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## Should an Obsessive-Compulsive Spectrum Grouping of Disorders Be Included in DSM-V?

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### Abstract

The obsessive-compulsive (OC) spectrum has been discussed in the literature for two decades. Proponents of this concept propose that certain disorders characterized by repetitive thoughts and/or behaviors are related to obsessive-compulsive disorder (OCD), and suggest that such disorders be grouped together in the same category (i.e., grouping, or “chapter”) in DSM. This paper addresses this topic and presents options and preliminary recommendations to be considered for DSM-V. The paper builds upon and extends prior reviews of this topic that were prepared for and discussed at a DSM-V Research Planning Conference on Obsessive-Compulsive Spectrum Disorders held in 2006. Our preliminary recommendation is that an OC-spectrum grouping of disorders be included in DSM-V. Furthermore, we preliminarily recommend that consideration be given to including this group of disorders within a larger supraordinate category of “Anxiety and Obsessive-Compulsive Spectrum Disorders.” These preliminary recommendations must be evaluated in light of recommendations for, and constraints upon, the overall structure of DSM-V.

## Keywords

obsessive compulsive spectrum; obsessive compulsive disorder; classification; DSM-V

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## INTRODUCTION

For the past two decades, the concept of the obsessive-compulsive (OC) spectrum has been discussed in the literature and increasingly studied.<sup>[1–10]</sup> The term “spectrum” has been used to mean many things; in the literature, and in this paper, “OC spectrum” refers to a group of disorders that are presumed to be distinct from, but related to, OCD, and which are characterized by repetitive thoughts and/or behaviors. This concept implies that such disorders might be grouped together in the same supraordinate category (i.e., grouping, or “chapter”) in DSM. DSM-IV does not include a category of OC spectrum disorders (OCSDs); in DSM-IV, candidates for inclusion in this category are classified under anxiety disorders, somatoform disorders, impulse control disorders (ICDs) not elsewhere classified, personality disorders, and disorders usually first diagnosed in infancy, childhood, or adolescence. Hoarding disorder, which is considered in this review, is not in DSM-IV but is a candidate for inclusion in DSM-V.<sup>[11]</sup>

This review will examine the relatedness of putative OCSDs to OCD and to selected other disorders. The goal is to inform deliberations about DSM-V’s organization/structure – namely, whether there is utility to including a category (i.e., grouping or chapter) of OCSDs in DSM-V, and, if so, which disorders might be included in this category. Any approach towards formulating how disorders might be categorized in DSM-V should be informed by several principles. First, the approach should have clinical utility – i.e., be helpful to clinicians in formulating diagnoses, assessing patients, and selecting appropriate interventions, as this is DSM’s primary purpose. It should also have face validity and be evidence-based to the extent possible.

This paper was commissioned by the DSM-V Anxiety, Obsessive-Compulsive 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 paper should be considered preliminary at this time; they do not necessarily reflect the final recommendations or decisions that will be made for DSM-V, as the DSM-V development process is still ongoing.* It is possible that this paper’s recommendations will be revised as additional data and input from experts and the field are obtained. In addition, the categorization of disorders discussed in this review needs to be harmonized with recommendations from other DSM-V workgroups and the DSM-V Task Force for the overall structure of DSM-V.

This paper builds upon and extends prior reviews of this topic that were prepared for and discussed at a DSM-V Research Planning Conference on Obsessive-Compulsive Spectrum Disorders. This conference, attended by 23 scientists from around the world in 2006, was one of 13 DSM-V Research Planning Conferences that focused on the research evidence for revisions of DSM-V and laid groundwork for the formal development of DSM-V. The primary aim of the conference was to review the evidence that certain disorders may be

related to OCD based on examination of similarities and differences in terms of phenomenology, comorbidity, course of illness, and additional validating domains discussed below. Papers from this conference have been published,<sup>[12, 13]</sup> and summaries are available at [www.dsm5.org](http://www.dsm5.org). Because the present review covers many disorders and comparisons among disorders, and because space is limited, all issues cannot be reviewed in detail. Readers are encouraged to refer to publications from this conference, other prior publications on this topic, and papers in this issue on individual disorders and on the relationship between OCD and other anxiety disorders.<sup>[2, 13, 14]</sup>

## STATEMENT OF THE ISSUES

1. Does available research evidence, and consideration of clinical utility and face validity, support inclusion of a category (i.e., grouping, or chapter) of OCSDs in DSM-V?
2. If an OCSD chapter of disorders is included in DSM-V, which disorders should it include? If this category is not included, where might disorders discussed in this review be classified?
3. If DSM-V includes a group of OCSDs, should they be in a separate chapter or classified in the same chapter as another group of disorders?

