

Review

OBSESSIVE–COMPULSIVE DISORDER: A REVIEW OF THE DIAGNOSTIC CRITERIA AND POSSIBLE SUBTYPES AND DIMENSIONAL SPECIFIERS FOR DSM-V

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Background: Since the publication of the DSM-IV in 1994, research on obsessive–compulsive disorder (OCD) has continued to expand. It is timely to reconsider the nosology of this disorder, assessing whether changes to diagnostic criteria as well as subtypes and specifiers may improve diagnostic validity and clinical utility. **Methods:** The existing criteria were evaluated. Key issues were identified. **Electronic databases of PubMed, ScienceDirect, and PsycINFO were searched for relevant studies. Results:** This review presents a number of options and preliminary recommendations to be considered for DSM-V. These include: (1) clarifying and simplifying the definition of obsessions and compulsions

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(criterion A); (2) possibly deleting the requirement that people recognize that their obsessions or compulsions are excessive or unreasonable (criterion B); (3) rethinking the clinical significance criterion (criterion C) and, in the interim, possibly adjusting what is considered “time-consuming” for OCD; (4) listing additional disorders to help with the differential diagnosis (criterion D); (5) rethinking the medical exclusion criterion (criterion E) and clarifying what is meant by a “general medical condition”; (6) revising the specifiers (i.e., clarifying that OCD can involve a range of insight, in addition to “poor insight,” and adding “tic-related OCD”); and (7) highlighting in the DSM-V text important clinical features of OCD that are not currently mentioned in the criteria (e.g., the major symptom dimensions). **Conclusions:** A number of changes to the existing diagnostic criteria for OCD are proposed. These proposed criteria may change as the DSM-V process progresses. *Depression and Anxiety* 27:507–527, 2010. © 2010 Wiley-Liss, Inc.

Key words: Obsessive compulsive disorder; DSM-V; nosology; tic disorders; early onset; symptom dimensions

INTRODUCTION

Obsessive-compulsive disorder (OCD) is an often-chronic and potentially disabling condition affecting from 1 to 3% of the general population around the globe.^[1–3] Individuals with OCD exhibit heterogeneous symptoms and a broad range of comorbid disorders and outcomes.^[1–3] In DSM-IV, OCD is classified in the section of anxiety disorders. DSM-IV criteria are given in Table 1.^[4] In ICD-10, OCD is classified in the section of Neurotic, Stress-related, and Somatoform disorders.^[5] This review focuses on some of the key issues pertaining to OCD that are being considered for the Diagnostic and Statistical Manual of Mental Disorders (DSM) Fifth Edition (DSM-V). Given that research on OCD has substantially increased since DSM-IV was published in 1994,^[4] it is important to consider whether changes in the diagnostic criteria are needed to reflect new knowledge. Our focus is limited to considering possible revisions to the diagnostic criteria and how best to parse OCD in terms of specifiers and dimensions. This article was commissioned by the DSM-V Anxiety, Obsessive-Compulsive (OC) Spectrum, Post-traumatic, and Dissociative Disorders Work Group. It represents the work of the authors for consideration by the work

group. Recommendations provided in this article should be considered preliminary. They do not necessarily reflect the final recommendations or decisions for DSM-V, as the DSM-V development process is still ongoing.

STATEMENT OF THE ISSUES

REFINEMENT OF THE DSM-IV CRITERIA

Criterion A.

- DSM-IV, in contrast to ICD-10, provides separate definitions of obsessions and compulsions. Should the separate definitions be maintained?
- Should “impulse” be changed to “urge”?
- Should “inappropriate” be changed to “unwanted”?
- Should it be stated explicitly that obsessions “cause marked anxiety or distress,” or should this be modified or deleted from DSM-IV?
- Should what is meant by “ignoring or suppressing obsessions” or “neutralizing” them with some other thought or action be clarified?
- Should the phrases, “the thoughts, impulses, or images are not simply excessive worries about real-life problems” and “the person recognizes that the obsessional thoughts, impulses, or images are a

Footnote continued.

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TABLE 1. DSM-IV diagnostic criteria for 300.3 obsessive–compulsive disorder

A. Either obsessions or compulsions: <i>Obsessions as defined by (1), (2), (3), and (4):</i>	
1. Recurrent and persistent thoughts, impulses, or images that are experienced, at some time during the disturbance, as intrusive and inappropriate and that cause marked anxiety or distress	
2. The thoughts, impulses, or images are not simply excessive worries about real-life problems	
3. The person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralize them with some other thought or action	
4. The person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion)	
<i>Compulsions as defined by (1) and (2):</i>	
1. Repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly	
2. The behaviors or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these behaviors or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent or are clearly excessive	
B. At some point during the course of the disorder, the person has recognized that the obsessions or compulsions are excessive or unreasonable. <i>Note:</i> This does not apply to children	
C. The obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day), or significantly interfere with the person's normal routine, occupational (or academic) functioning, or usual social activities or relationships.	
D. If another Axis I disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g., preoccupation with food in the presence of an Eating Disorder; hair pulling in the presence of Trichotillomania; concern with appearance in the presence of Body Dysmorphic Disorder; preoccupation with drugs in the presence of a Substance Use Disorder; preoccupation with having a serious illness in the presence of Hypochondriasis; preoccupation with sexual urges or fantasies in the presence of a Paraphilia; or guilty ruminations in the presence of major depressive disorder)	
E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition	
<i>Specify if:</i>	
• With Poor Insight: if, for most of the time during the current episode, the person does not recognize that the obsessions and compulsions are excessive or unreasonable.	

- product of his or her own mind (not imposed from without as in thought insertion)” be moved to criterion D (the diagnostic hierarchy criterion)?
- g. Should the notion that obsessions and compulsions are typically not pleasurable be emphasized in criterion A or B?
- h. Should the fact that avoidance is often associated with obsessions and compulsions be added to criterion A, B, or C?

Criterion B. Is the criterion that obsessions or compulsions are “excessive or unreasonable” valid? If

not, can this clinical criterion be better defined or operationalized?

Criterion C. Can the clinical significance criterion be better operationalized? Should the 1-hr portion of this criterion be maintained?

Criterion D. Should the diagnostic hierarchy with other mental disorders be retained? If so, should it specifically mention other disorders in addition to those already mentioned in the criterion?

Criterion E. What is meant by a “general medical condition”? For example, does this include Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS)?

OCD SUBTYPES

How strong is the evidence for specific OCD subtypes: tic-related OCD, early-onset OCD, and PANDAS? (Hoarding Disorder is covered by a separate review).^[6]

OC SYMPTOM DIMENSIONS

How strong is the evidence for incorporating OC symptom dimensions including: contamination/cleaning, hoarding, and symmetry/doing-redoing?

SIGNIFICANCE OF THE ISSUES

Although OCD is a prevalent disorder affecting as many as 3% of the population, many more individuals have sub-clinical forms of OCD that frequently lead to help-seeking behavior.^[1–3,7–9] How OCD is formulated in the diagnostic criteria has a major impact on prevalence rates, who will be selected to participate in future scientific research studies, and who will be offered evidence-based treatment for OCD. The inclusion of additional specifiers has the potential to improve clinical practice and may contribute to a deeper knowledge of the neurobiological and psychological substrates and cognitive-behavioral mechanisms that underlie this disorder. Our goal in reviewing the nosology of OCD is not only to incorporate new data but also to consider ways to simplify or clarify the wording and organization of the criteria to make them more clinically useful.

SEARCH METHODS

Electronic databases of PubMed, ScienceDirect, and PsycINFO were searched for relevant studies using relevant search terms (“obsessive–compulsive disorder” or “OCD”) and “DSM-IV” or “diagnostic criteria” or “factor analytic studies” or “factor” or “dimension” or “subtype” or “class.” The Annotated Listings of Changes in DSM-III, DSM-III-R, and DSM-IV; the DSM-IV Sourcebooks; and the DSM-IV Options Book were consulted for details of the DSM-III to DSM-IV OCD criteria revisions.^[10–15] The proceedings and/or monographs of the preparatory conference series for

DSM-V, particularly the *OC Spectrum Disorder* conference were also used. The reference sections of selected articles and reviews in this area were also searched for citations of further relevant published and unpublished research. As this review is complementary to the other reviews contained in this special issue, there are several important topics that are not covered, including where OCD should be positioned in the organization of DSM-V.^[16,17] Nor will we fully consider whether or not Hoarding Disorder should be a separate diagnostic entity, as this is being covered in a separate review.^[6] In addition, the issue of insight (criterion B) will be more fully discussed in a separate literature review. (Personal communication, Katherine Phillips, January 2010).

RESULTS

CRITERION A

Definition of Obsessions and Compulsions: DSM-IV, in contrast to ICD-10, provides separate definitions of obsessions and compulsions. Should the separate definitions be maintained? DSM-IV and ICD-10 take a somewhat different approach to defining obsessions and compulsions.^[4,5] In ICD-10, an obsession is a thought, idea, or image (i.e., a cognitive event), whereas a compulsion is an act. ICD-10 emphasizes that obsessions and compulsions share features such as being repetitive and unpleasant.

In contrast, DSM-IV defines obsessions as recurrent and persistent thoughts, images, or impulses; thus, obsessions are not only cognitive events. Moreover, in DSM-IV, compulsions are repetitive behaviors or mental acts; thus, compulsions also can be cognitive events. A key difference between obsessions and compulsions in DSM-IV is that obsessions cause anxiety or distress, whereas compulsions are aimed at preventing or reducing distress or some dreaded event. Thus, unlike ICD-10, DSM-IV assumes a functional relationship between obsessions and compulsions. Finally, there is an implicit distinction between obsessions and compulsions in the wording in DSM-IV: an obsession is involuntary ("intrusive") whereas a compulsion is voluntary ("act"). These distinctions are central to the cognitive-behavioral therapy (CBT) approach to the conceptualization and treatment of OCD.^[18]

For a practitioner who is used to the ICD-10 approach, there are several potential problems with the DSM-IV criteria. First, in DSM-IV a thought can be either an obsession or a compulsion, depending on whether it causes anxiety or is aimed at preventing anxiety. Without appreciating the functional relationship that DSM-IV assumes, this can be confusing. Second, not all obsessions generate anxiety or distress (as discussed in more detail below), and some patients report that the uncontrollability of their compulsions is a source of distress. Third, although DSM-IV assumes

a functional relationship between obsessions and compulsions, DSM-IV also allows a diagnosis of OCD to be made if a patient has only obsessions or compulsions. This is an apparent contradiction. Finally, not all patients experience their compulsions as voluntary (i.e., within their control). This raises the question of what is meant by voluntary.

On the other hand, there is a clear advantage to retaining the current DSM-IV criteria given that OCD is a prevalent and widely studied disorder. For clinical purposes and in research studies, the current criteria have worked reasonably well since 1994. Furthermore, there is a good deal of data to support the functional relationship between obsessions and compulsions, and embodying this relationship in the diagnostic criteria may be clinically useful insofar as it provides an accurate conceptualization of the nature of OCD of symptoms.

Summary and preliminary recommendations. DSM-IV defines obsessions and compulsions somewhat differently than ICD-10. There is no simple way to resolve this incompatibility without a fundamental change in either the ICD-10 or the DSM-IV definition. Given the possible clinical utility of emphasizing a functional relationship between obsessions and compulsions, and in the absence of evidence that the ICD-10 definition of obsessions and compulsions is preferable, we recommend that separate definitions of obsessions and compulsions remain in criterion A, although clarifications and simplifications of these definitions are recommended, as noted below.

Should "impulse" be changed to "urge"? Based on clinical experience, the term "impulse" in criterion A is possibly confusing, in that impulses are characteristic of the impulse control disorders. The latter are sometimes misdiagnosed as OCD. The use of the term "urge" may more accurately reflect some of the common symptoms of OCD, e.g., obsessions with aggressive, religious, or sexual content. Such patients can have intrusive urges, e.g., to harm themselves or others, which generate marked anxiety or distress because of fears of acting on these urges.

One option is to replace the term "impulse" with "urge."^[20] Urge is defined as a continuing impulse toward an activity or goal.^[21] Although both "urge" and "impulse" connote immediate, pre-reflexive acting, impulses are usually seen as more fleeting and urges more long-lasting and persistent. Both terms enrich the purely cognitive description of "obsession" presented in ICD-10.^[5]

There are both advantages and disadvantages to replacing "impulse" with "urge." Advantages include the fact that the word "urge" might decrease confusion of impulse disorders with OCD, and it conveys something more persistent than a fleeting impulse. On the other hand, "urge" may connote the drive in various behavioral addictions and the sensations some patients have prior to tics. Another disadvantage of the term "urge" is that it may be confused with a

compulsion, since “urge” is often used in the context of compulsive rituals, e.g., “the urge to do or think certain things repeatedly can dominate your life unhelpfully” (<http://www.rcpsych.ac.uk/mentalhealthinfo/problems/obsessivecompulsivedisorder/obsessivecompulsivedisorder.aspx>.) Finally, some may favor “impulse” because it emphasizes similarities between impulse control disorders and OCD.

