessions, writing that “differences in emphasis are, however, subtle” (p. 29).

By ignoring the importance of the anxiety-evoking nature of obsessional thoughts with this statement, Stein et al. (1995) disregarded a clear distinction between OCD and TTM. This is not a “subtle” difference, since it is obsessional fear and doubt that evoke the repetitive behavior (i.e., compulsive rituals) in OCD. Rituals in OCD are performed in response to specific obsessional thoughts and fears, and result in decreased anxiety (e.g., Rachman & Hodgson, 1980). In contrast, urges to pull hair in TTM are not precipitated by obsessional fears, but instead by feelings of general tension, depression, anger, boredom, frustration, indecision, or fatigue (Christensen, Ristvedt, & Mackenzie, 1993; Stanley & Mouton, 1996). Studies also suggest that hair pulling in TTM leads to pleasurable feelings, a phenomenon not observed with rituals in OCD (Stanley, Swann, Bowers, & Davis, 1992).

Thus, the approach to including spectrum disorders on the basis of superficial symptom presentation overlooks empirically demonstrated phenomenological differences between the repetitive behaviors in OCD and TTM. The following analogy illustrates the problematic reasoning in the OCD spectrum approach:

1. Vomiting is a symptom of bulimia nervosa.
2. Vomiting is a symptom of salmonella poisoning.
3. Therefore, bulimia nervosa and salmonella poisoning are related problems.

If one is heavily focused on sign and symptom lists and diagnostic criteria, it is easy to confuse some disorders that contain similar sign and symptom profiles (e.g., social phobia and avoidant personality disorder). However, overlooking the importance of obsessional thoughts in order to force clearly phenomenologically diverse problems under a more global OCD umbrella (e.g., Stein et al., 1995) reflects a critical misunderstanding of the features of OCD.

**Are Kleptomania, Pathological Gambling, Shopping, Binge Eating, and Compulsive Sex Forms of OCD?**

Although not as intensively studied as TTM, other impulse-control disorders show similar distinctions from OCD. That is, they do not involve anxiety-evoking obsessional phenomena as an essential component, and their repetitive behaviors have different functions than do compulsive rituals in OCD. Individuals with kleptomania report a “rush,” “thrill,” or “manic high” associated with their stealing, and those with compulsive buying describe a “high like taking cocaine” when purchasing products (McElroy et al., 1995). Similarly, patients with patho-
logical gambling report pleasure or gratification during and after gambling (Hollander & Wong, 1995b). The drive to perform these behaviors, and the emotional experiences associated with their completion, are qualitatively different from those present in OCD.

As a specific example, consider "compulsive" sexual behavior, referred to in DSM-IV as a paraphilia. Clinical observations suggest that individuals who engage in such pathological sexual activity do not do so in order to reduce obsessional anxiety. That is, logging onto Internet chat rooms, impulsive sex, and masturbation are not aimed at reducing uncertainty or the probability of feared outcomes. Instead, these sexual habits appear to be motivated by the physically and/or emotionally enjoyable states they produce. Certainly, the sexual activity itself is positively reinforced by its pleasurable physical consequences. This is different from repeated behaviors in OCD, which the individual feels driven to perform to reduce anxiety or fear. Similarly clear distinctions can be made between compulsive behaviors in OCD and those in pathological gambling, shopping, and stealing. All of those behaviors are obviously promoted by the tangible reinforcers they produce, and perhaps even by emotional excitement, but they would only barely be classified as behaviors motivated by the relief that comes from escaping some aversive condition. From a behavioral conceptual standpoint, however, OCD is primarily avoidance behavior that is maintained by negative reinforcement (escape), whereas these other behavior problems are maintained by positive reinforcement.

Another possible source of confusion in distinguishing between paraphilia and OCD is the presence of frequent thoughts about sex in both conditions. However, one must functionally examine such thoughts to observe the profound differences that distinguish sexual obsessions in OCD from other forms of sexual thoughts (i.e., in impulse control disorders). The following examples illustrate this distinction.

Consider a thought about having sexual intercourse with a child. For the obsessive-compulsive person the thought itself holds a high negative valence. It is egodystonic, meaning that the individual perceives the thought as inconsistent with his worldview and wishes not to engage in such behavior, not even the behavior of thinking the thought. In response, a person with OCD may take action to avoid situations that evoke the thought (e.g., playgrounds). He may also attempt to dismiss the thought or reduce the chances of acting on it, i.e., via compulsive rituals such as mentally "cancelling" the thought, or "confessing" it to others. The problem in OCD is the inflated sense of responsibility associated with such a thought, which leads to pathological fear and preoccupation (obsessions).

In contrast, for the person with paraphilia (pedophilia), the sexual thought itself is not experienced as distressing. In fact, it may be intentionally conjured up on a repeated basis because it is associated with desirable feelings of sexual excitement or release. Such thoughts may also become cued by various environmental stimuli (e.g., pornography, computers). In particularly impulsive individuals, the sexually arousing thoughts often lead to irresistible urges to engage in inappropriate sexual activity.

Thus, the example of compulsive sexual behavior illustrates the problem with relying solely on superficial observation in trying to understand behavior. Unfortunately, the words "obsession" and "compulsion" have lost their meaning in our society and the tendency has been to label any repetitive thought about sex as an "obsession" and any repetitive behavior a "compulsion." It is thus not difficult to see how such a diverse range of phenomena has come to be mistaken as symptoms of OCD. However, when the underlying functional aspects of these features are carefully considered, clear differences between OCD features and those of impulse-control disorders become apparent.

Is Tourette's Syndrome a Form of OCD?

Tourette's syndrome (TS) involves repetitive motor or vocal tics, which are defined as sudden rapid, recurrent, nonrhythmic, and stereotyped motor movements or vocalizations (APA, 1994). Given the presence of such repetitive phenomena, it is apparent why TS is included among the proposed OCD spectrum disorders (Hollander & Wong, 1995a, 2000). Nevertheless, researchers have observed important functional differences between tics in TS and compulsive rituals in OCD that clearly differentiate these two phenomena. Primarily, whereas compulsive rituals are deliberate and serve as an escape from affective distress, tics are spontaneous (sudden) and performed to reduce sensory discomfort or tension—not to neutralize obsessional fear or anxiety (Shapiro & Shapiro, 1992). As is the case with impulse-control disorders above, a relationship between TS and OCD has been assumed to exist on the basis of superficial description of the behavior as "repetitive" or "compulsive," as opposed to an analysis of the function of behavior.