## SIGNIFICANCE OF THE ISSUES

DSM-IV contains 16 supraordinate categories (i.e., groupings, or chapters) of disorders -- for example, schizophrenia and other psychotic disorders, mood disorders, and anxiety disorders. DSM-I had only three supraordinate categories,<sup>[15]</sup> and DSM-II had ten.<sup>[16]</sup> In DSM-III, the number of categories was increased to 16, reflecting diagnostic classes proposed by Washington University investigators.<sup>[17]</sup> These diagnostic classes were subsequently handed down, with minor modifications, to DSM-III-R and DSM-IV.<sup>[18, 19]</sup> They were based on clinical utility and enhancement of differential diagnosis.

How disorders are grouped together in DSM does not influence caseness (i.e., who receives a particular diagnosis), because groupings do not affect diagnostic criteria. However, how disorders are grouped has important implications. For example, disorders that are classified together in the same section of DSM are generally presumed to be related to one another and thus to perhaps have shared pathophysiology and etiology (although for most disorders, etiology and pathophysiology remain to be determined). Related disorders may be highly comorbid with one another, have an increased prevalence in family members, or have shared evaluation and treatment elements; in such cases, optimally grouping disorders may usefully guide clinical assessment and treatment approaches. Placing disorders in the same category can also enhance diagnosis and differential diagnosis, as clinicians may pay particular attention to differentiating disorders in the same grouping from one another. Not using such groupings – for example, listing disorders randomly or in alphabetical order – would be clinically unhelpful.

## METHODS

This review focuses on disorders commonly considered candidates for inclusion in an OCSD grouping (also see Results section for a discussion of the selection of disorders for this review). We use eleven validators, developed by the DSM-V Spectrum Study Group, to examine similarities and differences between disorders and thus how closely related they appear to be. These validators are: symptom similarity, high comorbidity among disorders, course of illness, familiarity, genetic risk factors, environmental risk factors, neural substrates, biomarkers, temperamental antecedents, cognitive and emotional processing abnormalities, and treatment response. These validators are based upon and extend those proposed by Robins and Guze in 1970 and subsequently by others.<sup>[20, 21]</sup> The field has used them to examine the validity of individual syndromes as well as the relatedness of disorders to other disorders. Most of these validators were considered by the DSM-V Research Planning Conference on the OCSDs. Because space is limited, it is not possible to present comprehensive data on all 11 validators for all of the comparisons considered in this review. Therefore, we focus on the most important findings regarding similarities and differences with OCD and selected other disorders.

We also consider clinical utility. Clinical utility can, to some extent, be differentiated from issues of diagnostic reliability and validity; in the context of diagnostic classification it refers to considerations such as improving conceptualization of disorders, enhancing communication, and helping to aid assessment and choose treatment.<sup>[22]</sup> There have also been efforts in the field, and as part of the DSM-V process, to determine whether application of latent structure statistical methods to nosological information (e.g., factor analysis and latent class analysis) may be helpful in optimizing the structure of the classification system.<sup>[23, 24]</sup> However, these approaches have limitations;<sup>[24, 25]</sup> in addition, data on OCD are only occasionally available in these analyses, and data are lacking on most putative OCSDs.

A literature search used WebofScience, PubMed, PsychINFO, and other relevant databases. An initial broad search was undertaken for articles that directly address the relationship between putative OCSDs, OCD, and other relevant disorders, and how these disorders might be classified. Search terms included “obsessive-compulsive spectrum,” “obsessive compulsive spectrum disorders,” “classification,” and the names of individual disorders. More directed searches used the above validators developed by the DSM-V Spectrum Study Group. There was no time limit to the search; only English language articles were sought. This review additionally summarizes comorbidity and family study data from recent secondary data analyses commissioned by the DSM-V Task Force; this work was also supported by grants from the National Institutes of Health. These data have been reviewed by the DSM-V Obsessive-Compulsive Spectrum Sub-Workgroup (Bienvenu OJ, et al, unpublished data).