Summary and preliminary recommendations.

Both “impulse” and “urge” capture the involuntary (losing control) nature of some obsessions. Since the concept “impulse” indirectly refers to the notion of impulse control disorders, which may complicate differential diagnosis, our recommendation at this time is to replace “impulse” with “urge” in criterion A and mention alternative terms such as “impulse” in the text. A potential limitation of the word “urge,” however, is that it may create less differentiation from tic disorders.

Should “inappropriate” be changed to “unwanted”? Classically, the content of obsessions has been considered to conflict with a person’s personality and prior history.^[22] DSM-IV requires that obsessions be experienced as “inappropriate,” in an attempt to operationalize the traditional concept of ego-dystonicity.^[4] Indeed, from a clinical perspective patients can experience their symptoms as “inappropriate.” However, it is not clear that defining an obsession as “inappropriate” is the clearest description, given that different cultures, genders, and ages have different definitions of what is appropriate and what is inappropriate.^[23] Furthermore, “inappropriate” may be interpreted as absolute (i.e., in relation to standard societal or cultural values) or as relative (i.e., in relation to patient personal norms and values). Although criterion A explicitly states that patients *experience* the content of their obsessions as inappropriate, thereby stressing the relative nature of “inappropriate,” “unwanted” may be a better word, as it is less value-laden. In addition, “unwanted” was the term used in DSM-I and DSM-II.^[10,11]

Summary and preliminary recommendations.

Operationalizing the concept of ego-dystonicity of obsessions is difficult. On balance, given that different cultures, genders, and ages have different definitions of what is appropriate and what is inappropriate, and that “unwanted” was used in earlier editions of DSM, we suggest replacing “inappropriate” with “unwanted” in criterion A. At the same time, “unwanted” might introduce other problems (e.g., it focuses on volition and ignores content of the obsession).

Should it be stated explicitly that obsessions “cause marked anxiety or distress” or should this be modified or deleted from DSM-IV? The DSM-III-R definition of obsessions expressed only indirectly that obsessions are distressing, while the definition of compulsions explicitly stated that compulsive behavior was designed to reduce discomfort. Although obsessions are not explicitly defined as aversive, the individual was said to attempt to “ignore, suppress, or neutralize”

them. In the DSM-IV Sourcebook,^[24] the proposal was made to specify that obsessions “are experienced as intrusive and inappropriate, and cause marked anxiety or distress,” and this change was made in DSM-IV.

The DSM-IV workgroup supported their proposal with findings from several studies indicating that “ruminative thoughts give rise to greater elevation of heart rate and increased skin conductance response than do neutral thoughts.”^[25,26] In addition, both actual and imaginary confrontation with contamination has been found to result in increases in heart rate, subjective anxiety and skin conductance response.^[27,28] Such physiological changes have been repeatedly found to be associated with anxiety.^[24,27]

Clinical experience and data from clinical trials and longitudinal studies generally support retaining anxiety and distress in the definition of obsessions.^[20,27,29–33] For example, OCD experts often consider whether a patient experiences anxiety or distress to help differentiate the obsessions of OCD from the recurrent thoughts or images that are present in other disorders (e.g., intrusive sexual images in paraphilias).^[30] As mentioned above, the anxiety-inducing element of obsessions is central to the CBT perspective on OCD.^[19] In addition, data from several large OCD studies demonstrate that the vast majority of people with OCD experience moderate or severe anxiety or distress from their obsessions. For example, in 122 untreated adults with OCD who were evaluated by trained raters before entering a randomized clinical trial, most reported that their obsessions caused either moderate or severe distress (none: 0%; mild: 12.3%; moderate: 41.8%; severe: 43.4%; extreme: 2.5%).^[31] These data are comparable to similar data from a large randomized controlled trial in children (POTS study) and data from a longitudinal naturalistic study of children and adults with OCD.^[32,33] At the same time, these data also demonstrate that not all obsessions generate “marked” anxiety or distress (i.e., that is at least moderate in severity). Taken together, these data support the notion that obsessions typically, but not always, cause marked anxiety or distress in people with OCD and that compulsions are usually performed in an effort to reduce anxiety or distress.

Summary and preliminary recommendations.

Given the available data, it is recommended that the phrase “cause marked anxiety or distress” be modified to state that obsessions “usually cause marked anxiety or distress.” In addition, the definition of “compulsion” should indicate that compulsions are behaviors or mental acts that are aimed at preventing or reducing “anxiety or distress.”

Should what is meant by “ignoring or suppressing obsessions” or “neutralizing” them with some other thought or action be clarified? As mentioned above, DSM-IV assumes a functional relationship between obsessions and compulsions, since compulsions are often done in response to an obsession. In fact, in the DSM-IV field

trial, most adults with OCD (96%) had both obsessions and compulsions when evaluated by trained raters using the Yale-Brown Obsessive Compulsive Scale Symptom Checklist (Y-BOCS-SC).^[34–36] However, DSM-IV also allows a diagnosis of OCD if a person has only obsessions or only compulsions.

The DSM-IV definition of an obsession requires that a person respond to an obsession by attempting to ignore, suppress, or neutralize it with some other thought or action. Attempts to ignore or suppress the obsession can take different forms (e.g., avoiding people, places, and things that generate obsessions, using distraction techniques). On the other hand, attempting to neutralize the obsession with some other thought or action refers to performance of a compulsion. This differentiation is not specified in the criteria and is potentially confusing, given that the definition of compulsion follows later, and it may not be clear to a clinician who does not have expertise in OCD what “neutralizing the obsession” means.

Summary and preliminary recommendation.

One option is to specify in criterion A what is meant by the phrase “to neutralize them with some other thought or action” by clarifying in a parenthesis that this means in many instances to perform a compulsion. However, in an attempt to keep the criteria relatively simple, we suggest that this be done in the text instead. The text needs to be clear that compulsive rituals are but one form of neutralizing behavior that people with OCD use to deal with their obsessions and reduce anxiety/distress (others being thought suppression, distraction, mental analyzing, avoidance, reassurance seeking, among others). Clarifying this point is crucially important and various specific examples should be described so that readers of the DSM do not simply look for overt compulsions to define OCD, but are also sufficiently knowledgeable to inquire about these more covert neutralizing strategies, which serve the same function as compulsions.

Should the phrases, “the thoughts, impulses, or images are not simply excessive worries about real-life problems” and “the person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion)” be moved to criterion D? It is important to differentiate OCD obsessions from worries about real-life problems that are present in generalized anxiety disorder (GAD) and from psychotic thoughts that characterize schizophrenia or other psychotic disorders. However, it is also important to differentiate obsessions from a whole range of other psychiatric symptoms. It is unclear why these particular disorders are mentioned in criterion A.

Summary and preliminary recommendations.

There is no clear advantage for the definition of obsessions to include information about the differential diagnosis specifically between OCD, GAD, and psychotic disorders. We recommend moving these components of criterion A to criterion D. This move

will simplify the definition of an obsession and enhance consistency by placing all diagnostic hierarchy issues in one criterion.

Should the notion that obsessions and compulsions are typically not pleasurable be emphasized in criterion A or B? DSM-III-R required that patients not derive pleasure from carrying out a compulsion, although compulsions could provide a release of tension; however, this was removed in the DSM-IV edition. ICD-10, on the other hand, explicitly mentions that both obsessive thoughts and compulsive acts are not in themselves pleasurable.

Although there is little empirical evidence on this issue, clinical experience indicates that the more a particular behavior is enjoyed, the less likely it is to be a compulsion of OCD. Moreover, the fact that the compulsive behavior does not in itself produce a pleasurable outcome (as opposed to reducing anxiety) is of value for the clinician in differentiating compulsions associated with OCD from other disorders with compulsive behaviors, and in particular from “purely” impulsive acts, sexual paraphilic behaviors, or pathological gambling. As noted above, the fact that obsessions typically are not pleasurable can help distinguish them from non-OCD intrusive thoughts. Thus, adding the fact that obsessions and compulsions are generally not pleasurable might increase the reliability of the diagnosis of OCD.

On the other hand, there are currently no strong empirical data to support this change and adding it to criterion B and thus requiring it for a diagnosis of OCD might change caseness in unexpected ways. In addition, criterion A states that obsessions cause marked anxiety or distress, already indicating, albeit indirectly, that at least obsessions are not pleasurable. Furthermore, some patients (e.g., those with symmetry/exactness concerns) report some pleasure from their compulsions. Finally, instead of adding a new criterion about pleasure, it may be simpler to indicate under criterion D those disorders that have pleasurable intrusive thoughts or compulsive behaviors and that can be confused with OCD.

Summary and preliminary recommendations.

Data are needed to determine whether the reliability of diagnosis is improved by including in the criteria the notion that obsessive thoughts and compulsive acts are not usually in themselves pleasurable. Without such data, it is probably preferable not to add this to the criteria. However, at a minimum, this should be described in the text, and disorders that can be confused with OCD should be specified under criterion D.

Should the fact that avoidance is often associated with obsessions and compulsions be added to criterion A, B, or C? It is well known from a clinical perspective that OCD patients actively avoid certain persons, places, objects, or actions because they fear that these will prompt the obsessions or lead to compulsions. Avoidance can lead to significant clinical impairment. Yet, avoidance is not specifically mentioned in DSM-IV criteria for OCD.

However, whether to include avoidance in the criteria is not clear.

One option is to introduce avoidance under criterion A (in the definition of either obsession or compulsion). However, although the concept of avoidance is often used to refer to avoidance of triggering obsessions, it can also refer to avoidance of anticipated anxiety/distress or to avoidance of starting compulsions (e.g., the person with a contamination obsession who has not cleaned the house in years because to start is never to stop). Thus, avoidance is not unique to obsessions. Likewise, avoidance, although in some senses a behavioral act that can reduce anxiety, is not quite the same thing as a compulsion as currently defined. A second option is to add avoidance to criterion B and require it for the diagnosis of OCD. In fact, for some of the other anxiety disorders, avoidance is a key feature of the diagnostic criteria in DSM-IV.^[4] However, empirical data are needed on the prevalence and nature of avoidance in OCD to understand how requiring avoidance might affect caseness. Finally, avoidance could be introduced in the clinical significance criterion (criterion C in DSM-IV), since obsessions, compulsions, anxiety/distress, and subsequent avoidance can all lead to impairment, and sometimes it is avoidance that is the most impairing. However, it is not clear how one would operationalize this since avoidance is the absence of behavior.

In sum, although avoidance is an important clinical feature of OCD, it is not clear how best to operationalize avoidance so that it can be adequately assessed by clinicians. Part of the challenge is that there are many different types of avoidance, as described above. Moreover, in common parlance, a patient who refrains from doing rituals could be described as avoiding their rituals, but this is considered a sign of health, not a sign of pathology.

Summary and preliminary recommendation. On balance, our preliminary recommendation is to discuss avoidance more clearly in the text rather than adding it to the criteria. Before final recommendations are made for DSM-V, it will be helpful to consider how avoidance will be addressed for other disorders.

CRITERION B

Is the criterion that obsessions or compulsions are “excessive or unreasonable” valid? If not, can this clinical criterion be better defined or operationalized? Criterion B requires that a person with OCD has at some point during the course of the disorder recognized that the obsessions or compulsions are excessive or unreasonable. One problem with this criterion is that neither “excessive” nor “unreasonable” are defined or operationalized and it is unclear how clinicians and researchers interpret them. Presumably this criterion pertains to insight about the accuracy of the beliefs that underlie the obsessions, (e.g., the belief that the house will burn down if the stove is not checked) or the rationality

of the compulsions (e.g., the need to check the stove 30 times to be safe). Studies have shown, however, that some patients with OCD have poor or even absent insight; in two studies in different samples, about 2% of patients completely lacked insight, i.e., the belief underlying their obsession was delusional.^[37] Technically speaking unless they had shown some insight during the course of the disorder, such patients should receive a diagnosis of delusional disorder or psychotic disorder NOS, not OCD.

Data from the DSM-IV field trial similarly demonstrated that OCD is characterized by a spectrum of insight, including delusional beliefs: only 13% of 250 OCD patients were certain that their feared consequences would *not* occur.^[36] The majority of patients were either uncertain (30%) or mostly certain that the consequence would occur (26%); 4% were completely certain that their feared consequence would occur.

These data from the DSM-IV OCD field trial led to the addition of the specifier “with poor insight” in DSM-IV. The addition of this specifier was useful in that it focused attention on poor insight as an important clinical feature of OCD. However, the wording of this specifier is problematic, as it uses the same unclear terminology as in criterion B, e.g., “excessive” and “unreasonable;” in addition, it focuses on the “current episode,” even though OCD is not typically an episodic illness.^[38] The poor insight specifier also does not reflect that insight varies along a continuum in OCD, from good to absent. Finally, this specifier does not resolve the question of whether an OCD patient who completely lacks insight should be diagnosed with a psychotic disorder instead of OCD.