Miguel et al. (1995) conducted a well-designed study in which 12 adults with TS were compared with 15 adults with OCD. The focus of this investigation was on so-called intentional repetitive behaviors (IRBs; p. 247), which at least superficially share characteristics with
both compulsions and complex motor tics. These authors found that despite their shared repetitiveness, IRBs in OCD and TS were clearly distinguished from each other, but mainly by their functional differences. As would be expected, IRBs reported by OCD patients (i.e., compulsions) were preceded by cognitive phenomena (i.e., obsessional fears), whereas IRBs in TS patients were preceded by such sensory phenomena as inner tension or urges to release energy. Indeed, all 12 of the OCD patients reported fears of disasters that might occur if the IRB was not performed; yet these kinds of fears were present in only 2 of the TS patients. Similarly, whereas all 12 of the TS patients reported the presence of sensory phenomena, none of the OCD patients reported these kinds of sensations. Finally, 13 of the 15 OCD patients reported mounting autonomic anxiety symptoms when they were prevented from performing IRBs, whereas none of the TS patients reported such symptoms.

These results highlight the critical differences between compulsions in OCD and tics in TS: antecedents of a decidedly different quality evoke each phenomenon. Tics, which often cannot be resisted, are evoked by sensory experiences; compulsive rituals, which can be resisted with effort, are evoked by cognitive or affective distress (e.g., fear or uncertainty). It is likely that the two types of antecedents have very different causes, neurobiologically or otherwise (Zinner, 2000). The case of TS further illustrates the problems with relying solely on the presence of repetitive behavior for “lumping” psychiatric disorders. TS and other neurological problems such as Asperger’s syndrome, Sydenham’s chorea, Huntington’s chorea, infantile autism, and epilepsy are very dissimilar from OCD and respond to dissimilar forms of treatment. Although they may share some superficial similarities—and there are clearly individuals in which these disorders co-occur—a careful functional analysis allows one to gain an appreciation of the differences between the symptoms of these two disorders. These neurological disorders are not forms of OCD.

**Is Hypochondriasis a Form of OCD?**

Hypochondriasis (HC), classified as a somatoform disorder in DSM-IV, is defined as a preoccupation with fears of having a serious disease (e.g., cancer), which persists despite appropriate medical evaluation and frequent checking with friends or relatives, health references, or doctors. Individuals with HC often monitor the stability of their own physical symptoms and are thus prone to misinterpreting slight or unfamiliar irregularities as signs of serious illnesses. Previous authors have likened unremitting health anxiety that endures despite reassurance in HC to obsessional thoughts or fears in OCD (Fallon, Javitch, Hollander, & Liebowitz, 1991). Similarly, it has been pointed out that repetitive attempts to seek reassurance in HC are reminiscent of certain compulsive rituals in OCD (Fallon et al., 1991).

Although these features appear topographically similar to those found in OCD, classification as an “OCD-related” problem requires empirical study and consideration of whether such symptoms possess similar functions as those in OCD. Neziroglu, McKay, and Yaryura-Tobias (2000) found empirical support for a similarity between HC and OCD based on similarities in affective states and characteristics of obsessions. However, they also found differences: individuals with HC showed higher levels of overvalued ideation and panic-related cognitions (e.g., fear of bodily sensations) compared with individuals with OCD.

Fine-grained analyses of HC symptoms also suggest similarities. Beliefs about illness in HC seem to be associated with perceived threat and evocation of subjective anxiety (Warwick & Salkovskis, 1990). It is also clear that repetitive checking behavior is performed in response to such illness beliefs, and function as a means of acquiring certainty about health status in a way that reduces distress, at least in the short term. Thus, checking in HC serves as an escape from preoccupation with disease much as compulsive rituals (e.g., washing) serve as an escape from obsessional anxiety (e.g., concerning germs). Despite the fact that HC has been classified as a somatoform disorder, it appears to feature signs and symptoms that are comparable in both form and function with those in anxiety disorders, particularly OCD and panic. HC therefore seems to be a good candidate for inclusion in any group of disorders proposed to have similarities with OCD.

**Is Body Dysmorphic Disorder a Form of OCD?**

Another somatoform disorder with features similar to OCD is body dysmorphic disorder (BDD), which involves excessive preoccupation with an imagined defect in appearance (e.g., “my nose is too small for my face”). Such faulty beliefs of unsightliness occur in the absence of any noticeable physical defect, yet lead to significant distress or anxiety about how the person appears to others. On the basis of these characteristics, Neziroglu and Yaryura-Tobias (1993a) highlighted the functional similarity between BDD beliefs and obsessions in OCD. In efforts to circumvent anticipated embarrassment, individuals with BDD often avoid social situations or en-
gage in various behaviors aimed to reduce the visibility of their imagined defect (Phillips, McElroy, Keck, Pope, & Hudson, 1993). Excessive grooming, frequent inspection of the imagined defect in mirrors, and comparing one’s body with others’ are also often observed (Rosen, 1996). Such behavior bears functional similarities to compulsive rituals in OCD; namely, both are performed as a means of escape from anxiety or threat when no actual danger is present. McKay, Neziroglu, and Yaryura-Tobias (1997) have also provided empirical support for the hypothesis that BDD may be a variant of OCD. They found that although individuals with BDD seem to be singly obsessed with their imagined defect (whereas obsessional themes in OCD vary widely), they did not differ from individuals with OCD on measures of general anxiety and depression.