## RESULTS

### The Obsessive-Compulsive Spectrum Concept and DSM-V Research Planning Conference

Earlier conceptualizations of the OC spectrum included a broad range and large number of potential members.<sup>[9]</sup> Indeed, many candidate disorders were reviewed and discussed at the DSM-V Research Planning Conference in 2006.<sup>[12, 13, 26]</sup> On the basis of literature reviews presented at the conference, which examined a broad range of disorders and validators, attendees concluded that the OC spectrum concept has merit but recommended that relatively few disorders be included in this category.<sup>[13, 26]</sup> Attendees concluded that research evidence most strongly supports inclusion of body dysmorphic disorder (BDD), Tourette's disorder, and hypochondriasis (although hypochondriasis was not reviewed in detail). They also concluded that available evidence offers some support for inclusion of hoarding (if hoarding is added as a separate disorder in DSM-V), obsessive-compulsive personality disorder (OCPD), and eating disorders (as well as Sydenham's/PANDAS, although PANDAS may be better conceptualized as a possible OCD subtype, rather than a separate disorder.<sup>[27]</sup> There was incomplete agreement about including trichotillomania, and less support for including other grooming/habit disorders such as pathological skin picking. Based on presented evidence, it was recommended that ICDs not be considered OCSDs.

We also mention here a recent survey of 187 OCD experts from around the world, as it is unique and highly relevant to this review.<sup>[28]</sup> These experts concurred with attendees at the DSM-V Research Planning Conference that if a new category of OCSDs is included in DSM-V, it should be kept narrow. Support was greatest for including BDD (72% agreed), trichotillomania (71% agreed), and tic disorders (61% agreed).<sup>[28]</sup> Support for including hypochondriasis (57% agreed) and OCPD (45% agreed) was more mixed. Relatively few experts agreed with including ICDs (33%) (excluding trichotillomania) or eating disorders (28%), and very few agreed with including autism (9%) or addictions (5%). These findings and those from the DSM-V Research Planning Conference influenced the disorders selected for discussion in this review. (Classification of stereotypic movement disorder is discussed in a separate review.<sup>[29]</sup>)

Other recent reviews of this topic have been published; some support the OC spectrum concept, whereas others do not. Storch and colleagues<sup>[30]</sup> discussed a very broad OCSD construct, which included not only the disorders focused on in the present review but also disorders such as ICDs and eating disorders.<sup>[31]</sup> They concluded, based on examination of a number of validators, that OCD has many similarities with other anxiety disorders and that it is premature to remove OCD from the anxiety disorders and classify it in a separate section of broadly defined OCSDs in DSM-V. In contrast, Hollander and colleagues<sup>[14]</sup> were more positive about the value of including a new category of OCSDs in DSM-V. Stein emphasized that there was unlikely to be a single criterion that would allow a decision about this complex matter; instead, good judgment would be needed to weigh a range of considerations, including those pertaining to diagnostic validity and clinical utility.<sup>[32]</sup>

## Theoretical Approaches

Various theoretical approaches have been taken toward the OC spectrum concept. Some approaches have been substantially informed by experience in clinical settings with different treatments. After serotonin reuptake inhibitors were introduced and demonstrated to be selectively efficacious for OCD,<sup>[33]</sup> research assessed their efficacy for various disorders characterized by unwanted repetitive thoughts or behaviors. Some putative OCSDs, such as BDD, may also selectively respond to this medication class.<sup>[34–37]</sup>

In recent decades, a range of additional research has informed the field's views, including research on the phenomenology of putative OCSDs as well as functional brain imaging research, cross-cultural and epidemiological research, and animal research on habits.<sup>[38, 39]</sup> Thus, we are closer to developing a view that is more translational, developmental, and evolutionary, rather than informed primarily by treatment response (which is somewhat non-specific). This kind of evidence, too, was summarized in the DSM-V Research Planning Conference on OCSDs.<sup>[12, 13, 26]</sup>

Animal motoric habits seem fairly analogous to certain human stereotypies (including those in stereotypic movement disorder, intellectual disability, or pervasive developmental disorders) and to tics in patients with tic disorder.<sup>[39]</sup> We might term these behaviors “motoric,” or “lower-order, repetitive behaviors.” These behaviors may also have some phenomenological and psychobiological overlap with hair-pulling in trichotillomania and with certain other body-focused repetitive symptoms (e.g., compulsive skin-picking).<sup>[29]</sup> From a psychological perspective, these behaviors may be undertaken to modulate self-arousal.<sup>[29]</sup>

It is more difficult to develop animal models of the more complex symptoms of OCD and cognitively focused OCSDs;<sup>[40]</sup> we might term these “cognitive,” or “higher-order,” OC symptoms. In humans, OCD is characterized by concerns in several different dimensions, including harm, cleanliness, order/symmetry, and possibly hoarding,<sup>[27]</sup> Candidate OCSDs with a prominent cognitive component focus on concerns about appearance (BDD) and health (hypochondriasis, or health anxiety disorder), and fears of discarding or not having available personally valuable possessions (hoarding disorder). Compulsions may be seen as a means of reducing anxiety induced by triggering obsessions or to neutralize future threat.<sup>[41]</sup> Indeed, there appears to be some overlap between some of these disorders (see below) in terms of symptoms and other validating domains, such as possible involvement of orbitofronto-striatal-thalamic circuitry.