Summary and preliminary recommendations. As neither “excessive” nor “unreasonable” are defined or operationalized, it is unclear how clinicians and researchers interpret them; indeed, they are difficult to define and can have different meanings. Given that insight varies in OCD patients, and can include delusional OCD beliefs, we suggest deleting criterion B from OCD. This may help prevent misdiagnosis of OCD as schizophrenia or another psychotic disorder, which clinical experience suggests sometimes occurs. However, the impact of this deletion on caseness should first be examined. In parallel, we recommend that OCD’s delusional variant be eliminated from the psychosis section of DSM.

We further recommend that OCD’s poor insight specifier be expanded to include not only poor insight but a broader range of insight options, such as: (a) good or fair insight, (b) poor insight, or (c) delusional OCD beliefs. Such specifiers have the potential advantage of conveying the broad range of insight that can characterize OCD beliefs, including delusional beliefs.^[37] These specifiers are similar to categories in the Brown Assessment of Beliefs Scale (BABS) and YBOCS.^[34,35,39] Further evidence regarding these specifiers, their potential clinical utility, and use and

definition of the term “insight” are further considered in a separate review (Personal communication, Katherine Phillips, January 2010). Alternatively, additional specifiers can be considered: fair insight could be separated from good insight, or “good or fair insight” could be replaced by separate categories of “excellent,” “good,” and “fair” insight, as in the BABS and YBOCS. Potential advantages of the greater specificity must be weighed against the possibility that more specifiers might be burdensome for clinicians. The proposed option would also allow compatibility with the insight specifiers proposed for other disorders such as body dysmorphic disorder (BDD). At the same time, the ability of clinicians to use such an insight specifier reliably and its clinical utility should be assessed.

CRITERION C

Can the clinical significance criterion be better operationalized? There are several reasons to consider possible changes to criterion C. One concern about this criterion is that in non-psychiatric medical disorders, a disease entity usually is defined in terms of its core symptomatology or underlying pathophysiology, not its functional consequences. On the other hand, the etiology and psychophysiology of most psychiatric disorders are not sufficiently understood to be useful for diagnostic purposes. In addition, many of the characteristic symptoms of psychiatric disorders are present in the same form, as part of everyday life, in many in the general population. For example, studies have demonstrated that most people have occasional intrusive thoughts, and at least half of people have repetitive behaviors or rituals.^[8,9,40] Thus, the clinical significance criterion serves as a clinically useful cut point, one that is helpful in differentiating disorder from normalcy.

A second potential concern about this criterion is that different individuals may have different thresholds for experiencing subjective distress and for displaying objective impairment in functioning. Thus, a more emotionally demonstrative person may voice distress in response to milder symptoms. A more resilient person may not display impairment in functioning despite suffering from severe symptoms. The functional impact of a particular level of symptoms also may differ depending upon the level of demand required by the person's occupation, or the amount of available social or financial support. Clinical judgment is therefore required in delineating the cut points of clinically relevant distress and impairment.

Third, the requirement that OCD symptoms take more than 1 hr a day to be considered time-consuming seems somewhat arbitrary. As is noted in the accompanying BDD review, a potential disadvantage of including a specific time criterion is that there are no data to support a particular cut point, and any cut point is somewhat arbitrary.^[41] Moreover, recent data have shown that even people with obsessions and

compulsions which last less than 1 hr a day may have significant impairment in functioning.^[8,9] For example, Denys et al. used data from the Netherlands Mental Health Survey and Incidence Study (NEMESIS), a large representative sample of the general Dutch population ($N = 7,076$), to compare subjects without obsessions or compulsions (94.2%), subjects with obsessions and compulsions who did not meet full DSM-III-R criteria (4.9%), and subjects with “full-blown” OCD according to DSM-III-R (0.9%).^[8] These three groups were compared on psychological vulnerability, health and functional status, psychiatric comorbidity and seeking treatment. Sub-threshold subjects scored equally to OCD subjects on all measures, except for seeking treatment. OCD subjects and sub-threshold subjects scored worse on most measures when compared to the subjects without obsessions or compulsions. These data challenge the validity of the clinical significance criterion as it is formulated in DSM-III-R and DSM-IV.

At the same time, changing clinical significance criteria can produce large differences in prevalence rates. For example, Crino et al. found that the 12-month prevalence according to the former DSM-III criteria and the more stringent DSM-IV criteria differed more than 3-fold (2.1 versus 0.6%, respectively).^[42] Thus, changes in clinical significance criteria should be supported by very strong evidence.

Summary and preliminary recommendations. Because intrusive thoughts and repetitive behaviors are so common in the general population, it is important that the criteria differentiate clinically significant from clinically non-significant symptoms to avoid overpathologizing normal experience. How to determine clinical significance optimally is a key issue across the diagnostic system. Ideally, distress and impairment should be better operationalized in DSM-V. In addition, the language of this criterion in OCD differs from other disorders. We suggest rewording it so that it is more consistent (e.g., deleting the phrase “significantly interfere with the person's normal routine” and rewording the rest of that sentence), although the impact of this change on caseness needs testing. Otherwise, we are not aware of any evidence-based suggestions for how to improve this criterion for OCD. However, for the reasons discussed above, we recommend indicating that more than 1 hr a day is a guide to what is time consuming (by using it as an example) rather than being an absolute requirement for the diagnosis.

CRITERION D

Should the diagnostic hierarchy with other mental disorders be retained? If so, should it specifically mention other disorders in addition to those already mentioned in the criterion? Since obsessions can be confused with other intrusive or recurrent thoughts (e.g., worries, intrusions, ruminations, and delusions), and compulsions can be confused with stereotypies, tics, impulses, urges, habits, and perseveration, it is useful to identify

those disorders that need to be differentiated diagnostically. For example, the ruminative thoughts of depressed patients may resemble obsessive preoccupations, and the excessive worries of patients with GAD may resemble obsessive thinking. Patients with hypochondriasis may present with unrealistic preoccupations with physical disease that closely resemble obsessive ruminations of OCD; somatic checking rituals and frequent visits to the physician's office for reassurance may play an anxiety-reducing role similar to that of compulsive rituals. Patients with anorexia nervosa or BDD may have obsessive preoccupations with physical appearance as well as compulsive behaviors, e.g., excessive grooming in BDD or ritualistic behaviors around food in anorexia nervosa. The diagnosis of obsessive-compulsive personality disorder (OCPD), with its preoccupation with orderliness and perfectionism, can be confused with OCD.

Summary and preliminary recommendations.

We recommend that the diagnostic hierarchy criterion be retained. However, we recommend that the opening sentence of this criterion be reworded for clarity and consistency with how this criterion is worded for other disorders. We further recommend that the most important differential diagnoses continue to be explicitly listed in the new criterion C (the old criterion D). As mentioned above (see criterion A), we also recommend moving the phrase that obsessions are not simply excessive worries about real-life problems (as in GAD) and the phrase that the person recognizes that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion and a psychotic disorder) to the new criterion C (the old criterion D). Finally, we recommend adding to this list ritualized eating behavior in the presence of an eating disorder, compulsive sexual behavior, and preoccupation with gambling or other relevant behaviors in behavioral addictions or other impulse control disorders. If hoarding disorder and skin-picking disorder are added to DSM-V, they will also need to be mentioned in the new criterion C.^[6]

CRITERION E

What is meant by a “general medical condition”? For example, does this include Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal Infections (PANDAS)? Criterion E states that OCD is not due to the direct physiological effects of a substance or a general medical condition. In such cases, DSM-IV has other relevant diagnostic categories, such as Anxiety Disorders Due to a General Medical Condition. This criterion reminds clinicians that general medical disorders may masquerade as psychiatric disorders; e.g., hyperthyroidism may present with panic attacks and be misdiagnosed as panic disorder.

With increasing knowledge of the underlying neurobiology of OCD, the question arises of whether this approach remains valid and what is really meant by a “general medical condition.” There is strong evidence that OCD is mediated by frontal cortico-striatal thalamic circuitry. Thus, anything that affects this circuitry has the potential to lead to symptoms of OCD. Emerging data demonstrate that this circuitry may be impacted by relatively gross neurological insults (e.g., stroke) or by a range of more subtle mechanisms, including multiple uncommon gene variants, or environmental factors such as streptococcal infection.^[43] Currently, if OCD occurs with a tic disorder in the context of a streptococcal infection, both OCD and the tic disorder are diagnosed since the link between the streptococcal infection and the development of OCD remains tenuous, although the presumed mechanism is dysfunction in the striatum. Yet if a striatal stroke leads to OCD,^[44] this might be diagnosed as an Anxiety Disorder due to a General Medical Condition. Thus, the effect of criterion E is that certain etiological agents (e.g., a stroke) exclude a diagnosis of OCD whereas unknown etiological agents (including those that might be described as a “general medical condition” such as a streptococcal infection) permit a diagnosis of OCD. Without changes to criterion E, the result may be that as we learn more and more about the etiology of OCD, we will exclude more and more cases of OCD, and OCD will end up being diagnosed only in those whose etiology is idiopathic.

Several options are available. First, criterion E can remain unchanged with discussion in the text of the range of neurobiological mechanisms that are being discovered to contribute to the pathogenesis of OCD. The advantage is that clinicians would remain aware that in some cases, substance abuse and general medical conditions can masquerade as OCD. This approach may reflect the reality that in most cases what causes OCD is unknown. The disadvantage of keeping criterion E unchanged is that this draws a clear line between psychiatric and medical disorder, whereas in fact the reality seems more complex.

Another option is for criterion E to exclude explicitly known medical disorders that should not be confused with OCD. For example, some would propose that OCD following a streptococcal infection or OCD following stroke should be explicitly excluded on this basis. The advantage is that clinicians may then be encouraged to consider such medical disorders and, for example, to assess and treat OCD due to these causes in an appropriate way. The disadvantage is that there is controversy in the field regarding the role that streptococcal infection plays in the etiology of OCD and little data to support alternative treatments at the moment.^[45–49] Also, if OCD is due to dysfunction in fronto-striatal-thalamic circuits, then a stroke in this circuit might not be masquerading as OCD but may actually be causing the symptoms of the syndrome called OCD.

A third approach would be to omit the diagnosis of Anxiety Disorder Due to a General Medical Condition and code all cases of OCD on axis I, and the known medical contributing factors on Axis III (if Axis III is retained in DSM-V). The advantage of this is that it would convey that psychiatric disorders are not merely “mental” but are instead brain–mind disorders, with complex biological underpinnings. Then, one could sub-classify OCD based on the different etiologies. The disadvantage of this option is that in the absence of a clear medical exclusion criterion, clinicians may not remember to pay enough attention to ruling out treatable medical disorders that can masquerade as OCD. Also, in most cases we do not know enough about etiology in an individual case to subtype OCD according to etiology. Finally, Axis III now records all medical disorders, not just the ones that contribute to an Axis I disorder.

Summary and recommendations. The issues raised above are not unique to OCD but need to be addressed across DSM-V. What is meant by a “general medical condition” needs better definition. In the interim, we suggest that no change be made to the language of the old criterion E (the new criterion D). However, we suggest that the text describe what is known about the etiology and pathophysiology of OCD and clarify which medical conditions can masquerade as OCD and require treatments other than our current treatments for OCD.

OCD SUBTYPES

A number of potential subtypes of OCD have been proposed for inclusion in DSM-V. They include: tic-related OCD, early-onset OCD, and PANDAS.^[50] Each of these potential subtypes is associated with onset in the prepubertal period.