**Summary**

Decisions concerning the inclusion of disorders in the OCD spectrum have occurred primarily on the basis of apparent similarities between repetitive thought and behavior patterns in OCD and the other conditions (Hollander & Wong, 1995a, 2000). But this approach involves judging the book by its cover, and as we have repeatedly pointed out, this approach overlooks essential functional characteristics of the thoughts and behaviors in question. Indeed, once one recognizes the role of antecedents and consequences in the maintenance of repetitive behaviors and thoughts, profound differences in the various spectrum disorders are evident. These differences call into question the notion that all of these disorders can be conceptualized as different presentations of OCD. Only in the cases of HC and BDD do empirical studies or functional analysis (rather than simply the presence of repetitive thoughts and behaviors) support a relationship with OCD.

**The Appeal to Neurobiology**

Proposed neurobiological similarities have also been used as the basis for including disorders (e.g., TS, anorexia nervosa) in the OCD spectrum. Proponents of this view assert that OCD spectrum disorders are produced by shared neurobiological abnormalities, including a dysfunctional serotonin system and abnormally functioning brain regions such as the caudate nucleus and the orbital-frontal limbic region (Hollander & Wong, 2000). However, such conclusions are premature given the largely inconsistent research results and lack of convincing data. Moreover, data have been misrepresented as implicating neuroanatomical dysfunction as a cause of OCD when research designs do not warrant this conclusion. We discuss these faulty interpretations in the following sections.

**Serotonin Functioning**

The most consistent finding in the biological literature on OCD is that obsessive-compulsive symptoms respond specifically to pharmacotherapy by selective serotonin reuptake inhibitors (SSRIs; e.g., fluoxetine, sertraline), as opposed to antidepressants with other mechanisms of action (e.g., desipramine, imipramine). On the basis of these findings, some investigators have derived the “serotonin hypothesis,” which proposes that OCD is caused by abnormalities in the serotonergic system (Barr, Goodman, & Price, 1993). However, such a conclusion is unwarranted because specific models of etiology cannot be derived solely from knowledge of successful treatment response. This is a logical error known as *ex juvantibus* reasoning, or “reasoning backward from what helps,” which is a variation of the fallacy known as *post hoc ergo propter hoc*, or “after this, therefore, because of this.” Such reasoning is pandemic in the mental health field and has been used repeatedly over the past half century. The logical fallacy is clear if you consider the following example: “When I take aspirin, my headache goes away. Thus, the reason I get headaches is because my aspirin level is too low.”

Just as there may be many possible mechanisms by which aspirin makes headaches go away, there may be many possible mechanisms by which SSRIs decrease OCD symptoms. For example, because neurotransmitter systems do not work in isolation, serotonergic neurons in one area of the brain may have synaptic relationships with, say, dopaminergic neurons elsewhere in the brain. Therefore, increasing serotonin levels in one region (e.g., by administration of SSRIs) may effectively increase or decrease (depending on the relationship) dopamine levels elsewhere. From an epistemological standpoint, successful response to a treatment derived from a particular conceptual framework may in some instances provide clues to etiology; however, definitive conclusions regarding causes of disorders are generally not warranted on the basis of treat-

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1. Coincidentally, recent studies have found that cognitive-behavioral therapy by exposure and response prevention is more effective in reducing OCD signs and symptoms than are SSR1 medication (e.g., Kozak, Liebowitz, & Foa, 2000). Thus, the flawed line of reasoning used to derive the serotonin hypothesis actually leads to the conclusion that OCD must be caused by cognitive-behavioral factors.
ment response alone. As we describe below, this is a particular problem with treatment by SSRIs.

Ironically, one of the great scientific, as opposed to practical, problems with the SSRI drugs is that they seem to work for such a wide variety of disorders. The SSRIs appear to be largely universal “happy pills.” It is as though instead of finding the silver bullet, we have found the silver cluster bomb. There is not much chance of meaningful pharmacological dissection of disorders with the SSRIs, because they appear to improve so many conditions that they are unlikely to lead to any useful information about mechanisms that might be candidates for further etiological investigation. The fact that OCD responds to SSRI, but not to other types of antidepressant medicine, does not alone prove that serotonin is the culprit in OCD.

Serotonergic models of OCD could be supported by evidence from controlled studies demonstrating differences in serotonergic functioning between individuals with and without OCD. Over the last few decades a considerable amount of energy has been devoted to conducting numerous biological markers (e.g., Zohar, Mueller, Insel, Zohar-Kadouch, & Murphy, 1987) and pharmacological challenge (e.g., Goodman et al., 1995) studies designed to test hypotheses. Collectively, the findings from these investigations have been remarkably inconsistent (for a review, see Barr, Goodman, Price, & McDougle, 1992; Barr et al., 1993). Some have interpreted the highly discrepant results from this body of literature to support the serotonin hypothesis. However, researchers have not identified specific underlying abnormalities in serotonergic functioning that, if present at all, generate OCD symptoms. One possibility yet to be considered is that excessively high levels of serotonin are present in some areas of the brain, whereas excessively low levels are present in other areas. A further problem for the OCD spectrum idea is that virtually no controlled marker or challenge studies of the serotonin system have been conducted on other proposed spectrum disorders.

A consequence of this misinterpretation of serotonin data is that it has led to inadequate and misleading animal models of OCD. For example, Rapoport proposed canine acral lick dermatitis (ALD) as one such model because it involves repetitive behavior (excessive licking of the paws that leads to infection) and responds to SSRI medication. Problems with using ALD as an animal model of OCD are similar to the problems with including disorders such as kleptomania and compulsive gambling in the OCD spectrum. In both cases, a superficial depiction of OCD symptoms and misinterpretation of treatment response data lead to the presumption of a prevailing abnormality. OCD is an anxiety disorder that involves ritualistic behaviors performed to reduce anxiety and fear. Such a relationship has not yet been found in dogs with ALD. Indeed, there is a plausible reinforcement explanation for ALD wherein licking leads to irritation that the dog tries to relieve by continued licking. When one considers the functional relationship between obsessional fear and compulsive rituals, ALD is misleading as a model of OCD. Incidentally, a coherent animal model of OCD based on learning theory, which accounts for anxiety, avoidance, ritualistic behaviors, and their elimination via behavioral procedures (exposure and response prevention) does exist (Solomon, Kamin, & Wynne, 1953; Solomon & Wynne, 1953, 1954), although its description is beyond the scope of this discussion. For reasons that are unclear, this model has received little or no consideration by OCD spectrum advocates.