A third group of repetitive behaviors are classified in DSM-IV as ICDs not elsewhere classified, which have also been termed “behavioral addictions.”<sup>[13, 26, 42]</sup> Substance addiction seems to be driven initially by brain reward systems and then becomes habitual in nature; this may also apply to some of the behavioral addictions.<sup>[43]</sup> Whether these disorders should be construed as “compulsive,” “impulsive,” or “impulsive-compulsive” is controversial. There may be some overlap in the underlying psychobiology of OCD and certain behavioral addictions,<sup>[44]</sup> although, as noted above, with the possible exception of trichotillomania, a review of relevant research suggests more differences than similarities

between these disorders and OCD.<sup>[42]</sup> In addition, clinical approaches to assessment and treatment differ.<sup>[43]</sup>

We now turn to a discussion of individual disorders.

### Body Dysmorphic Disorder (BDD)

DSM-IV classifies BDD, a distressing or impairing preoccupation with an imagined or slight defect in appearance, as a somatoform disorder. However, BDD shares features with OCD, social phobia, eating disorders, and major depressive disorder (MDD).<sup>[2, 7, 45–53]</sup> Three published studies directly compared BDD to OCD across a broad range of clinical features,<sup>[54–56]</sup> other studies compared them in selected domains,<sup>[57–64]</sup> and two studies directly compared BDD to eating disorders.<sup>[52, 65]</sup> No published studies to our knowledge have directly compared BDD to other disorders, including somatoform disorders. More comprehensive reviews of BDD's relationship to OCD,<sup>[66]</sup> eating disorders,<sup>[67]</sup> and other disorders<sup>[68]</sup> can be found elsewhere.

**Symptom Similarity**—OCD and BDD are both characterized by recurrent, time-consuming, intrusive, persistent, and unwanted thoughts.<sup>[69]</sup> These thoughts cause anxiety or distress and are usually resisted to at least some extent.<sup>[54]</sup> Nearly all BDD patients perform compulsive behaviors (e.g., mirror checking)<sup>[67, 70, 71]</sup> that are similar to OCD compulsions. They are repetitive, time-consuming, difficult to resist or control, and not pleasurable; are performed intentionally, in response to an obsession; and aim to reduce anxiety or distress.<sup>[54, 66]</sup> Direct comparison studies with the Yale-Brown Obsessive-Compulsive Scale<sup>[72]</sup> and a slightly modified version for BDD<sup>[73]</sup> found that scores for preoccupations/obsessions and compulsive behaviors did not significantly differ for BDD vs OCD, suggesting similarities in these symptoms.<sup>[54, 55]</sup>

However, insight is poorer in BDD than in OCD, with 27–60% of BDD patients currently having delusional beliefs versus only 2% of OCD patients.<sup>[55, 74–76]</sup> Some BDD compulsions do not appear to follow a simple model of anxiety reduction, which is common in OCD.<sup>[77]</sup> Core beliefs in BDD appear to focus more on unacceptability of the self (e.g., being inadequate, worthless, or unlovable);<sup>[78]</sup> such negative self-focused thoughts also occur in social phobia<sup>[79]</sup> and MDD.<sup>[80]</sup> And preliminary data suggest suicidality rates may be higher in BDD than in OCD.<sup>[54, 55]</sup>

Shared features with social phobia include high social anxiety and social avoidance,<sup>[51, 81, 82]</sup> low extraversion,<sup>[83]</sup> embarrassment, and shame.<sup>[50]</sup> However, one study found that BDD was characterized by relatively low levels of fear of being negatively evaluated by others as a person (in a nonphysical sense), which may differ from social phobia.<sup>[68]</sup> And unlike social phobia, BDD usually involves compulsions as a prominent symptom.

Eating disorders and BDD share body image preoccupation, dissatisfaction, and distress, which appear equally severe in both disorders.<sup>[52, 65]</sup> Some BDD patients diet and excessively exercise.<sup>[70, 71]</sup> However, dissatisfaction and preoccupation involve more diverse body areas in BDD.<sup>[52, 65, 70]</sup> And compared to patients with eating disorders, those

with BDD appear to have more negative self-evaluation and self-worth due to appearance concerns, more avoidance of activities due to self-consciousness about appearance, and poorer functioning and quality of life due to appearance concerns.<sup>[52, 65]</sup>