Tic-related OCD: It has been suggested that some forms of OCD can be etiologically related to chronic tic disorders.^[51] The tic-related subtype (in which OCD occurs in an individual with a lifetime history of a chronic tic disorder) may account for as many as 10–40% of OCD cases diagnosed in childhood or adolescence.^[51–64] Even in family-genetics studies where probands with Tourette syndrome (TS) were excluded, at least 10% of the early-onset OC cases were tic-related.^[59,60] Early-onset cases with a personal history of tics typically show a male predominance.^[51,53,57,60–65] Individuals with tic-related OCD are also much more likely to report the presence of antecedent sensory phenomena.^[66–68] Descriptions of sensory phenomena include: localized tactile and muscle–skeletal sensations “just-right” perceptions associated with visual, tactile, or auditory stimuli; feelings of “incompleteness;” and an “urge.” Across cultures, many of the studies of individuals with OCD and a comorbid chronic tic disorder report a high incidence of obsessions concerning symmetry and exactness and related ordering and arranging

compulsions as well as obsessions involving forbidden thoughts.^[53–58,69–71] In contrast, lower rates of OCD symptoms concerning contamination obsessions and cleaning compulsions have frequently been reported, but this may not be true in all cultures.^[63]

In addition to a chronic tic disorder, children with tic-related OCD typically have high rates of disruptive behavior disorders (attention deficit hyperactivity disorder (ADHD) and oppositional defiant disorder) and trichotillomania, as well as other specific and pervasive developmental disorders.^[56,60,62,71–73] Tic-related OCD is highly familial.^[56,59–61,74] For example, Rosario-Campos et al.^[61] studied 325 first-degree relatives of 106 children and adolescents with early-onset OCD (<18 yrs) and found that when the OCD probands were stratified according to presence of a comorbid chronic tic disorder, the case-relatives of the probands with a chronic tic disorder had a significantly higher recurrence risk of OCD: 23.8% (probands with a tic disorder) versus 14.9% (probands without a tic disorder). They also reported that a comorbid diagnosis of tics in the relatives was the best predictor of a diagnosis of OCD (OR = 7.35; 95% CI: 3.79–14.25, $P < .0001$). However, no specific genes have been associated with tic-related OCD.^[74]

Although early-onset OCD tends to respond well to cognitive-behavioral interventions, particularly when combined with selective serotonin reuptake inhibitors (SSRIs),^[32,75,76] it appears that the presence of tics reduces the beneficial effects of SSRI treatment with sertraline in children.^[77] The situation in adults is less clear. Fluvoxamine has been reported to be less effective in OCD patients with a comorbid tic disorder,^[78] while clomipramine had comparable benefits in OCD patients with or without comorbid tics.^[79] In addition, individuals with tic-related OCD may respond better to neuroleptic augmentation than do OCD patients without a personal history of a tic disorder.^[80,81] The course and outcome of tic-related OCD may also be distinctive; characterized by an early peak in OC symptom severity at 12.5 years and followed by an increased likelihood of remission.^[82,83]

Summary and recommendations. The available scientific evidence provides empirical support for the inclusion in DSM-V of a tic-related subtype of OCD. This is a highly familial condition with specific clinical characteristics, such as sensory phenomena and a characteristic clinical course. The strongest argument against this recommendation is that children with both OCD and a chronic tic disorder respond equally well to cognitive-behavioral interventions.^[76,77] However, these tic-related cases may be less likely to respond to SSRIs alone and more likely to benefit from SSRI augmentation with a neuroleptic.^[77,78,80] Although the literature on augmentation trials is about current tics, the value of classifying by lifetime tics is that it encourages a developmental perspective. Indeed, the inclusion of this subtype will require that

practitioners collect information about the individual's developmental history.

Options include applying this subtype broadly to all individuals with OCD who have a personal lifetime history of any tic disorder. However, the inclusion of individuals with a lifetime history of a transient tic-disorder is problematic as data are lacking concerning whether or not such individuals show a differential treatment response to SSRIs alone or to neuroleptic augmentation of SSRI treatment. Finally, a survey of 187 OCD experts from around the world found that 81% supported the inclusion of a tic-related subtype of OCD in DSM-V.^[84]

Early-onset OCD: OCD has been characterized as "early-onset" if the symptoms present before puberty.^[85] The nature of the OC symptoms reported for early-onset OCD across cultures is generally similar to adult-onset OCD.^[70,85-94] However, there are some potentially important differences in sex distribution, natural history, comorbidity, family-genetic data, neuroimaging findings, and treatment response. At puberty, the sex ratio of affected individuals may switch from predominantly males to predominantly females.^[87,88,91,92,95] Early-onset OCD also appears to include a substantial proportion of individuals whose symptoms will remit before early adulthood.^[55,83,87,96] Nevertheless as expected, early-onset OCD has a high rate of comorbidity with chronic tic disorders and ADHD.^[51,71,73,76,77,86,87]

There are conflicting data from family-genetic studies. As reviewed by Pauls,^[74] there have been at least seven family-genetic studies where the ascertainment was through children and/or adolescents with OCD. In these studies, the rate of OCD among relatives of children and adolescents with OCD was increased about tenfold in those studies where comparison to controls was possible. In contrast, when the probands were adults, the rate of OCD among relatives was about two times that among controls. However, at least two studies have found no difference in the rate of OCD in first-degree relatives if an age of onset of 18 years was used as the threshold.^[97,98]

Early-onset OCD also has a distinctive pattern of comorbidity. For example, Hemmings et al.^[99] reported that early onset (≤ 15 yrs. versus > 15 yrs.) of OCD cases was associated with an increased frequency of chronic tic disorders and trichotillomania. Carter et al.^[100] also reported that an early age at onset (< 10 yrs versus ≥ 10 yrs.) in adult and adolescent OCD probands was associated with higher rates of anxiety and depression among case relatives with OCD but not among case relatives without OCD.

A relatively small number of neuroimaging studies have been conducted in children and adolescents with OCD.^[101] To a large extent their findings are consistent with the prevailing frontal-striatal-thalamo-cortical model of the neural substrates derived from studies of adults with OCD.^[102-104] However, there is a suggestion that some differences may exist. For

example, Bussato et al.^[105] found that early-onset OCD cases (age of onset < 10 yrs.) showed decreased blood flow in the right thalamus, left anterior cingulate cortex, and bilateral inferior prefrontal cortex relative to late-onset subjects; however, the early-onset sample included both those with and without tic disorders. Similarly, Gilbert et al. using an fMRI activation paradigm first developed for use in adult OCD subjects, found that OCD symptom provocation in OCD subjects aged 10 to 17 (with age of onset < 14 years) was associated with *reduced* levels of brain activation in the right insula, putamen, thalamus, dorsolateral prefrontal cortex, and left orbitofrontal cortex.^[106] However, the study design cannot distinguish whether the differences between these findings and those in adult OCD subjects were due to differences in age of OCD onset or age of the subjects or to some other difference between the samples or the imaging procedures. Finally, the pattern of treatment response for early-onset OCD, after the tic-related cases are excluded, appears quite similar to that seen in post-pubertal onset OCD.^[76,86-93]

Summary and recommendations. While it is possible that individuals with early-onset OCD may constitute a distinct subtype of OCD, the data, at present, are not compelling enough to include a separate subtype or specifier. Furthermore, studies have used varying definitions of early-onset OCD (in terms of what ages it covers). In addition, it is unclear whether such a subtype should apply to age of onset of subclinical OCD symptoms^[8,107,108] or to the age when the disorder itself begins. Finally, early-onset and the tic-related subtypes seriously confound each other, as chronic tic disorders typically have an age of onset before 10 years of age.^[109]

Given our present state of knowledge, it is recommended that the text accompanying the DSM-V criteria discuss this important issue and encourage practitioners to note age of onset of OCD symptoms as well as the age of onset of OCD per se. Future research is needed to determine whether early-onset OCD, in the absence of a chronic tic disorder, is a valid subtype.

Pediatric Autoimmune Neuropsychiatric Disorders Associated with *Streptococcus* (PANDAS): It has been hypothesized that some susceptible individuals develop OCD symptoms and tic disorders as a result of post-infectious autoimmune processes. Infections with group A beta hemolytic streptococci (GABHS) have been hypothesized to be responsible. Swedo et al.^[43] have proposed that this subgroup, identified by the acronym PANDAS, follows a unique "saw tooth" waxing and waning clinical course that is closely temporally linked to GABHS infections.

Perhaps the strongest evidence that GABHS may be involved in the onset of TS and OCD comes from a recent report by Mell et al.^[110] This is a case-control study of 144 children who received their first diagnosis of OCD, TS, or tic disorder within set time interval (3 months to 1 yr.). Patients with OCD, TS, or tic

disorder were significantly more likely than controls to have had streptococcal infection in the 3 months before onset date. The risk was highest among children with multiple streptococcal infections within 12 months (OR: 3.10; 95% CI: 1.77, 8.96). These findings were recently replicated in a national sample with five times the number of cases and controls.^[111]

Prospective longitudinal studies have yielded less compelling data.^[112–115] For example, Kurlan et al.^[114] recently reported equivocal findings from a 2-year long prospective longitudinal study in which 85% of the documented exacerbations were not associated with a new GABHS infection.^[114] It is noteworthy, however, that this study did report a significantly higher rate of GABHS infections among the PANDAS subgroup as well as a higher rate of rheumatic fever in family members in the PANDAS cases. Hounie et al. also recently reported that OCD and OC spectrum disorders are common in the first-degree relatives of individuals with rheumatic fever.^[116]

It has been postulated that GABHS infection must be the initial autoimmune response-inciting event but that subsequent symptom exacerbations can be triggered by other infectious agents. In fact, a number of other precipitants have been identified including the common cold and *Mycoplasma pneumoniae*.^[117,118] A number of immunological mechanisms have been proposed. Molecular mimicry between GABHS and human brain proteins has been the most widely studied mechanism.^[119–127] There is now preliminary evidence that dopamine can directly influence key immunological mechanisms that may be involved in PANDAS and this has led to a new model, as yet unproven, of PANDAS pathogenesis.^[122,124] Brain imaging studies of PANDAS cases have consistently implicated the basal ganglia. Specific findings include the transient enlargement of the striatum and the basal ganglia as a whole.^[125] Efforts to develop animal models of PANDAS have also led to mixed results although one recent study by Yaddanapudi et al. appears quite promising.^[126,127] Finally although immunomodulatory treatments may be effective in treating PANDAS, independent replication of these findings is lacking.^[45] Likewise, the use of prophylactic antibiotics remains controversial, and it appears that PANDAS is frequently diagnosed in the community without the application of rigorous diagnostic criteria.^[46–49] This phenomenon may have resulted in the unwarranted use of antibiotic treatment for tics/OCD without laboratory evidence of infection.^[48]

Summary and recommendations. A growing body of evidence supports the existence of the PANDAS subtype.^[124] However, PANDAS remains a controversial area of science, with a significant fraction of experts doubting its existence.^[49,128] In addition, in a recent survey of 187 OCD experts from around the world only 53% of the respondents endorsed the inclusion of PANDAS as a subtype of OCD in DSM-V.^[84] Options for DSM-V include adding

PANDAS as a subtype of OCD or simply describing it in the text with cautionary statements, particularly with regard to the use of prophylactic antibiotics. Another alternative would be to exclude PANDAS cases from being diagnosed with OCD per se based on criterion E: “the disturbance is not due to the direct physiological effects of a general medical condition.” Although a case can be made that PANDAS is a form of acute local encephalitis of the basal ganglia manifested by a multiplicity of neuropsychiatric symptoms (i.e., emotional lability, separation anxiety, cognitive deficits, dysgraphia, oppositional behaviors, and motoric hyperactivity) in addition to OCD symptoms, we recommend, given our present state of knowledge, that exclusionary criterion E not be used. We further recommend that PANDAS be discussed in the text.

OC SYMPTOM DIMENSIONS

We next consider whether or not incorporating OCD symptom dimension specifiers in DSM-V is warranted in light of the available scientific evidence.^[129] “Symptom dimensions” refer to the thematic content of an individual’s obsessions and related compulsions. Examples include symptoms associated with obsessions of symmetry and related ordering compulsions or obsessions involving contamination worries and cleaning compulsions. As reviewed below, a growing body of scientific data indicates that a dimensional approach to OC symptoms may have value in genetic, neurobiological, comorbidity and treatment response studies.^[50,84,130,131]

A major limitation in adopting a dimensional approach had been the lack of assessment tools capable of encompassing the dimensionality of OC symptoms and the burden associated with collecting the data needed to complete the newly developed dimensional rating scales.^[132]

Phenomenology: The first study to factor-analyze the Yale-Brown Obsessive-Compulsive Scale-Symptom Checklist (YBOCS-SC)^[34,35] was that of Baer, who identified three factors accounting for 48% of the variance.^[69] Subsequently, Leckman et al.^[70] evaluated the 13 a priori categories used to group types of obsessions and compulsions in the YBOCS-SC in two large groups of OCD patients totaling over 300 cases. Both data sets yielded nearly identical results that in total accounted for >60% of the variance. The first factor (30% of the variance) included obsessive thoughts and images associated with aggressive, sexual and/or religious content, and related checking compulsions. A second factor accounted for 13.8% of the variance and included OCD symptoms concerning symmetry/ordering. A third factor that accounted for 10.2% of the variance was composed of contamination obsessions and cleaning compulsions. The fourth factor included hoarding obsessions (8.5% of the variance). Subsequently, using confirmatory factor analyses, Summerfeldt et al.^[133] examined four models:

a single-factor, i.e., OCD as a single dimension, a two-factor (i.e., obsessions and compulsions), the three-factor model of Baer,^[69] and the four-factor model of Leckman et al.^[70] Adequate fit was found only for the four-factor model. Two recent studies of OCD symptoms provide strong support for the validity of the hoarding, symmetry/ordering and contamination/cleaning dimensions while leaving in doubt how best to divide obsessions related to over-responsibility for harm as well as aggressive, sexual, and religious obsessions and checking compulsions.^[93,134] In the first study, the participants were 485 adults in the six-site OCD Collaborative Genetics Study.^[134] YBOCS-SC data were factor analyzed at both the individual item and symptom category levels. The item- and category-level factor analyses yielded nearly identical 5-factor solutions. Hoarding, symmetry/ordering and contamination/cleaning dimensions were present in both the item-level and category-level analyses. In addition, aggressive, sexual and religious obsessions constituted a fourth dimension, and over-responsibility for harm obsessions and checking compulsions separated out as a fifth dimension. In contrast, Bloch et al.^[93] performed a systematic meta-analysis that examined the data from 21 studies involving 5,124 individuals with OCD. Stratified meta-analysis was conducted to determine the factor structure of OCD in studies involving children and adults separately. The four factors generated from category-level symptom data were: forbidden thoughts (harm, aggressive, sexual, religious, and somatic obsessions and checking compulsions); symmetry/ordering; contamination/cleaning; and hoarding symptoms. Factor analysis of studies including adults yielded an identical factor structure compared to the overall meta-analysis. Of note is that eight studies in this meta-analysis, involving 1,917 OCD subjects, were from non-English-speaking countries in Europe and Asia, indicating that these symptom dimensions have cross-cultural validity.