For certain, obsessive-compulsive behavior involves the serotonin system; yet the existing evidence does not suggest that OCD is caused by an abnormal serotonin system. One problem with lumping together a group of disorders, such as those in the OCD spectrum, on the basis of their relationship with serotonin is that serotonin is involved with many aspects of human behavior such as emotion, temperature regulation, memory, motor activity, pain, sleep, digestion, and hunger. Thus, the argument that OCD spectrum disorders are all related to serotonin function does not reveal anything specific about these conditions. In fact, by the same logic, sleeping and eating, for example, should also be included in the spectrum because they are both repetitive and involve the serotonin system. As already noted, a second problem with classifying OCD spectrum disorders in this fashion is that it ignores phenomenological aspects of behaviors that are critical to distinguishing OCD from other conditions (e.g., OCD vs. TS vs. sleeping and eating).

Neurophysiology

The results of brain imaging studies are also championed as supporting a neuropsychiatric cause of OCD (e.g., Rauch et al., 1994). Despite some inconsistencies, several studies have found higher blood flow and glucose meta-

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2. Solomon and colleagues demonstrated that dogs readily learned to avoid electrical shock by jumping into an adjacent “safe” space when given a signal that preceded the shock. Even when the shock was no longer imminent, the dogs continued to ritualistically jump to safety. The avoidance behavior (jumping) was negatively reinforced by the failure of shock to occur. Solomon and colleagues also demonstrated that the ritualistic jumping behavior could be extinguished if the dog was forced to remain in the feared space for long periods of time without the occurrence of shock (the principles of exposure and response prevention).
bolic rates in various brain regions among OCD patients compared with nonpatients. For example, Baxter et al. (1988) observed that OCD patients had elevated metabolic rates in the caudate and orbital gyri compared with non-OCD individuals. However, these studies do not employ the kind of methodology needed to address the question of whether abnormal brain mechanisms cause OCD symptoms—that is, they do not manipulate neurophysiologic functioning and measure its effects on behavior. Instead, these studies report observations of differences between people with and without OCD. Undoubtedly, these correlational studies yield interesting data; however, as with all correlational studies, there are problems of causal arrow ambiguity and potential third variables. Indeed, the observed increases in brain metabolic functioning in OCD may be an epiphenomenon, such as the result of heightened anxiety due to obsessional thinking. Another possibility is that both phenomena result from an unknown third variable (or combination of variables) such as neurotransmitter interactions.

One might expect that people with OCD (who experience increased anxiety and worry) would show elevated blood flow to certain regions of the brain. Indeed, Cottraux et al. (1996) found that OCD patients and nonpatients both evidenced higher regional cerebral blood flow when exposed to anxiety-evoking, as opposed to neutral, stimuli. Thus, findings that symptom provocation results in activation of the caudate, cingulate, and orbital-frontal cortex in OCD patients (e.g., Rauch et al., 1994) may not reveal anything other than neuroanatomical correlates of a normally functioning brain. Similarly, it should not be surprising that studies have found reductions in metabolic activity in the orbital-frontal cortex following successful treatment of OCD with either medication or behavioral therapy by exposure and response prevention (e.g., Baxter et al., 1992; Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996). Yet because such findings are correlational in nature, they do not demonstrate that OCD results from structural or functional brain abnormality; nor do they suggest that successful treatment works by reducing abnormal brain functioning, as has been espoused in a popular OCD self-help book (Schwartz, 1996).

Taken together, the results of available brain imaging studies provide little in the way of definitive support for neuroanatomical dysfunction in OCD and in other proposed spectrum disorders. Whereas obsessional thinking, compulsive rituals, and feelings of anxiety certainly involve measurable changes in brain states, the same can be said of reading this article. The measurable differences in brain states reported in many studies are what would be expected when a group of anxious individuals (i.e., OCD patients) is compared with a group of individuals without an anxiety disorder. Further data to support this view were reported in a controlled study by Cohen et al. (1996). These researchers attempted to demonstrate the specificity of apparent neuropsychological impairment to OCD, but instead found general functional impairments in both OCD and social phobia patients relative to nonpatients. Cohen et al. (1996) concluded that these findings raise questions regarding the specificity (and etiology) of neuropsychiatric dysfunction in OCD. Moreover, their data suggested that deficits in performance in neuropsychiatric functioning may be the result of situational anxiety (i.e., test anxiety) as opposed to a specific problem with the brains of OCD patients. Correlational and experimental neuropsychiatric studies therefore largely suggest that brain operation in OCD (and in other anxiety disorders) is no less normal than would be observed in most individuals experiencing a state of anxiety or worry.

A further problem for claims that the proposed spectrum disorders are neurologically related to one another (Hollander & Wong, 2000) is that very few neuroimaging studies of other spectrum disorders have been carried out. For example, only two magnetic resonance imaging (MRI) studies of TTM exist to date (O’Sullivan, Rauch, & Brieter, 1997; Stein, Coetzee, & Lee, 1997). Interestingly, both investigations reported results that were inconsistent with MRI studies of OCD patients. Thus, given the largely inconsistent findings and relative paucity of data, arguments for inclusion of OCD spectrum disorders on the basis of a common neuroanatomical "disorder" are unconvincing.