BDD and MDD share low self-esteem,<sup>[84, 85]</sup> similar core beliefs,<sup>[78, 80]</sup> and rejection sensitivity,<sup>[86, 87]</sup> but compulsions do not occur in MDD. BDD appears to have few similarities with somatoform disorders. For example, in BDD, concern about bodily malfunctioning is uncommon,<sup>[67]</sup> and somatic/somatization symptoms are elevated but do not appear higher than in other psychiatric disorders.<sup>[88]</sup> In addition, women with BDD are less alert to being or becoming ill compared to population norms.<sup>[89]</sup>

**Comorbidity**—In studies of BDD's clinical features (n=293 and n=200), a high proportion of BDD subjects have comorbid lifetime OCD (32%–33%),<sup>[71, 90]</sup> Comorbid lifetime MDD (75–76%) and social phobia (37%–39%) are even more common,<sup>[71, 90]</sup> although these disorders have higher base rates than OCD does.<sup>[91, 92]</sup> Lifetime anorexia nervosa or bulimia nervosa occur in 10–15% of BDD subjects; only 2–7% have a comorbid somatoform disorder.<sup>[71, 90]</sup> Conversely, BDD's lifetime prevalence ranges from 3–37% in patients with OCD (average of 15–20% across studies), 8–42% in MDD, 11–13% in social phobia, and, in one study, 39% of hospitalized patients with anorexia nervosa.<sup>[6, 49, 54, 87, 93–100]</sup> In a blinded and controlled study, BDD was more common in OCD subjects than in community controls (Bienvenu et al., unpublished data).<sup>[6]</sup>

Three BDD-OCD comparison studies found no significant differences in lifetime comorbidity for many disorders, but two of the three studies found that subjects with BDD were more likely than those with OCD to have lifetime MDD and substance use disorders.<sup>[54–56]</sup>

**Course of Illness**—BDD's mean age at onset is 16–17.<sup>[70, 71]</sup> Two BDD-OCD comparison studies found no significant difference in age at onset,<sup>[54, 55]</sup> but one study found earlier onset for BDD.<sup>[56]</sup> BDD's mean age at onset is similar to that of anorexia nervosa, somewhat later than that of social phobia, and somewhat earlier than that of MDD and bulimia nervosa.<sup>[92, 101, 102]</sup>

BDD appears to usually be chronic.<sup>[103]</sup> Studies using methods nearly identical to those in a prospective BDD course study<sup>[103]</sup> found similarly low remission rates for OCD, social phobia, and anorexia nervosa,<sup>[104–106]</sup> but higher remission rates for MDD and bulimia nervosa.<sup>[106, 107]</sup> In the BDD study, which examined time-varying associations between BDD and comorbid disorders, change in the status of BDD and MDD were closely linked in time, with MDD improvement predicting BDD remission, and, conversely, BDD improvement predicting MDD remission.<sup>[108]</sup> OCD improvement predicted BDD remission, but BDD improvement did not predict OCD remission; no significant longitudinal associations were found for BDD and social phobia (social phobia and OCD results were less numerically stable). These findings suggest that BDD may be etiologically linked to MDD and OCD. However, BDD does not appear to simply be a symptom of these comorbid disorders, as BDD persisted in a sizable proportion of subjects who remitted from them.<sup>[108]</sup>



**Familiarity**—In a blinded family history study, first-degree relatives (n=325) of BDD and OCD probands did not significantly differ in terms of lifetime OCD, social phobia, MDD, or somatoform disorders.<sup>[54]</sup> In a blinded and controlled family study, BDD was more common in first-degree relatives of OCD probands than in relatives of control probands, whether or not case probands also had BDD.<sup>[6]</sup> Bienvenu et al (unpublished data) replicated this result with a larger sample of case families. The latter studies suggest BDD may be part of a familial OCD spectrum.

**Genetic and Environmental Risk Factors**—In a small candidate gene study of BDD subjects and healthy controls, association was shown for GABA<sub>A</sub>- $\gamma$ 2 (5q31.1-q33.2)<sup>[66]</sup> but not 5-HT1D- $\beta$  (5HT1B), which has been associated with OCD.<sup>[109]</sup> BDD subjects appear to have a high prevalence of childhood abuse and neglect;<sup>[110, 111]</sup> such experiences are found in many disorders, including OCD, MDD, social phobia, and eating disorders<sup>[111–113]</sup> and thus may not substantially inform the question of BDD's relatedness to other disorders. However, in one study, childhood emotional abuse and sexual abuse were more common in BDD than in OCD (28% vs 2%, and 22% vs 6%, respectively).<sup>[111]</sup>

**Neural Substrates**—No neuroimaging studies have directly compared BDD to other disorders. A small MRI study found a leftward shift in caudate volume asymmetry and greater total white matter volume in BDD than control subjects.<sup>[114]</sup> A second small study similarly found greater total white matter volume in BDD relative to controls, as well as smaller orbitofrontal cortex and anterior cingulate and larger thalamic volumes.<sup>[115]</sup> However, a third study found no significant volumetric differences in BDD vs healthy controls.<sup>[116]</sup> These studies' results differ from OCD,<sup>[117]</sup> although the first two studies' findings can be considered partially consistent with conceptualization of BDD as an OCSD.<sup>[114]</sup> A small BDD SPECT study showed relative perfusion deficits in bilateral anterior temporal and occipital regions and asymmetric perfusion in the parietal lobes,<sup>[118]</sup> unlike OCD.