Factor analytic approaches have their limitations and other approaches may be more useful in sorting out the clinical heterogeneity of OCD. For example several groups of investigators have begun to use latent class modeling to identify specific OCD subgroups. Results of a first latent class analysis of Y-BOCS symptoms conducted in a large group of OCD patients ($n = 1611$) provides compelling evidence for OCD defined as a single spectrum based on severity or symptom endorsement rates rather than the distinct subgroups of symptoms that have been identified via factor analyses.^[135]

Temporal stability: Large-scale studies using adult subjects identified in both epidemiological and clinical settings provide support for the temporal stability of these symptom dimensions.^[131–133] For example, Mataix-Cols et al.^[137] completed a large study of adult OCD patients who were repeatedly administered the YBOCS-SC over a period of 2 years. For the most

part, the patients maintained their symptoms across follow-up, and the strongest predictor of having a particular symptom was having had that symptom in the past. The partial correlations over the 2-year period were the highest for the contamination/cleaning dimension ($r = .73$), but each of the other dimensions (hoarding, symmetry, aggressive, sexual/religious) had partial correlations of $r = .57$ or higher. Further study is needed to examine the temporal stability of symptom dimension over the course of development.

Comorbidity: Aggressive, sexual, religious, and somatic obsessions, and checking compulsions have been differentially associated with comorbid anxiety disorders and depression.^[139] Patients with prominent OCD symptoms in the symmetry/ordering dimension have been found to be more likely to have tic chronic disorders, bipolar disorder, OCPD, panic disorder, or agoraphobia.^[70–73,139–143] In contrast, OCD patients with prominent contamination/cleaning symptoms were more likely to have an eating disorder.^[139] Finally, hoarding symptoms have been strongly related to the presence and number of all personality disorders, especially from the anxious–fearful cluster.^[142,143] For other disorders, this pattern may in part be due to gender differences. For example, among men, hoarding appears to be associated with GAD and tic disorders, whereas, among women, hoarding was associated with social phobia disorder, post-traumatic stress disorder, BDD, nail biting, and skin picking.^[143]

Twin and family-genetic studies: OC symptom dimensions have rarely been evaluated in the context of twin studies with the one exception being the recent study by van Grootheest et al.^[144] In this study, data from a population sample of 1,383 female twins from the Virginia Twin Registry was examined. Using a 20-item self-report questionnaire (the Padua Inventory) to assess OCD symptoms, the authors performed a factor analysis and identified three factors: rumination, contamination, and checking. These OCD dimensions were analyzed with multivariate genetic models. They found that all OCD symptom dimensions shared variation with a latent common factor, that is, OCD behavior in general. Variation in this common factor was explained by both genes (36%) and environmental factors (64%). Only the contamination/washing dimension appeared to be influenced by specific genes. The results of this twin study could be interpreted as providing support for only the contamination/washing dimension. However, the questionnaire used in this study did not contain any “precision” (symmetry/ordering) or hoarding items.

Family-genetic and affected sibling studies also suggest that familial factors play a role in the expression of OCD, but unlike twin studies they cannot distinguish between environmental versus genetic factors. Alsobrook et al.^[145] were the first to use OCD symptom dimensions in a family-genetic study. They found that the relatives of OCD probands

who had high scores on the aggression/sexual dimension and the symmetry/ordering dimensions were at significantly greater risk for having OCD compared to relatives of probands who had low scores on those factors. This finding for symmetry has been replicated in two subsequent studies.^[146,147] Specifically, the OCD Collaborative Genetics Study Group has reported significant sib-sib intraclass correlations for the hoarding dimension ($P=.001$) and for aggressive, sexual, and religious obsessions and checking compulsions ($P=.002$). Smaller, but still significant, sib-sib intraclass correlations were also found for the contamination/cleaning dimension; ($P=.02$) and the symmetry/ordering dimension ($P=.04$). More recently they did both an item-level and a categorical analysis and found robust sib-sib intraclass correlations for the hoarding and taboo thoughts (aggressive, sexual, and religious obsessions).^[147]

Neuropsychological findings: Neuropsychological studies of OCD to date have been characterized by inconsistent findings, which may be due to small sample sizes, the range of neuropsychological tasks performed, as well as disorder heterogeneity.^[148–153] Relatively few studies have examined the utility of the OCD symptom dimensions, as defined above, as they relate to neuropsychological functions.^[151–153] Lawrence et al.^[151] studied 39 OCD patients and 40 controls using tests of decision-making and set-shifting. OCD patients and controls showed comparable decision-making, but patients with prominent hoarding symptoms showed impaired decision-making. OCD patients as a group had poorer set shifting abilities than controls, and symmetry/ordering symptoms were negatively associated with set shifting.

In vivo neuroimaging studies: Taken as a whole, brain imaging studies strongly link OCD with altered activation of the orbitofrontal cortex, with less consistent involvement of anterior cingulate gyrus, lateral frontal and temporal cortices, caudate nucleus, thalamus, amygdala, and insula.^[102–104,149,150] A growing number of functional imaging studies are now incorporating ratings of OCD symptom dimensions.^[154–161] For example, Phillips et al.^[155] compared OCD patients with mainly contamination/cleaning OCD symptoms to age-matched normal controls while viewing pictures of either normally disgusting scenes or washer-relevant pictures using fMRI. When viewing washing-related pictures, only the patients with higher levels of contamination/cleaning symptoms demonstrated activations in regions implicated in disgust perception, i.e., visual regions and insular cortex. Another recent fMRI study used a symptom provocation paradigm to examine, within the same patients, the neural correlates of washing, checking, and hoarding symptom dimensions of OCD.^[159] Each of these dimensions was mediated by a distinct but partially overlapping neural system. Structural neuroimaging studies also suggest that OC symptom dimensions may be useful. Pujol et al.^[162] reported that

patients with high scores on the aggressive/checking dimension had reduced gray matter (GM) volume in the right amygdala compared to age- and sex-matched control subjects. Another recent study used whole-brain voxel-based morphometry to examine the common and distinct neuroanatomical substrates of the major symptom dimensions of OCD in 55 medication-free patients with OCD and 50 age-matched healthy control subjects.^[163] Scores on the contamination/washing dimension were negatively correlated with GM volumes in the caudate. Scores on the harm/checking dimension were negatively correlated with regional GM volume in bilateral temporal lobes. Scores on the symmetry/ordering dimension were negatively correlated with regional GM volume in right motor cortex, left insula and left parietal cortex and positively correlated with bilateral temporal GM volume. If replicated, these functional and structural findings add significant support for the view that OCD is a heterogeneous disorder, with both overlapping and distinct neural correlates associated with specific symptom dimensions.

A counterpoint to this conclusion is that it should be no surprise that viewing or listening to different kinds of stimuli would activate different brain regions. However, the fact that the degree of activation in specific regions corresponds to the level of OCD symptom severity within a given symptom dimension may point to the existence of partially distinct neural systems associated with specific symptom dimensions.

Treatment Adherence and Response to Behavioral Interventions: In many respects, CBT for OCD is based on a dimensional perspective.^[164] Therapists typically develop a hierarchy of OCD symptoms, which often reflect symptom dimensions, that then guide the therapy. The efficacy of CBT for OCD has been demonstrated in numerous controlled and meta-analytic studies.^[130,131,165–167] While definitive studies have not been undertaken, OCD patients with prominent hoarding symptoms have been consistently found to respond less well to standard CBT.^[130,131,165–167] For example, Mataix-Cols et al.^[166] in a study of 153 individuals who participated in computer versus clinician-guided behavior therapy for OCD found that patients with hoarding were more likely to drop out or improve less. This study also found that after controlling for symptom severity, higher scores on the sexual/religious dimension predicted worse outcome with CBT, especially when the treatment was computer-guided.

Prediction of Treatment Response to Medications: SSRIs have been demonstrated to have superior efficacy to placebo for OCD, particularly at higher doses.^[168,169] However, 40–60% of OCD patients do not have a satisfactory outcome to pharmacotherapy alone. Non-response to treatment in OCD is associated with serious social disability.^[170] These differences in treatment outcome emphasize the heterogeneity of OCD and the need for identifying predictors of

treatment response. While definitive studies have not been undertaken, recent studies have suggested that a symptom-based dimensional approach may prove to be valuable for identifying predictors of treatment outcome. For example, several studies have found that OCD patients with prominent hoarding symptoms respond worse than non-hoarding OCD patients to SRIs (including clomipramine and the SSRIs).^[171–173] However, others have reported that OCD patients with prominent hoarding symptoms responded just as well as other OCD patients to paroxetine.^[174] One of the possible explanations for these mixed results may be the use of different selection criteria and recruitment strategies across studies. At least three groups from Spain, India, and Brazil have reported that the presence of sexual obsessions was a predictor of non-response to SSRIs.^[175–177] There are also data from three separate studies that individuals who score high on the symmetry/ordering dimension tend to respond less well to SSRIs or clomipramine alone.^[178–181] These data are consistent with the finding that individuals with tic-related forms of OCD are likely to benefit from neuroleptic augmentation, as individuals with tic-related OCD frequently report prominent symptoms in the symmetry/ordering domain.^[59,70,130,131] Patients with aggressive obsessions and related checking compulsions, in the absence of sexual/religious symptoms may be among the best responders to pharmacotherapy. For example, Landeros-Weisenberger et al.^[181] evaluated the results from a total of 165 adult OCD subjects who participated in one or more 8-week randomized controlled clinical trials with clomipramine, fluvoxamine, or fluoxetine. All subjects were classified as having major or minor symptoms in specific OC symptom dimensions (cleaning/contamination; aggressive obsessions; sexual/religious obsessions; hoarding; and ordering/symmetry obsessions and compulsions). More than 60% of patients with high scores on the aggressive obsessions, the absence of sexual/religious obsessions were rated as very improved or very much improved after SRI treatment with clomipramine, fluvoxamine, or fluoxetine ($P < .001$). Finally, results from deep brain stimulation studies show that patients with perfectionism, hoarding, or symmetry do not respond well to the treatment, whereas patients with contamination and cleaning behavior show average decreases of 75% on the YBOCS (Denys, Personal Communication, 2009).

Summary and preliminary recommendations. OCD is a clinically heterogeneous condition. This heterogeneity can reduce the power and obscure the findings from natural history studies to genome scans, neuroimaging, and clinical trials. Although categorical approaches are critical to selecting the group to be studied, i.e., development of inclusion/exclusion criteria, dimensional approaches may help clinicians appreciate variations in symptom presentation from patient to patient. If these variations can be well defined and are associated with

distinctive patterns of comorbidity, recurrence risk in families, areas of brain activation, and most importantly a differential treatment response, then their inclusion in DSM-V may be justified. There is substantial evidence that this approach may also be useful in guiding initial treatment choices as well as the subsequent management of individual patients. The strongest evidence supports the validity and clinical utility of the symmetry/ordering and contamination/cleaning, and hoarding symptom dimensions.^[50,70,84,130,131] There is less clarity about how best to parse out OC symptoms that relate to “taboo or forbidden” thoughts with aggressive, sexual and/or religious content.^[93,134] In addition, a number of OC symptoms, often termed “miscellaneous” symptoms, have not been fully addressed using a dimensional approach.^[182,183] Likewise, if hoarding disorder is included in DSM-V, the utility of a separate hoarding dimensional specifier for OCD is not clear.^[6]

Although there may be clinical value in identifying the OCD symptom dimensions in order to guide treatment,^[50,70,84,130,131] it is not required in order to establish the diagnosis of OCD. Other disadvantages to including a dimensional approach in the diagnostic criteria include the inherent complexity of dimensions and the burden they would likely place on the practitioner.^[132] Finally, it may be premature to include OC symptom dimensions as specifiers in the OCD criteria as there are likely to be a range of other approaches to OCD, dimensional or otherwise, which will ultimately prove to be more valid or more useful in parsing OCD, but which are based on cognitive-affective processes or perhaps well-validated endophenotypes.^[135,149,150,184,185]

In sum, it is recommended that OCD symptom dimensions be described in the text of DSM-V. Whether to introduce OCD symptom dimensions as a specifier as well in the diagnostic criteria remains unresolved, given the issues raised above.