**The Appeal to Associated Features**

Supporters of the OCD spectrum concept also appeal to alleged similarity in features such as demographics, comorbidity, and family history as a basis for inclusion. It is argued that because spectrum disorders seem to have similar onset ages, gender ratios, and comorbidity with OCD, they therefore belong to a family of OCD-related disorders (e.g., Hollander, 1993b; Hollander & Wong, 1995a; 2000). However, this argument (and the data on which it is based) suffers from important difficulties. First, there are alternative explanations for the observed similarity in associated features. Second, large, controlled family studies suggest that many of the hypothesized OCD spectrum disorders are actually less related to OCD than are other anxiety and mood disorders not proposed as part of the spectrum.
Age of Onset, Course, and Gender Ratio

Consistent findings across numerous studies suggest that although OCD may begin at any time from childhood through old age, it has a mean age of onset in the late teenage years into the mid-twenties (see Antony, Downie, & Swinson, 1998, for a review). OCD is also a chronic condition that, absent effective treatment, waxes and wanes throughout its course. Likewise, many of the disorders included in the OCD spectrum begin in late adolescence through early adulthood and follow similar courses (e.g., kleptomania, compulsive shopping, TTM; McElroy et al., 1995; Stein et al., 1995). A noteworthy exception is TS, which tends to begin earlier in childhood (e.g., age 6–7; Zinner, 2000). Similarity in age of onset and course is not, however, persuasive evidence that spectrum disorders are related to one another or to OCD. This is primarily because these demographic features are not in any way specific to OCD and the proposed spectrum disorders. A look through the DSM–IV reveals that many depressive, bipolar, anxiety, factitious, sexual, sleep, personality, psychotic, somatoform, substance abuse, and eating disorders begin during this time of life and evidence a chronic course if effective treatment is not sought. Thus, the fact that proposed spectrum disorders share these associated characteristics with OCD does not indicate anything specific about these conditions, much less that they are related to OCD.

A second problem with appeals to associated demographic features is that there are many discrepancies. For example, as mentioned above, the mean onset age in OCD and TS is quite different. Additionally, whereas OCD seems to affect males and females in approximately equal numbers (Karno, Golding, Sorenson, & Burnam, 1988; Kolada, Bland, & Newman, 1994), the male:female ratio in TS ranges from 2:1 to 4:1 (Zinner, 2000). On the other hand, kleptomania, compulsive shopping, binge eating disorder, TTM, and body dysmorphic disorder appear to be more common in females than in males (McElroy et al., 1995; Tukel, Keser, Karali, Olgun, & Calikusu, 2001). It should be noted that only preliminary data on many spectrum disorders are available at present, and the overall reporting rates of these disorders may be underestimated due to the tendency for many individuals to hide their symptoms from others. Nevertheless, appeals to demographic characteristics such as age of onset, course, and male:female ratio do not appear to provide a cogent argument for conceptualizing the putative spectrum disorders as related to OCD.

Comorbidity and Family History

Investigations of (a) patterns of comorbidity and (b) prevalence of spectrum disorders in first-degree relatives of identified OCD sufferers (proband) have also been used to argue for relationships between OCD and the spectrum disorders. In family studies, diagnostic interviews of OCD probands and their first-degree relatives are conducted to determine the frequency of various diagnoses in this sample. Several family studies of OCD probands have been conducted, although not all employed control groups of healthy individuals. Because of the hypothesized neurological relationship between TS and OCD, researchers have been particularly interested in examining the occurrence of TS and chronic tics in OCD probands and their first-degree relatives. Family studies of OCD have also examined the prevalence of other proposed spectrum disorders, as well as many mood and anxiety disorders.

Researchers have found that perhaps half of patients with TS have comorbid OCD symptoms (e.g., Pauls, Towbin, Leckman, Zahnner, & Cohen, 1986; Pitman, Green, Jenike, & Mesulam, 1987), leading to the suggestion that OCD and TS are “alternative manifestations of the same underlying illness or biological defect” (Pigott, Meyers, & Williams, 1996, p. 136). However, this assertion seems somewhat strong in relation to the present comorbidity data. First, although OCD symptoms have been reported to occur with relatively high frequency among patients with TS, tics are somewhat less common among individuals with OCD and their families (e.g., only 4.6% in first-degree relatives of OCD probands; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995). Second, although there is evidence that TS is associated with lesions to the basal ganglia, there is no evidence that such structural deficits are implicated in OCD (Pigott et al., 1996). Third, Pauls et al. (1995) found that the rates of TS and tic disorders among relatives of individuals with OCD with comorbid tics was significantly higher (10.6%) than among relatives of individuals with OCD without tics (3.2%).

One interpretation of Pauls et al.’s (1995) findings described above is that some presentations of OCD (namely, those in which tics are also present) are related to TS, whereas others are not. Alternatively, this result might be an artifact of the well-known lack of diagnostic precision in distinguishing tics from some types of compulsive rituals (e.g., Miguel et al., 1995; O’Connor, 2001; Pitman et al., 1987). Indeed, some have noted that sampling techniques and diagnostic criteria vary widely across epidemiologic studies (Comings, Himes, & Com-
ings, 1990). It is also worth noting that this patient sample was drawn from a psychiatric research clinic, and methodological problems in evaluating such samples have been noted (e.g., Fallon & Schwab-Stone, 1992). For example, clinic samples disproportionately represent patients with severe psychopathology including high levels of comorbidity. Therefore, in light of the other important differences between TS and OCD, these comorbidity data do not lend themselves to strong conclusions that the two disorders are related.

Two recent controlled studies completed at Johns Hopkins University examined the prevalence of various psychiatric disorders in OCD probands and their first-degree relatives. In one investigation, Bienvenu et al. (2000) found no differences in the lifetime prevalence rates of spectrum disorders such as anorexia nervosa, bulimia nervosa, pathological nail biting, TTM, kleptomania, pathological gambling, and pyromania between individuals with and without OCD. Other spectrum disorders, such as pathological skin picking and nail biting, BDD, and hypochondriasis, did occur more frequently in individuals with than without OCD. There were no differences in the rates of any of these spectrum disorders, except for BDD, among relatives of OCD patients compared with control participants. These findings are largely consistent with our earlier assertions that hypochondriasis and BDD are likely the only proposed spectrum disorders that are related to OCD. Pathological skin picking and nail biting, which were more common among OCD probands than among controls, can be understood as nervous habits, and would thus be likely reported by individuals with OCD, which often involves chronic anxiety and worry.

In a second family study, Nestadt et al. (2001) reported the rates of mood and anxiety disorders among OCD probands, their relatives, and a control group. Results from this study were particularly problematic for the OCD spectrum hypothesis for two reasons. First, Nestadt et al. (2001) found that disorders currently not included in the spectrum (i.e., generalized anxiety disorder, panic disorder, social phobia, specific phobia, and major depression) were significantly more prevalent among OCD probands and their first-degree relatives compared with controls. Second, findings from this study revealed that these disorders, which do not involve stereotypic or repetitive behaviors, were more prevalent than were any of the purported spectrum disorders in previous controlled family studies. Using the argument espoused by spectrum proponents (e.g., Hollander & Wang, 2000), the results described above suggest that mood and anxiety disorders are more strongly related to OCD than any of the putative spectrum disorders such as kleptomania, TTM, TS, compulsive gambling, and shopping.