In an fMRI study, while matching photographs of faces, BDD subjects showed greater left hemisphere activity than controls (particularly in lateral prefrontal cortex and temporal lobe) and abnormal amygdala activation.<sup>[119]</sup> In another study, BDD subjects, compared to controls, had relative hyperactivity in left orbitofrontal cortex and bilateral head of the caudate when viewing a photograph of their own face vs. a familiar face.<sup>[120]</sup> This finding provides preliminary evidence of a possible similarity between BDD and OCD in functional neuroanatomy of orbitofrontal-subcortical circuits, which may be associated with obsessive thoughts and compulsive behaviors.<sup>[121, 122]</sup> However, the exaggerated left hemisphere and amygdala activation in BDD differ from most OCD studies; amygdala activation in response to face stimuli has been found in anxiety disorders, including social phobia.<sup>[123]</sup>

**Biomarkers**—In a small study, platelet 5-HT transporter binding density was significantly lower in OCD, BDD, Tourette's disorder, and ICDs than in controls, suggesting a shared abnormality at the level of the pre-synaptic 5-HT transporter.<sup>[61]</sup>

**Temperamental Antecedents**—No longitudinal studies have prospectively examined temperamental antecedents of BDD. Cross-sectional studies, however, indicate that patients

with BDD, OCD, and eating disorders are more perfectionistic than healthy controls.<sup>[62, 124]</sup> Low levels of extraversion and high levels of neuroticism have been found in BDD,<sup>[83]</sup> social phobia,<sup>[79, 125]</sup> and MDD.<sup>[126–129]</sup> Harm avoidance is also higher in BDD, OCD, social phobia, eating disorders, and MDD<sup>[130–135]</sup> than reported for healthy controls, indicating that individuals with these disorders share an increased tendency for intense adverse responses to aversive stimuli and behavioral inhibition in novel situations.<sup>[130–135]</sup>

**Cognitive and Emotional Processing**—BDD, OCD, and anorexia nervosa patients share poor immediate and delayed visual recall on the Rey-Osterreith Complex Figure Test, which is mediated by deficits in organizational strategy.<sup>[136–139]</sup> All three groups tend to focus on details rather than the overall organization of visual stimuli (although BDD was not directly compared to OCD or anorexia). These findings suggest dysfunction in frontal-striatal circuits and prefrontal regions that mediate executive functioning.<sup>[136–139]</sup> However, BDD subjects rate attractive faces as more attractive than do OCD subjects or healthy controls.<sup>[62]</sup> And BDD subjects have more negative and threatening interpretations of ambiguous social and appearance-related information than do OCD subjects.<sup>[58]</sup> Negative interpretation of ambiguous social information has also been found in social phobia.<sup>[140]</sup> In another study, relative to healthy control and OCD subjects, BDD subjects more often misidentified emotional expressions as angry.<sup>[141]</sup>

**Treatment Response**—BDD and OCD share preferential response to SRIs as monotherapy<sup>[34–37]</sup> (although very preliminary open-label studies raise the possibility that BDD might also respond to venlafaxine and the anti-epileptic medication levetiracetam as monotherapy).<sup>[142, 143]</sup> Both BDD and OCD require higher SRI doses than those typically needed for MDD (although dose finding studies have not been done in BDD).<sup>[33, 35]</sup> However, unlike OCD, neuroleptic augmentation of SRIs does not appear efficacious for BDD (although data are limited).<sup>[35, 144, 145]</sup> While MDD, social phobia, and bulimia nervosa respond well to SRIs, they also respond to other medications, which may not be the case for BDD.<sup>[34, 146–148]</sup> BDD appears to respond more frequently and robustly to SRIs than does anorexia nervosa.<sup>[35, 149]</sup>