CONCLUSIONS

The following are the diagnostic criteria that, on the basis of this review, we propose at this time for DSM-V. These proposed criteria may change as the DSM-V process progresses:

Preliminary Recommendations for DSM-V Diagnostic Criteria for OCD:

A. Either obsessions or compulsions:

Obsessions as defined by (1) and (2):

1. recurrent and persistent thoughts, urges, or images that are experienced, at some time during the disturbance, as intrusive and unwanted and that usually cause marked anxiety or distress.
2. the person attempts to ignore or suppress such thoughts, urges, or images, or to neutralize them with some other thought or action (i.e., by performing a compulsion).

Compulsions as defined by (1) and (2):

1. repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly
2. the behaviors or mental acts are aimed at preventing or reducing anxiety or distress, or preventing some dreaded event or situation; however, these behaviors or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive

B. The obsessions or compulsions are time consuming (for example, take more than 1 hour a day), or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

D. The content of the obsessions or compulsions is not restricted to the symptoms of another mental disorder (e.g., excessive worries about real life problems in Generalized Anxiety Disorder; preoccupation with food or ritualized eating behavior in an Eating Disorder; hair pulling in Trichotillomania; stereotypies in Stereotypic Movement Disorder; preoccupation with appearance in Body Dysmorphic Disorder; preoccupation with drugs in a Substance Use Disorder; preoccupation with having a serious illness in Hypochondriasis; preoccupation with sexual urges or fantasies in a Paraphilia or compulsive sexual behavior; preoccupation with gambling or other behaviors in behavioral addictions or impulse control disorders; guilty ruminations in Major Depressive Disorder; paranoia or thought insertion in a Psychotic Disorder).^aIf Hoarding Disorder and Skin-Picking Disorder are added to DSM-V, they will also need to be mentioned in criterion C.

Specify whether OCD beliefs are currently characterized by:

1. Good or fair insight: Recognizes that OCD beliefs are definitely or probably not true, or that they may or may not be true
2. Poor insight: Thinks OCD beliefs are probably true
3. Delusional beliefs: Completely convinced OCD beliefs are true

Specify if:

Tic-related OCD: The individual has a personal lifetime history of a chronic tic disorder.

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REFERENCES

1. Flament MF, Whitaker A, Rapoport JL, et al. Obsessive compulsive disorder in adolescence: An epidemiological study. *J Am Acad Child Adolesc Psychiatry* 1988;27:764-771.
2. Fontenelle LF, Mendlowicz MV, Versiani M. The descriptive epidemiology of obsessive-compulsive disorder. *Prog Neuropsychopharmacol Biol Psychiatry* 2006;30:327-337.
3. Ruscio AM, Stein DJ, Chiu WT, Kessler RC. The epidemiology of obsessive-compulsive disorder in the National Comorbidity Survey Replication. *Mol Psychiatry* 2010;15:53-63.
4. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Text rev. Washington, DC: American Psychiatric Association; 2000.
5. International Classification of Diseases. Geneva, Switzerland: World Health Organization; 1992.
6. Mataix-Cols D, Frost RO, Pertusa A, et al. Hoarding disorder: A new diagnosis for DSM-V? *Depress Anxiety*, in press.
7. Rachman S, De Silva P. Abnormal and normal obsessions. *Behav Res Ther* 1978;16:233-248.
8. Fullana M, Mataix-Cols D, Caspi A, et al. Obsessions and compulsions in the community: Prevalence, interference, help-seeking, temporal stability and co-occurring psychiatric conditions. *Am J Psychiatry* 2009;166:329-336.
9. de Bruijn C, Beun S, de Graaf R, et al. Subthreshold symptoms and obsessive compulsive disorders: Evaluating the diagnostic threshold. *Psychol Med* 2009 Sep 7:1-9. [Epub ahead of print].
10. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. Washington, DC: American Psychiatric Association; 1952.
11. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 2nd ed. Washington, DC: American Psychiatric Association; 1968.
12. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 3rd ed. Washington, DC: American Psychiatric Association; 1980.
13. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, DC: American Psychiatric Association; 1994.
14. American Psychiatric Association. DSM-IV Options Book. Washington, DC: American Psychiatric Association; 1991.
15. Kupfer DJ, First MB, Regier DA, eds. A Research Agenda for DSM-V. Washington, DC: American Psychiatric Association; 2002.
16. Phillips KA, Stein DJ, Rauch SL, et al. Should an obsessive-compulsive spectrum grouping of disorders be included in DSM-V? *Depress Anxiety*, in press.
17. Stein DJ, Fineberg NA, Bienvenu OJ, et al. Should OCD be classified as an anxiety disorder in DSM-V? *Depress Anxiety*, in press.
18. Storch EA, Mariaskin A, Murphy TK. Psychotherapy for obsessive-compulsive disorder. *Curr Psychiatry Rep*. 2009;11: 296-301.
19. Kozak MJ, Foa EB. Mastery of Obsessive-compulsive Disorder: A Cognitive-Behavioral Approach. San Antonio: The Psychological Corporation. Harcourt Brace & Company; 1997.
20. Stein DJ, Denys D, Gloster AT, et al. Obsessive-compulsive disorder: Diagnostic and treatment issues. *Psychiatr Clin North Am* 2009;32:665-685.

21. Webster Dictionary Webster's Third New International Dictionary, Unabridged. New York: Merriam-Webster; 2000.
22. Hoogduin K. On the diagnosis of obsessive-compulsive disorder. *Am J Psychother* 1986;40:36–51.
23. Lewis-Fernández R. Editorial: The cultural formulation. *Transcult Psychiatry* 2009;46:379–382.
24. Foa EB, Jenike M, Kozak MJ, et al. Obsessive Compulsive Disorder. In: Widiger TA, Pincus HA, Ross R, First MB, Davis WW, eds. *DSM IV Sourcebook, Volume 2*, pp. 552–575. Washington, DC: American Psychiatric Association, 1996.
25. Boulougouris JC, Rabavilas AD, Stefanis C. Psychophysiological responses in obsessive-compulsive patients. *Behav Res Ther* 1977;15:221–230.
26. Rabavilas AD, Boulougouris JC. Physiological accompaniments of ruminations, flooding and thought-stopping in obsessive patients. *Behav Res Ther* 1974;12:239–243.
27. Rachman SJ, Hodgson RJ. Obsessions and Compulsions. Englewood Cliffs, NJ: Prentice-Hall; 1980.
28. Kozak MJ, Rossi M, McCarthy PR, Foa EB. Effects of imipramine on the autonomic responses of obsessive-compulsives to auditory tones. *Biol Psychiatry* 1989;26:707–716.
29. Hornsveld RH, Kraaijmaat FW, van Dam-Baggen RM. Anxiety/discomfort and handwashing in obsessive-compulsive and psychiatric control patients. *Behav Res Ther* 1979;17:223–228.
30. Purcell CE, Arrigo BA. *The Psychology of Lust Murder: Paraphilia, Sexual Killing, and Serial Homicide*. New York: Academic Press; 2006.
31. Foa EB, Liebowitz MR, Kozak MJ, et al. Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *Am J Psychiatry* 2005;162:151–161.
32. Pediatric OCD Treatment Study (POTS) Team. Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder: The Pediatric OCD Treatment Study (POTS) randomized controlled trial. *J Am Med Assoc* 2004;292:1969–1976.
33. Mancebo MC, Greenberg B, Grant JE, et al. Correlates of occupational disability in a clinical sample of obsessive-compulsive disorder. *Compr Psychiatry* 2008;49:43–50.
34. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale. I. Development, use, and reliability. *Arch Gen Psychiatry* 1989;46:1006–1011.
35. Goodman WK, Price LH, Rasmussen SA, Mazure C. The Yale-Brown Obsessive Compulsive Scale II: Validity. *Arch Gen Psychiatry* 1989;46:1012–1016.
36. Foa EB, Kozak MJ, Goodman WK, et al. DSM-IV field trial: Obsessive-compulsive disorder. *Am J Psychiatry* 1995;152:90–96.
37. Eisen JL, Phillips KA, Coles ME, Rasmussen SA. Insight in obsessive compulsive disorder and body dysmorphic disorder. *Compr Psychiatry* 2004;45:10–1536.
38. Skoog G, Skoog I. A 40-year follow-up of patients with obsessive-compulsive disorder. *Arch Gen Psychiatry* 1999;56:121–127.
39. Eisen JL, Phillips KA, Baer L, Beer DA, Atala KD, Rasmussen SA. The Brown Assessment of Beliefs Scale: Reliability and validity. *Am J Psychiatry* 1998;155:102–108.
40. Muris P, Merckelbach H, Clavan M. Abnormal and normal compulsions. *Behav Res Ther* 1997;35:249–252.
41. Phillips KA, Wilhelm S, Koran LM, et al. Body Dysmorphic Disorder: Some key issues for DSM-V, *Depress Anxiety*, in press.
42. Crino R, Slade T, Andrews G. The changing prevalence and severity of obsessive-compulsive disorder criteria from DSM-III to DSM-IV. *Am J Psychiatry* 2005;162:876–882.
43. Swedo SE, Leonard HL, Garvey M, et al. Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections: Clinical description of the first 50 cases. *Am J Psychiatry* 1998;155:264–271.
44. Carmin CN, Wiegartz PS, Yunus U, Gillock KL. Treatment of late-onset OCD following basal ganglia infarct. *Depress Anxiety* 2002;15:87–90.
45. Perlmutter SJ, Leitman SE, Garvey MA, et al. Therapeutic plasma exchange and intravenous immunoglobulin for obsessive-compulsive disorder and tic disorders in childhood. *Lancet* 1999;354:1153–1158.
46. Garvey MA, Perlmutter SJ, Allen AJ, et al. A pilot study of penicillin prophylaxis for neuropsychiatric exacerbations triggered by streptococcal infections. *Biol Psychiatry* 1999;45:1564–1571.
47. Snider LA, Lougee L, Slattery M, et al. Antibiotic prophylaxis with azithromycin or penicillin for childhood-onset neuropsychiatric disorders. *Biol Psychiatry* 2005;57:788–792.
48. Gabbay V, Coffey BJ, Babb JS, et al. Pediatric autoimmune neuropsychiatric disorders associated with streptococcus: comparison of diagnosis and treatment in the community and at a specialty clinic. *Pediatrics* 2008;122:273–278.
49. Shulman ST. Pediatric autoimmune neuropsychiatric disorders associated with streptococci (PANDAS): Update. *Curr Opin Pediatr* 2009;21:127–130.
50. Leckman JF, Bloch MH, King RA. Symptom dimensions and subtypes of obsessive-compulsive disorder: A developmental perspective. *Dialogues Clin Neurosci* 2009;11:21–33.
51. Leonard HL, Lenane MC, Swedo SE, et al. Tics and Tourette's disorder: A 2- to 7-year follow-up of 54 obsessive-compulsive children. *Am J Psychiatry* 1992;149:1244–1251.
52. George MS, Trimble MR, Ring HA, Sallee FR, Robertson MM. Obsessions in obsessive compulsive disorder with and without Gilles de la Tourette's syndrome. *Am J Psychiatry* 1993;150:93–97.
53. Leckman JF, Grice DE, Barr LC, et al. Tic-related vs. non-tic-related obsessive compulsive disorder. *Anxiety* 1994;1:208–215.
54. Holzer J, Goodman WK, Price LH, et al. Obsessive-compulsive disorder with and without a chronic tic disorder. A comparison of symptoms in 70 patients. *Br J Psychiatry* 1994;164:469–473.
55. Pauls DL, Alsobrook J, Goodman W, et al. A family study of obsessive compulsive disorder. *Am J Psychiatry* 1995;152:76–84.
56. Zohar AH, Pauls DL, Ratzoni G, et al. Obsessive-compulsive disorder with and without tics in an epidemiological sample of adolescents. *Am J Psychiatry* 1997;154:274–276.
57. Cath DC, Spinhoven P, Hoogduin CAL, et al. Repetitive behaviours in Tourette's syndrome and OCD with and without tics: What are the differences? *Psychiatry Res* 2001;101:171–185.
58. Eichstedt JA, Arnold SL. Childhood-onset obsessive-compulsive disorder: A tic-related subtype of OCD? *Clin Psychol Rev* 2001;21:137–157.
59. Nestadt G, Samuels J, Riddle M, et al. A family study of obsessive-compulsive disorder. *Arch Gen Psychiatry* 2000;57:358–363.
60. Grados MA, Riddle MA, Samuels JF, et al. The familial phenotype of obsessive-compulsive disorder in relation to tic disorders: The Hopkins OCD family study. *Biol Psychiatry* 2001;50:559–565.
61. do Rosario-Campos MC, Leckman JF, Curi M, et al. A family study of early-onset obsessive-compulsive disorder. *Am J Med Genet B Neuropsychiatr Genet* 2005;136B:92–97.
62. Diniz JB, Rosario-Campos MC, Hounie AG, et al. Chronic tics and Tourette syndrome in patients with obsessive-compulsive disorder. *J Psychiatr Res* 2006;40:487–493.