In summary, the appeal to common associated features in defining OCD spectrum disorders is problematic on several grounds. Primarily, although the age of onset and gender ratio of OCD is similar to that of many proposed spectrum disorders, this pattern is not at all unique to these conditions, and therefore is not compelling evidence for a specific group of OCD-related spectrum disorders. Second, when the spectrum disorders are considered closely, clear evidence of diversity in these features can be found. Third, although family history studies report an overlap between TS and OCD, this data is open to alternative interpretation given that the overlap appears to exist mainly among a select group of patients with obsessive-compulsive symptoms that are often difficult to distinguish from tics in TS. Finally, family studies suggest a more comprehensive overlap between OCD and mood as well as other anxiety disorders, as opposed to the impulse-control and neurological disorders purported to belong in the OCD spectrum. Some have suggested that the entirety of these conditions belong to an affective disorders spectrum that is characterized by comorbidity with mood disturbance, similar response to pharmacologic treatment, and similar underlying pathology (e.g., Goldsmith, Shapiro, & McElroy, 1998). However, much as with OCD spectrum notion, we feel that this solution offers little or no conceptual or clinical advantage over the functional analytic framework for understanding these symptoms.

**OCD Spectrum to Treatment Response**

OCD spectrum advocates claim that the putative spectrum disorders should be grouped together on the basis of their preferential response to specific kinds of treatment. Primarily, it is claimed that these conditions respond more selectively to SSRIs than to other types of medications (Hollander & Wong, 1995a). Indeed several well-designed randomized, double-blinded comparison studies (e.g., Stein, Hollander, Mullen, DeCaria, & Liebowitz, 1992; Volavka, Neziroglu, & Yaryura-Tobias, 1985) and meta-analyses of the literature (Abramowitz, 1997) support the claim of preferential response to SSRIs in OCD. However, the conclusion that each of the proposed OCD spectrum disorders shows a similar preferential response is not supported by the data. First, with the exception of trichotillomania (Swedo, Leonard, & Rapport, 1989), nail biting (Leonard, Lenane, & Swedo, 1991), and stereotypic behaviors in childhood (Castellanos, Ritchie,
& Marsh, 1996), randomized, controlled, double-blind comparisons between serotonergic and nonserotonergic medications in the treatment of OCD spectrum disorders have not been conducted. Hollander and Wong (1995a) based their assertion of preferential response solely on results of preliminary open-trial drug studies that are not designed to answer the question of relative efficacy of medication. It is troubling that such broad speculations about treatment response have been made given the lack of convincing data.

Second, and perhaps more objectionable, is that assertions about preferential response to SSRIs have been made despite preliminary data to the contrary. A survey of open medication trials suggests that non-SSRIs may be quite helpful in treating many of the proposed OCD spectrum disorders, such as kleptomania (McElroy, Keck, & Pope, 1989), compulsive shopping (McElroy, Satlin, & Pope, 1991), and pathological gambling (Moskowitz, 1980), to name a few. Also, neuroleptic medications (e.g., Haldol) that are ineffective as monotherapies for OCD are typically used in the treatment of Tourette’s syndrome (Leckman et al., 1991; Shapiro et al., 1989). Instead of deriving cogent hypotheses on the basis of preliminary research findings, and subjecting such predictions to empirical scrutiny, Hollander and Wong (1995a) appear to have misrepresented this open trial data in a way that lends support for the OCD spectrum hypothesis.

Proponents of this hypothesis have also argued that OCD and the other proposed spectrum disorders show an “overlap in their response to selective antiobsessional behavioral therapies” (Hollander & Wong, 1995a, p. 3). But this claim is also not supported by data. Moreover, the statement reveals a basic misunderstanding of the principles of behavior therapy. Indeed, this is equivalent to stating that all psychiatric illnesses respond to medications. That is, just as depression and schizophrenia are treated using different kinds of medications, OCD and other disorders in the proposed OCD spectrum (e.g., TTM) are treated using highly diverse behavior therapy procedures based on different behavioral principles.

Behavior therapy is a set of experimentally established procedures based on principles of learning, and these procedures are used to weaken and eliminate inappropriate or maladaptive responses (e.g., anxiety, avoidance) to particular circumstances or stimuli (Wolpe, 1958, 1969). The development of a rational and effective behavior therapy program relies on precise information about the antecedents and consequences of the response to be eliminated. The collection of this information (functional [behavioral] analysis; Wolpe, 1958, 1969) is critical to the success of behavior therapy because it influences the choice of therapeutic procedures. Having to consider the functional aspects of OCD in order to treat it is likely what promotes careful and empirical attention to OCD symptoms per se, as compared to the view of OCD as a collection of repetitive behaviors. It is this second view that leads to lumping OCD spectrum disorders together and asserting that all respond to “behavioral therapy.”

As we have noted, clinical functional analyses and research findings reveal that in OCD, obsessional stimuli are associated with increases in anxiety and distress, and compulsive rituals are associated with reductions in this anxiety or distress. Compulsive rituals provide an escape from feared consequences and thus maintain irrational obsessional fears by preventing the sufferer from learning that anticipated catastrophic consequences are unlikely to occur. Thus, effective behavior therapy for reducing OCD symptoms must weaken the associations between (a) obsessional stimuli and excessive anxiety and (b) compulsive rituals and relief from anxiety.