CBT containing cognitive restructuring as well as exposure and response prevention appears efficacious for BDD (although research is limited), and it is efficacious for near-neighbor disorders.<sup>[36, 37, 147, 150–155]</sup> Exposure and response prevention are key elements of CBT for OCD, which also often involves cognitive restructuring.<sup>[33, 156]</sup> In social phobia, cognitive restructuring and exposure to feared social situations are core elements; prevention of safety behaviors is used as needed.<sup>[157]</sup> CBT for BDD shares treatment elements with both OCD and social phobia, but clinical experience indicates that because of their poor insight, BDD patients are more difficult to engage in treatment and typically require more intensive cognitive restructuring than OCD patients. CBT for BDD involves greater focus on response prevention than social phobia does because compulsive behaviors are more characteristic of BDD.<sup>[71]</sup> And unlike social phobia and OCD, habit reversal (for BDD-related skin picking and hair plucking) and perceptual retraining are components of some BDD treatments.<sup>[158]</sup> CBT has some, but fewer, shared features with CBT for MDD, eating disorders, and somatoform disorders.

**Conclusions, Clinical Utility, and Preliminary Recommendations**—While more research is needed, BDD appears most similar to OCD and may be part of a familial OC spectrum, consistent with views over the past century that BDD appears related to OCD.<sup>[159–162]</sup> BDD and OCD have some similarities in all of the above domains. Data on symptom similarity, comorbidity, and familiarity offer the strongest support for BDD's relatedness to OCD, although other validators, such as treatment response, also indicate some shared features. Emerging data from some validators, such as neural substrates, suggest a possible relationship between these disorders, although data are still very limited, and more research is needed. However, BDD also has similarities with social phobia and some but fewer similarities with eating disorders and MDD. BDD seems least similar to somatoform disorders; thus, classifying BDD with them does not seem warranted.

Classifying BDD as an OCSD would have clinical utility, as it might prompt clinicians to consider comorbidity of BDD and OCD, a family history of BDD in OCD patients, and use of somewhat similar treatment approaches. However, BDD and OCD have differences that need to be considered (for example, poorer insight in BDD and the need for a somewhat modified treatment approach), especially when treating BDD patients. If OCSDs are classified with anxiety disorders in DSM-V, placing BDD within this grouping would be reasonable, given BDD's similarities to social phobia and its association with social phobia in Japanese psychiatry.<sup>[51, 163]</sup>

### Tourette's Disorder and Chronic Tic Disorders

DSM-IV classifies tic disorders as disorders usually first diagnosed in infancy, childhood, or adolescence.<sup>[164]</sup> This review focuses on Tourette's disorder and chronic tic disorders.

**Symptom Similarity**—Direct comparisons of the symptoms of Tourette's disorder and OCD indicate that these disorders have some similarities.<sup>[165, 166]</sup> Both disorders involve a sequence of events in which a stimulus precedes a largely habitual response. In adolescents and adults with tic disorders, the stimulus is typically a sensory urge followed by a sudden, rapid, recurrent, non-rhythmic, stereotyped motor movement or vocalization.<sup>[167–172]</sup> However, a difference between these disorders is that compulsions are almost always (but not necessarily) performed in response to mental ideational content,<sup>[173]</sup> whereas patients with Tourette's disorder report more sensory phenomena and many fewer cognitive phenomena.<sup>[167]</sup> Additional similarities are that in both TS and OCD, many patients have the need to perform the tic or the compulsion so they are “just right,”<sup>[167, 168]</sup> and a number of ‘sensory phenomena’ frequently accompany OCD.<sup>[164, 166, 174]</sup> At times, it may be difficult to distinguish complex motor tics that appear goal directed from compulsions (which are not always preceded by obsessions). Both tics and obsessions can be suppressed for brief periods of time, but in both cases discomfort and anxiety may be induced.<sup>[166, 167]</sup>

**Comorbidity**—Comorbid lifetime OCD ranges from 23% to 50% in patients with Tourette's disorder.<sup>[49, 174]</sup> Conversely, comorbid lifetime tic disorders affect nearly 30% of OCD patients.<sup>[5]</sup> (Bienvenu, unpublished data). Given that tic disorders affect an estimated 1 in 10,000 of the population, these markedly elevated rates suggest that tic disorders and

OCD may be related. In one study, comorbid tic disorders were significantly more prevalent in OCD patients than in patients with either panic disorder or social phobia.<sup>[5]</sup>

**Course of Illness**—Both Tourette’s disorder and OCD may have early onset (childhood or adolescence) and an extended course.<sup>[175]</sup> The median age of Tourette’s disorder onset is 5.5 years.<sup>[176]</sup> In contrast, while OCD may begin prepubertally, it often begins later in life (e.g., during or after pregnancy),<sup>[5]</sup> with a median age of onset from 13 to 20 years of age.<sup>[49, 174, 175]</sup> Chronic tics, Tourette’s disorder, and OCD symptoms may both wax and wane in severity.<sup>[164–166, 172, 174]</sup> Tic disorders often peak in severity in early adolescence and show a noticeable decline by early adulthood;<sup>[169]</sup> early onset OCD (<10 years) frequently shows a similar course.<sup>[178–180]</sup>