63. Jaisooriya TS, Reddy YC, Srinath S, Thennarasu K. Obsessive-compulsive disorder with and without tic disorder: A comparative study from India. *CNS Spectr* 2008;13:705–711.
64. Nestadt G, Di CZ, Riddle MA, Grados MA, et al. Obsessive-compulsive disorder: Subclassification based on co-morbidity. *Psychol Med* 2009;39:1491–1501.
65. de Mathis A, Diniz JA, Shavitt RG, et al. Early onset Obsessive-compulsive disorder with and without tics. *CNS Spectr* In press.
66. Leckman JF, Walker W K, Goodman WK, et al. "Just right" perceptions associated with compulsive behaviors in Tourette's syndrome. *Am J Psychiatry* 1994;151:675–680.
67. Miguel EC, do Rosário-Campos MC, Prado HS, et al. Sensory phenomena in obsessive-compulsive disorder and Tourette's disorder. *J Clin Psychiatry* 2000;61:150–156.
68. Prado HS, Rosário MC, Lee J, Hounie AG, Shavitt RG, Miguel EC. Sensory phenomena in obsessive-compulsive disorder and tic disorders: A review of the literature. *CNS Spectr* 2008;13:425–432.
69. Baer L. Factor analysis of symptom subtypes of obsessive-compulsive disorder and their relation to personality and tic disorders. *J Clin Psychiatry* 1994;55:18–23.
70. Leckman JF, Grice DE, Boardman J, et al. Symptoms of obsessive-compulsive disorder. *Am J Psychiatry* 1997;154:911–919.
71. Hanna GL. Demographic and clinical features of obsessive-compulsive disorder in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 1995;34:19–27.
72. Stewart SE, Jenike MA, Keuthen NJ. Severe obsessive-compulsive disorder with and without comorbid hair pulling: Comparisons and clinical implications. *J Clin Psychiatry* 2005;66:864–869.
73. Peterson BS, Pine DS, Cohen P, Brook JS. Prospective, longitudinal study of tic, obsessive-compulsive, and attention-deficit/hyperactivity disorders in an epidemiological sample. *J Am Acad Child Adolesc Psychiatry* 2001;40:685–695.
74. Pauls DL. The genetics of obsessive compulsive disorder: A review of the evidence. *Am J Med Genet C Semin Med Genet* 2008;148:133–139.
75. Geller DA. Obsessive-compulsive and spectrum disorders in children and adolescents. *Psychiatr Clin North Am* 2006;29:353–370.
76. Storch EA, Merlo LJ, Larson MJ, et al. Impact of comorbidity on cognitive-behavioral therapy response in pediatric obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* 2008;47:583–592.
77. March JS, Franklin ME, Leonard H, et al. Tics moderate treatment outcome with sertraline but not cognitive-behavior therapy in pediatric obsessive-compulsive disorder. *Biol Psychiatry* 2007;61:344–347.
78. McDougle CJ, Goodman WK, Leckman JF, Barr LC, Heninger GR, Price LH. The efficacy of fluvoxamine in obsessive-compulsive disorder: Effects of comorbid chronic tic disorder. *J Clin Psychopharmacol* 1993;13:354–358.
79. Shavitt RG, Belotto C, Curi M, et al. Clinical features associated with treatment response in obsessive-compulsive disorder. *Compr Psychiatry* 2006;47:276–281.
80. Bloch MH, Landeros-Weisenberger A, Kelmendi B, Coric V, Bracken MB, Leckman JF. A systematic review: Antipsychotic augmentation with treatment refractory obsessive-compulsive disorder. *Mol Psychiatry* 2006;11:622–632.
81. Ipser JC, Carey P, Dhansay Y, et al. Pharmacotherapy augmentation strategies in treatment-resistant anxiety disorders. *Cochrane Database Syst Rev* 2006;4:CD005473
82. Bloch MH, Peterson BS, Scahill L, et al. Adulthood outcome of tic and obsessive-compulsive symptom severity in children with Tourette syndrome. *Arch Pediatr Adolesc Med* 2006;160:65–69.
83. Bloch MH, Craiglow BG, Landeros-Weisenberger A, et al. Predictors of early adult outcomes in pediatric-onset obsessive-compulsive disorder. *Pediatrics* 2009;124:1085–1093.
84. Mataix-Cols D, Pertusa A, Leckman JF. Issues for DSM-V: How should obsessive-compulsive disorder be classified? *Am J Psychiatry* 2007;164:1313–1314.
85. Kalra SK, Swedo SE. Children with obsessive-compulsive disorder: are they just "little adults"? *J Clin Invest* 2009;119:737–746.
86. Swedo SE, Rapoport JL, Leonard H, et al. Obsessive-compulsive disorder in children and adolescents. Clinical phenomenology of 70 consecutive cases. *Arch Gen Psychiatry* 1989;46:335–341.
87. Geller DA, Biederman J, Faraone S, et al. Developmental aspects of obsessive compulsive disorder: Findings in children, adolescents, and adults. *J Nerv Ment Dis* 2001;189:471–477.
88. Reddy YC, Srinath S, Prakash HM, et al. A follow-up study of juvenile obsessive-compulsive disorder from India. *Acta Psychiatr Scand* 2003;107:457–464.
89. Mancebo MC, Garcia AM, Pinto A, et al. Juvenile-onset OCD: Clinical features in children, adolescents and adults. *Acta Psychiatr Scand* 2008;118:149–159.
90. Masi G, Millepiedi S, Mucci M, et al. A naturalistic study of referred children and adolescents with obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* 2005;44:673–681.
91. Tükel R, Ertekin E, Batmaz S, et al. Influence of age of onset on clinical features in obsessive-compulsive disorder. *Depress Anxiety* 2005;21:112–117.
92. Janowitz D, Grabe HJ, Ruhrmann S, et al. Early onset of obsessive-compulsive disorder and associated comorbidity. *Depress Anxiety* 2009;26:1012–1017.
93. Bloch MH, Landeros-Weisenberger A, Rosario-Campos MC, et al. Systematic review of the factor structure of obsessive-compulsive disorder. *Am J Psychiatry* 2008;165:1532–1542.
94. Noshirvani HF, Kasvikis Y, Marks IM, et al. Gender-divergent aetiological factors in obsessive-compulsive disorder. *Br J Psychiatry* 1991;158:260–263.
95. Castle DJ, Deale A, Marks IM. Gender differences in obsessive compulsive disorder. *Aust N Z J Psychiatry* 1995;29:114–117.
96. Stewart SE, Geller DA, Jenike M, et al. Long-term outcome of pediatric obsessive-compulsive disorder: A meta-analysis and qualitative review of the literature. *Acta Psychiatr Scand* 2004;110:4–13.
97. Fyer AJ, Lipsitz JD, Mannuzza S, et al. A direct interview family study of obsessive-compulsive disorder. I. *Psychol Med* 2005;35:1611–1621.
98. Lipsitz JD, Mannuzza S, Chapman TF, et al. A direct interview family study of obsessive-compulsive disorder. II. Contribution of proband informant information. *Psychol Med* 2005;35:1623–1631.
99. Hemmings SM, Kinnear CJ, Lochner C, et al. Early- versus late-onset obsessive-compulsive disorder: Investigating genetic and clinical correlates. *Psychiatry Res* 2004; 128:175–182.
100. Carter AS, Pollock RA, Suvak MK, Pauls DL. Anxiety and major depression comorbidity in a family study of obsessive-compulsive disorder. *Depress Anxiety* 2004;20:165–174.
101. MacMaster FP, O'Neill J, Rosenberg DR. Brain imaging in pediatric obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* 2008;47:1262–1272.
102. Saxena S, Rauch SL. Functional neuroimaging and the neuroanatomy of obsessive-compulsive disorder. *Psychiatr Clin North Am* 2000;23:563–586.

103. Harrison BJ, Soriano-Mas C, Pujol J, et al. Altered corticostriatal functional connectivity in obsessive-compulsive disorder. *Arch Gen Psychiatry* 2009;66:1189–1200.
104. Rotge JY, Langbour N, Guehl D, et al. Gray matter alterations in obsessive-compulsive disorder: An anatomic likelihood estimation meta-analysis. *Neuropsychopharmacology* 2009 Nov 4. [Epub ahead of print].
105. Busatto GF, Buchpiguel CA, Zamignani DR, et al. Regional cerebral blood flow abnormalities in early-onset obsessive-compulsive disorder: an exploratory SPECT study. *J Am Acad Child Adolesc Psychiatry* 2001;40:347–354.
106. Gilbert AR, Akkal D, Almeida JR, et al. Neural correlates of symptom dimensions in pediatric obsessive-compulsive disorder: a functional magnetic resonance imaging study. *J Am Acad Child Adolesc Psychiatry* 2009;48:936–944.
107. Evans DW, Leckman JF, Carter A, et al. Ritual, habit, and perfectionism: The prevalence and development of compulsive like behavior in normal young children. *Child Dev* 1997;68:58–68.
108. Zohar AH, Felz L. Ritualistic behavior in young children. *J Abnorm Child Psychol* 2001;29:121–128.
109. Leckman JF, King RA, Cohen DJ. Tics and tic disorders. In: Leckman JF, Cohen DJ, editors. *Tourette's Syndrome Tics, Obsessions, Compulsions—Developmental Psychopathology and Clinical Care*. New York: Wiley; 1998:23–42.
110. Mell LK, Davis RL, Owens D. Association between streptococcal infection and obsessive-compulsive disorder. *Tourette's syndrome, and tic disorder*. *Pediatrics* 2005;116:56–60.
111. Leslie DL, Kozma L, Martin A, et al. Neuropsychiatric disorders associated with streptococcal infection: A case-control study among privately insured children. *J Am Acad Child Adolesc Psychiatry* 2008;47:1166–1172.
112. Luo F, Leckman JF, Katsoyich L, et al. Prospective longitudinal study of children with tic disorders and/or obsessive-compulsive disorder: Relationship of symptom exacerbations to newly acquired streptococcal infections. *Pediatrics* 2004;113:e578–e585.
113. Perrin EM, Murphy ML, Casey JR, et al. Does group A β -hemolytic streptococcal infection increase risk for behavioral and neuropsychiatric symptoms in children? *Arch Pediatr Adolesc Med* 2004;158:848–856.
114. Kurlan R, Johnson D, Kaplan EL, and the Tourette Syndrome Study Group. Streptococcal infection and exacerbations of childhood tics and obsessive-compulsive symptoms: A prospective blinded cohort study. *Pediatrics* 2008;121:1188–1197.
115. Lin H, Williams KA, Katsoyich L, et al. Streptococcal upper respiratory tract infections and psychosocial stress predict future tic and obsessive-compulsive symptom severity in children and adolescents with Tourette syndrome and/or obsessive-compulsive disorder. *Biol Psychiatry* 2009 Oct 13. [Epub ahead of print].
116. Hounie AG, Pauls DL, do Rosario-Campos MC, et al. Obsessive-compulsive spectrum disorders and rheumatic fever: A family study. *Biol Psychiatry* 2007;61:266–272.
117. Hoekstra PJ, Manson WL, Steenhuis M-P, et al. Association of common cold with exacerbations in pediatric but not adult patients with tic disorder: A prospective longitudinal study. *J Child Adolesc Psychopharm* 2005;15:285–292.
118. Ercan TE, Ercan G, Severge B, et al. *Mycoplasma pneumoniae* infection and obsessive-compulsive disease: A case report. *J Child Neurol* 2008;23:338–340.
119. Church AJ, Dale RC, Lees AJ, et al. Tourette's syndrome: A cross sectional study to examine the PANDAS hypothesis. *J Neurol Neurosurg Psychiatry* 2003;74:602–607.
120. Kirvan CA, Swedo SE, Snider LA, Cunningham MW. Antibody-mediated neuronal cell signaling in behavior and movement disorders. *J Neuroimmunol* 2006;179:173–179.
121. Kansy JW, Katsoyich L, McIver KS, et al. Identification of pyruvate kinase as an antigen associated with Tourette syndrome. *J Neuroimmunol* 2006;181:165–176.
122. Cunningham MW, Kirvan C, Brimberg L, et al. Autoimmunity and behavior: Sydenham's chorea and related disorders. *J Neuroimmunol* 2008;203:204.
123. Singer HS, Gause C, Morris C, et al. Serial immune markers do not correlate with clinical exacerbations in pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections. *Pediatrics* 2008;121:1198–1205.
124. Martino D, Dale RC, Gilbert DL, et al. Immunopathogenic mechanisms in Tourette syndrome: A critical review. *Mov Disord* 2009;24:1267–1279.
125. Giedd JN, Rapoport JL, Garvey MA, Perlmutter S, Swedo SE. MRI assessment of children with obsessive-compulsive disorder or tics associated with streptococcal infection. *Am J Psychiatry* 2000;157:281–283.
126. Singer HS, Mink JW, Loisele CR, et al. Microinfusion of antineuronal antibodies into rodent striatum: Failure to differentiate between elevated and low titers. *J Neuroimmunol* 2005;163:8–14.
127. Yaddanapudi K, Hornig M, Serge R, et al. Passive transfer of streptococcus-induced antibodies reproduces behavioral disturbances in a mouse model of pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection. *Mol Psychiatry* 2009 Aug 11. [Epub ahead of print].
128. Gilbert DL, Kurlan RM. PANDAS: Horse or zebra. *Neurology* 2009;73:1252–1253.
129. Regier DA. Dimensional approaches to psychiatric classification: Refining the research agenda for DSM-V: An introduction. *Int J Methods Psychiatr Res* 2007;16:S1–S5.
130. Mataix-Cols D, Rosario-Campos MC, Leckman JF. A multi-dimensional model of obsessive-compulsive disorder. *Am J Psychiatry* 2005;162:228–238.
131. Leckman JF, Rauch SL, Mataix-Cols D. Symptom dimensions in obsessive-compulsive disorder: Implications for the DSM-V. *CNS Spectr* 2007;12:376–387, 400.
132. Rosario-Campos MC, Miguel EC, Quatrano S, et al. The Dimensional Yale-Brown Obsessive-Compulsive Scale (DY-BOCS): An instrument for assessing obsessive-compulsive symptom dimensions. *Mol Psychiatry* 2006;11:495–504.
133. Summerfeldt LJ, Richter MA, Antony MM, Swinson RP. Symptom structure in obsessive-compulsive disorder: A confirmatory factor-analytic study. *Behav Res Ther* 1999;37:297–311.
134. Pinto A, Greenberg BD, Grados MA, et al. Further development of YBOCS dimensions in the OCD collaborative genetics study: Symptoms vs. categories. *Psychiatry Res* 2008;160:83–93.
135. Mathews CA, Andresen M, Cath D, et al. Latent structures and latent classes in obsessive compulsive disorder. Presented at the World Congress of Psychiatric Genetics 2009.
136. Rettew DC, Swedo SE, Leonard HL, et al. Obsessions and compulsions across time in 79 children and adolescents with obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* 1992;31:1050–1056.
137. Mataix-Cols D, Rauch SL, Baer L, Eisen JL, et al. Symptom stability in adult obsessive-compulsive disorder: Data from a naturalistic two-year follow-up study. *Am J Psychiatry* 2002;159:263–268.
138. Fullana MA, Tortella-Feliu M, Caseras X, et al. Temporal stability of obsessive-compulsive symptom dimensions in an