The chief behavioral procedure used to weaken pathological fear responses in OCD (and other anxiety disorders such as claustrophobia) is exposure to fear cues. Exposure entails systematic repeated and prolonged confrontation with fearful stimuli, in the absence of feared consequences, until the pathological anxiety response no longer occurs (extinction). A second procedure, response prevention, is used to weaken associations between compulsive rituals and anxiety reduction. For example, an OCD patient with handwashing rituals and fears of “floor germs” would be repeatedly exposed to floors while refraining from compulsive washing. These procedures have also been applied successfully in the treatment of HC (Visser & Bouman, 2001) and BDD (Neziroglu & Yaryura-Tobias, 1993b), as these can be conceptualized as functionally similar to OCD (as we have discussed above). Exposure for HC and BDD involves evocation of thoughts about illness or appearance, and response prevention entails refraining from checking medical references or one’s appearance. Although there is some debate regarding the mechanism underlying the efficacy of exposure and response prevention, the prevailing cognitive view is that these procedures provide corrective information about the true dangerousness of obsessional situations, leading to a reduction in pathological fear (Foà & Kozak, 1986).

Whereas exposure with response prevention is often effective in reducing OCD symptoms of fears and urges to ritualize, functional analyses suggest that most other disorders included in the OCD spectrum are not appro-
priately treated using these procedures. For example, TTM, as discussed above, involves neither pathological obsessional fears nor urges to perform compulsive rituals designed to escape or neutralize anxiety. Thus, contrary to Hollander and Wong’s (1995a) contention, there would be no logic in using “antiosessional” behavioral procedures, such as exposure and response prevention, in the treatment of this disorder. As we have discussed earlier, hair pulling in TTM is not evoked by obsessional anxiety, but rather preceded by sensations of general tension and fatigue, or feelings of depression, anger, boredom, frustration, or indecision. Unlike compulsive rituals in OCD, which are performed to reduce the probability of danger or distress, hair pulling in TTM is performed because of the pleasurable state it engenders. The hair pulling feels good.

As can be seen, TTM involves considerably different behavioral mechanisms than does OCD. Thus, the therapeutic procedures used in reducing this behavior are necessarily different. Functional analysis logically leads to the use of treatment procedures that hinder attempts to pull (stimulus control), such as wearing mittens, covering hair, or remaining around other people. Procedures that compete with pulling, such as handling a rubber ball, are also implemented along with repeated practice in “high risk” situations. Finally, procedures that help patients avoid strong urges to pull (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 impulse control disorders such as skin picking, pathological gambling, and pathological sexual behavior. Behavioral therapy for alcohol and other forms of substance abuse (which are curiously left out of the OCD spectrum) also features similar procedures.

The foundation for the OCD spectrum is the notion that behaviors in OCD and other spectrum disorders are so peculiar as to be explained only by a brain abnormality. Perhaps this is what has led to the medicalization of behavior therapy and even the claim that this treatment reduces OCD by “unlocking your brain” (Schwartz, 1996). What is problematic about this “broken brain” model is the failure to consider that the symptoms of these disorders adhere entirely to the principles of normal human behavior and can be understood in such terms. Unlike medication, which directly effects neurotransmitter activity, behavior therapy procedures directly modify undesirable behaviors (although they of course affect the brain at some level of functioning). Such effects are possible precisely because we have an understanding of how such behaviors came to be acquired, how they are maintained (i.e., functional analysis), and how to make them go away. All of this is under the rubric of learning, or the analysis of behavior, and none of it either presumes or requires appeal to abnormal or “broken” brains. Thus, current psychological conceptualizations lead to a richer understanding of these problems in that biological processes are implicit, but the more specific psychological mechanisms are the focus of theory and therapy.

Compulsivity vs. Impulsivity

Some OCD spectrum advocates (e.g., Hollander & Wong, 1995a; McElroy, Phillips, & Keck, 1994) have proposed the idea that disorders in the spectrum can be viewed on a continuum from compulsivity to impulsivity. This perspective is based on the notion that compulsivity reflects harm/risk avoidance, whereas impulsivity reflects risk seeking. According to proponents of this view, findings that OCD seems to be characterized by serotonin hyperreactivity, and impulsive behavior by serotonin hyporeactivity support this view. Similarly, whereas OCD has been found to be associated with frontal hyperactivity, impulsivity is known to be associated with a diminution in prefrontal function. According to this dimensional approach, OCD would be placed on the compulsive end of the spectrum, and such impulse control disorders as compulsive gambling, shopping, and sexual behavior, on the impulsive end. Such problems as TTM, kleptomania, or TS, which may have overlapping features of compulsivity and impulsivity, are proposed to lie toward the center of the spectrum.

Nevertheless, the idea that various brain dysfunctions correspond to the proposed OCD spectrum disorders on a continuum from compulsivity to impulsivity is a considerable oversimplification. To see the problem with this hypothesis, one need only to observe that OCD patients sometimes show impulsive or aggressive symptoms, and that patients with impulse-control disorders sometimes show OCD symptoms. Although the usefulness of attempting to conceptualize behavior on a continuum from impulsive to compulsive (as opposed to determining its function) is unclear, the proposed spectrum disorders differ from each other in more ways than simply their degree of impulsivity or compulsivity, as we discussed ear-

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3. Note that in TTM, avoidance of hair-pulling cues is a therapeutic strategy, whereas in OCD, it is exposure to obsessional cues that is therapeutic. Avoidance of obsessional cues would not be therapeutic in the treatment of OCD symptoms because it would prevent the realization that such fears are unfounded.
lier. Moreover, from a neurobiological perspective, it would seem that evidence for that serotonergic hypofunction in OCD (Barr et al., 1992), and in disorders containing impulsive or more stereotypic symptoms, indicates the failure of this approach (Stein, 2000).

**Conclusions**

OCD is distinguished by a specific and unmistakable pattern of thinking and behavior involving (a) intrusive thoughts or ideas that are considered objectionable, repugnant, and anxiety evoking, and (b) attempts to counteract the associated fear or distress through some purposeful action or thought (ritual). Many sufferers engage in observable compulsive rituals (such as washing or checking), whereas others exhibit primarily mental rituals. Nevertheless, a clear relationship between obsessions and subsequent compulsive rituals is present. Understanding OCD symptoms requires a careful and thorough assessment. The condition rewards such an analysis with rich and meaningful patterns of phenomenology that, even in the most impaired patients, can be accounted for (and treated) using little more than an understanding of normal behavioral (i.e., learning) and mental processes.