- undergraduate sample: A prospective 2-year follow-up study. *Behav Modif* 2007;31:815–824.
139. Hasler G, LaSalle-Ricci VH, Ronquillo JG, et al. Obsessive-compulsive disorder symptom dimensions show specific relationships to psychiatric comorbidity. *Psychiatry Res* 2005;135:121–132.
 140. Coles ME, Pinto A, Mancebo MC, et al. OCD with comorbid OCDP: A subtype of OCD? *J Psychiatr Res* 2008;42:289–296.
 141. Samuels JF, Bienvenu 3rd OJ, Pinto A, et al. Hoarding in obsessive-compulsive disorder: Results from the OCD Collaborative Genetics Study. *Behav Res Ther* 2007;45:673–686.
 142. Mataix-Cols D, Baer L, Rauch SL, Jenike MA. Relation of factor-analyzed symptom dimensions of obsessive-compulsive disorder to personality disorders. *Acta Psychiatr Scand* 2000;02:199–202.
 143. Wheaton M, Timpano KR, LaSalle-Ricci VH, Murphy D. Characterizing the hoarding phenotype in individuals with OCD: associations with comorbidity, severity and gender. *J Anxiety Disord* 2008;22:243–252.
 144. van Grootheest DS, Boomsma DI, Hettema JM, Kendler KS. Heritability of obsessive-compulsive symptom dimensions. *Am J Med Genet B Neuropsychiatr Genet* 2008;147B:473–478.
 145. Alsobrook II JP, Leckman JF, Goodman WK, et al. Segregation analysis of obsessive-compulsive disorder using symptom-based factor scores. *Am J Med Genet* 1999;88:669–675.
 146. Leckman JF, Pauls DL, Zhang H, et al. Obsessive-compulsive symptom dimensions in affected sibling pairs diagnosed with Gilles de la Tourette syndrome. *Am J Med Genet B Neuropsychiatr Genet* 2003;116:60–68.
 147. Hasler G, Pinto A, Greenberg BD, et al. Familiality of factor analysis-derived YBOCS dimensions in OCD-affected sibling pairs from the OCD Collaborative Genetics Study. *Biol Psychiatry* 2007;61:617–625.
 148. Simpson HB, Rosen W, Huppert JD, et al. Are there reliable neuropsychological deficits in obsessive-compulsive disorder? *J Psychiatr Res* 2006;40:247–257.
 149. Menzies L, Chamberlain SR, Laird AR, et al. Integrating evidence from neuroimaging and neuropsychological studies of obsessive-compulsive disorder: The orbitofronto-striatal model revisited. *Neurosci Biobehav Rev* 2008;32:525–549.
 150. Chamberlain SR, Menzies L, Hampshire A, et al. Orbitofrontal dysfunction in patients with obsessive-compulsive disorder and their unaffected relatives. *Science* 2008;321:421–422.
 151. Lawrence NS, Wooderson S, Mataix-Cols D, et al. Decision making and set shifting impairments are associated with distinct symptom dimensions in obsessive-compulsive disorder. *Neuropsychology* 2006;20:409–419.
 152. Goldman BL, Martin ED, Calamari JE, et al. Implicit learning, thought-focused attention and obsessive-compulsive disorder: a replication and extension. *Behav Res Ther* 2008;46:48–61.
 153. Nedeljkovic M, Kyrios M, Moulding R, et al. Differences in neuropsychological performance between subtypes of obsessive-compulsive disorder. *Aust N Z J Psychiatry* 2009;43:216–226.
 154. Rauch SL, Dougherty DD, Shin LM, et al. Neural correlates of factor-analyzed OCD symptom dimension: A PET study. *CNS Spectr* 1998;3:37–43.
 155. Phillips ML, Marks IM, Senior C, et al. A differential neural response in obsessive-compulsive patients with washing compared with checking symptoms to disgust. *Psychol Med* 2000;30:1037–1050.
 156. Shapira NA, Liu Y, He AG, et al. Brain activation by disgust-inducing pictures in obsessive-compulsive disorder. *Biol Psychiatry* 2003;54:751–756.
 157. van den Heuvel OA, Veltman DJ, Groenewegen HJ, et al. Amygdala activity in obsessive-compulsive disorder with contamination fear: A study with oxygen-15 water positron emission tomography. *Psychiatry Res* 2004;132:225–237.
 158. Saxena S, Brody AL, Maidment KM, et al. Cerebral glucose metabolism in obsessive-compulsive hoarding. *Am J Psychiatry* 2004;161:1038–1048.
 159. Mataix-Cols D, Wooderson S, Lawrence N, et al. Distinct neural correlates of washing, checking, and hoarding symptom dimensions in obsessive-compulsive disorder. *Arch Gen Psychiatry* 2004;61:564–576.
 160. Lawrence NS, An SK, Mataix-Cols D, et al. Neural responses to facial expressions of disgust but not fear are modulated by washing symptoms in OCD. *Biol Psychiatry* 2007;61:1072–1080.
 161. Rauch SL, Wedig MM, Wright CI, et al. Functional magnetic resonance imaging study of regional brain activation during implicit sequence learning in obsessive-compulsive disorder. *Biol Psychiatry* 2007;61:330–336.
 162. Pujol J, Soriano-Mas C, Alonso P, et al. Mapping structural brain alterations in obsessive-compulsive disorder. *Arch Gen Psychiatry* 2004;61:720–730.
 163. van den Heuvel OA, Remijnse PL, Mataix-Cols D, et al. The major symptom dimensions of obsessive-compulsive disorder are mediated by partially distinct neural systems. *Brain* 2009;132:853–868.
 164. Rachman SJ, Hodgson RJ. *Obsessions and Compulsions*. Englewood Cliffs, NJ: Prentice-Hall; 1980.
 165. Abramowitz JS, Franklin ME, Foa EB. Empirical status of cognitive-behavioral therapy for obsessive-compulsive disorder: A meta-analytic review. *Rom J Cogn Behav Psychother* 2002;2:89–104.
 166. Mataix-Cols D, Marks IM, Greist JH, et al. Obsessive-compulsive symptom dimensions as predictors of compliance with and response to behaviour therapy. *Psychother Psychosom* 2002;71:255–262.
 167. Abramowitz JS, Franklin ME, Schwartz SA, Furr JM. Symptom presentation and outcome of cognitive-behavioral therapy for obsessive-compulsive disorder. *J Consult Clin Psychol* 2003;71:1049–1057.
 168. Soomro GM, Altman D, Rajagopal S, Oakley-Browne M. Selective serotonin re-uptake inhibitors (SSRIs) versus placebo for obsessive compulsive disorder (OCD). *Cochrane Database Syst Rev* 2008 Jan 23;CD001765.
 169. Bloch MH, McGuire J, Landeros-Weisenberger A, et al. Meta-analysis of the dose-response relationship of SSRI in obsessive-compulsive disorder. *Mol Psychiatry* 2009 May 26. [Epub ahead of print].
 170. Pallanti S, Quercioli L. Treatment-refractory obsessive-compulsive disorder: Methodological issues, operational definitions and therapeutic lines. *Prog Neuropsychopharmacol Biol Psychiatry* 2006;30:400–412.
 171. Black DW, Monahan P, Gable J, et al. Hoarding and treatment response in 38 nondepressed subjects with obsessive-compulsive disorder. *J Clin Psychiatry* 1998;59:420–425.
 172. Winsberg ME, Cassic KS, Koran LM. Hoarding in obsessive-compulsive disorder: A report of 20 cases. *J Clin Psychiatry* 1999;60:591–597.
 173. Mataix-Cols D, Rauch SL, Manzo PA, et al. Use of factor-analyzed symptom dimensions to predict outcome with serotonin reuptake inhibitors and placebo in the treatment of obsessive-compulsive disorder. *Am J Psychiatry* 1999;156:1409–1416.
 174. Saxena S, Brody AL, Maidment KM, Baxter Jr LR. Paroxetine treatment of compulsive hoarding. *J Psychiatr Res* 2007;41:481–487.
 175. Alonso MP, Menchón JM, Pifarré J, et al. Long-term follow-up and predictors of clinical outcome in obsessive-compulsive

- patients treated with serotonin reuptake inhibitors and behavioral therapy. *J Clin Psychiatry* 2001;62:535-540.
176. Shetti CN, Reddy YC, Kandavel T, et al. Clinical predictors of drug nonresponse in obsessive-compulsive disorder. *J Clin Psychiatry* 2005;66:1517-1523.
 177. Ferrão YA, Shavitt RG, Bedin NR, et al. Clinical features associated to treatment response in obsessive-compulsive disorder. *J Affect Disord* 2006;94:199-209.
 178. Stein DJ, Andersen EW, Overo KF. Response of symptom dimensions in obsessive-compulsive disorder to treatment with citalopram or placebo. *Revista Bras Psiquiatr* 2007;29:303-307.
 179. Stein DJ, Carey PD, Lochner C, et al. Escitalopram in obsessive-compulsive disorder: Response of symptom dimensions to pharmacotherapy. *CNS Spectrums* 2008;6:492-498.
 180. Matsunaga H, Nagata T, Hayashida K, et al. A long-term trial of the effectiveness and safety of atypical antipsychotic agents in augmenting SSRI-refractory obsessive-compulsive disorder. *J Clin Psychiatry* 2009;70:863-868.
 181. Landeros-Weisenberger A, Bloch MH, Pittenger C, Leckman JF. Dimensional predictors of response to SRI pharmacotherapy in obsessive-compulsive disorder. *J Affect Dis* 2009 Jul 2. [Epub ahead of print].
 182. Storch EA, Lack C, Merlo LJ, et al. Associations between miscellaneous symptoms and symptom dimensions: An examination of pediatric obsessive-compulsive disorder. *Behav Res Ther* 2007;45:2593-2603.
 183. Storch EA, McNamara J, Jordan C, et al. Associations between miscellaneous symptoms and symptom dimensions in adults with obsessive-compulsive disorder. *Anxiety Stress Coping* 2008;21:199-212.
 184. McKay D, Abramowitz JS, Calamari JE, et al. A critical evaluation of obsessive-compulsive disorder subtypes: Symptoms versus mechanisms. *Clin Psychol Rev* 2004;24:283-313.
 185. McKay D, Neziroglu F. Methodological issues in the obsessive-compulsive spectrum. *Psychiatry Res* 2009;170:61-65.