The current adoration of diagnostic labels in the form of lists of signs and symptoms, in conjunction with the refusal to recognize mental health problems as anything other than indicators of structural or functional brain defects, has blurred an otherwise clear understanding of OCD. Indeed, OCD and other disorders brought under the spectrum concept share a superficial similarity. Hair pulling, tics, and compulsive checking have in common their repetitiveness. However, so do the arm of a baseball pitcher and the jaw of a person chewing gum. Each of these behaviors likely has a similar electrophysiological signature in the brain’s motor cortex (and likely involves the serotonergic system). However, there is hardly a shared underlying cause, much less common neurological defects involved.

The OCD spectrum notion is based largely on an incomplete understanding of the nature of OCD and other spectrum disorders as variants of the same underlying medical illness involving uncontrollable repetitive thoughts, actions, or both. The insinuation that these phenomenologically diverse conditions each represent presentations of OCD is detrimental to advances in clinical treatment and experimental investigation. For example, the spectrum concept promotes the mistaken idea that OCD and, say, kleptomania should respond similarly to behavioral treatment using exposure and response prevention. As we have shown, exposure and response prevention are highly effective in reducing OCD symptoms because these procedures facilitate the extinction of anxiety responses to nonthreatening stimuli (e.g., obsessional thoughts). However, they cannot be logically applied to such impulse control disorders as kleptomania, which do not involve anxiety. Mislabeling individuals with kleptomania as having a form of OCD, and then attempting to apply exposure treatment procedures, will lead to treatment failure, and perhaps misperceptions regarding the effectiveness of behavior therapy for OCD.

Overinclusive criteria for defining OCD also have implications for understanding this complex problem through research. OCD is already a highly heterogeneous condition with many presentations (e.g., checking, washing, hoarding; see Foia et al., 1995), and this diversity is an inherent challenge for researchers attempting to accrue patient samples. Indeed, various presentations of OCD may systematically differ from one another in one or more ways (e.g., treatment response). In efforts to maximize internal validity of both treatment and psychopathology studies, investigators sometimes deal with this heterogeneity problem by studying only a single “type” of OCD (e.g., “checkers” [Constans, Foia, Franklin, & Matthews, 1995]; “washers” [Foia, Steketee, & Milby, 1980]). However, because of the difficulty in accumulating large samples of patients with specific OCD symptoms, most studies have necessarily included all patients meeting the criteria for OCD. If individuals with disorders such as TTM, kleptomania, and pathological gambling were to be conceptualized as having OCD, they might also be included as participants in OCD research, potentially undermining both internal and external validity. Indeed, data gathered using such contaminated samples would likely (and may already) lead to further confusion regarding the nature of OCD.

Another difficulty with the spectrum concept is that it unnecessarily pathologizes OCD as a structural or functional brain defect on the basis of select studies from a body of largely disjunctive research that has produced inconsistent results. Many of these studies are uncontrolled and include small sample sizes. Even if there exist similar neurological abnormalities across the proposed spectrum disorders (e.g., pathological gambling, kleptomania, compulsive buying), they have yet to be identified by controlled research. Thus, strong claims that these disorders share common neuropsychological irregularities are premature. Ultimately, biological research will be extremely important in furthering our understanding of OCD. However, as of yet, a comprehensive neurobio-
logical theory that can be subjected to experimental scrutiny has yet to be clearly articulated.

Finally, spectrum advocates neglect an entire body of research that supports a clearly articulated and logically consistent hypothesis for the etiology and maintenance of OCD that does not hinge on questionable claims of discrete brain abnormalities. This cognitive-behavioral model of OCD also elucidates the distinction between OCD and other proposed spectrum disorders. The model begins with the understanding that intrusive thoughts (e.g., of accidents occurring to loved ones) are universal phenomena that have been found to occur in 90% of the population (Rachman & deSilva, 1978; Salkovskis & Harrison, 1984). Thus, it is not the occurrence of such thoughts, per se, but instead their sequelae that differentiate individuals with OCD from other individuals. Studies have found that individuals with OCD are more likely than nonclinical individuals to misunderstand such thoughts as threatening, or even synonymous with the occurrence of the actual negative event (e.g., Amir, Freshman, Ramsey, Neary, & Brigidi, 2001; Shafar, Thordarson, & Rachman, 1996). This, in turn, leads to affective distress and urges to neutralize the thought (i.e., via compulsive rituals) or engage in behaviors to reduce uncertainty regarding a dreaded outcome. However, responding to thoughts in this way seems to lead to further preoccupation with the thought and the development of obsessive fears (Rassin, Merkelbach, Muris, & Spaan, 1999; Wegner, Schneider, Carter, & White, 1987). Compulsive rituals performed to reduce anxiety are subsequently negatively reinforced by the short-term reduction in affective distress that they engender, and thus are more likely to be repeated under similar circumstances. Avoidance of obsessive situations and stimuli occur because of the desire to evade affective distress, disastrous consequences, or having to perform time-consuming rituals, and thus in severe instances can become extremely debilitating.

This cognitive-behavioral model accounts for obsessions, compulsions, and avoidance, no matter how severe and impairing, and confirmation of this account can be gained via a functional analysis of behavior by interviewing patients with these problems. Further, behavioral treatment procedures derived from this model (i.e., exposure and response prevention) are the most successful techniques for reducing obsessions and compulsions. It therefore seems difficult to justify the adoption of a neuropsychiatric model of OCD given an empirically substantiated and more parsimonious approach of conceptualizing this problem as involving the activation of largely normal psychological learning processes.

The OCD spectrum concept represents an attempt to lump disorders together based on superficial similarities. It is almost as if its originators searched the DSM for disorders with diagnostic criteria involving repetitive behavior and then sought out any data to support inclusion in the spectrum. Instead of adopting an empirical approach, such as factor or cluster analysis, or conducting meaningful research that could place their speculations at risk of refutation, proponents of the OCD spectrum concept appear to have conceived the spectrum concept and then sought evidence to support their claim. OCD is complicated enough by itself, and as much as we, too, long for a meaningful and scientifically sound integration of the disparate disorders of the DSM, the OCD spectrum concept is not a lump we are willing to take.

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