**Familiality**—Family-genetic studies reveal an increased risk of tics, Tourette’s disorder, and OCD among first-degree relatives of Tourette’s disorder probands (compared to control probands), with a higher prevalence of OCD among female relatives (up to 15%) and a higher prevalence of tics (up to 20%) and Tourette’s disorder (up to 17%) among male relatives, independent of whether Tourette’s disorder probands have co-morbid OCD.<sup>[176, 177, 181–186]</sup> Conversely, there is an increased prevalence of tics in relatives of OCD probands (6.2%) compared to relatives of controls (1.7%).<sup>[183]</sup> These findings offer fairly strong support for a familial relationship between Tourette’s disorder, tics, and OCD.

**Genetic and Environmental Risk Factors**—There is evidence that Tourette’s disorder has a genetic basis<sup>[187–190]</sup> and that environmental factors, including psychosocial stress,<sup>[188]</sup> interact with genetic factors to influence severity and course of the syndrome.<sup>[189]</sup> There is some evidence that some specific genes confer vulnerability for TS<sup>[177, 187–190]</sup> and others for OCD,<sup>[182–185]</sup> with both OCD and tic disorders rarely associated with the same single genetic variants.<sup>[188, 189]</sup> Gene pathways involving neurotransmitter (serotonin, dopamine, glutamate) and neurodevelopmental (synaptic, homeobox) domains have been examined, but more complex genetic mechanisms of TS and OCD remain largely unexplored.<sup>[183]</sup> A major gene for TS and/or OCD has not yet been identified, probably owing to both genetic and phenotypic heterogeneity of these disorders.

**Neural Substrates**—Extensive neuroimaging evidence indicates the presence of abnormalities in frontostriatal circuits in children and adults with Tourette’s disorder and OCD.<sup>[191–197]</sup> These frontostriatal circuits are thought to subservise regulatory control, for example, the ability to resist an underlying urge to move or to perform a compulsive behavior.<sup>[194, 197]</sup> On average, caudate nucleus volume is 5% smaller in both children and adults with Tourette’s disorder than in healthy controls, and caudate nucleus volume in childhood is predictive of both future Tourette’s disorder and OCD symptom severity in early adulthood.<sup>[191]</sup>

Functional neuroimaging studies have found that voluntary tic suppression involves deactivation of the putamen and globus pallidus, coupled to partial activation of prefrontal cortex and caudate nucleus.<sup>[192]</sup> Patients with Tourette’s disorder exhibit decreased activity in the caudate and thalamus, together with increased activity of the lateral and medial premotor cortex, supplementary motor areas, anterior cingulate gyrus, dorsolateral-rostral

prefrontal cortex, inferior parietal cortex, putamen, caudate, primary motor cortex, Broca's area, superior temporal gyrus, insula, and claustrum.<sup>[193, 194]</sup> Vocal tics appear to activate prerolandic and postrolandic language regions, as well as insula, caudate, thalamus, and cerebellum. Thus, compared to OCD, Tourette's disorder appears to activate cortical areas other than the orbitofrontal cortex,<sup>[195–198]</sup> especially sensorimotor brain areas. While OCD involves more ventral structures, with hyperactivation of orbitofrontal-caudate-thalamic-cortical areas, circuits involved in Tourette's disorder encompass projections of primary, secondary, and somatosensory cortex to the putamen and dorsolateral caudate nucleus, putamen, and globus pallidus.

**Biomarkers**—There is not yet strong evidence for specific Tourette's disorder biomarkers.

**Temperamental Antecedents**—Only two studies have investigated temperament and character in Tourette's disorder (neither had a prospective longitudinal design), and no studies have directly compared Tourette's disorder and OCD. One study found that harm avoidance tend to be higher in patients with Tourette's disorder and comorbid OCD than in patients with Tourette's disorder alone,<sup>[199]</sup> which was associated with reward deficiency in another study.<sup>[200]</sup>

**Cognitive and Emotional Processing**—Neuropsychological studies of OCD and chronic tic disorders have been characterized by inconsistent findings, which may be due to small sample sizes, the range of neuropsychological tasks performed, and disorder heterogeneity.<sup>[201–206]</sup> Only a few studies<sup>[207]</sup> have directly compared participants with Tourette's disorder and participants with OCD on neurocognitive measures sensitive to frontal-striatal system dysfunction while controlling for each disorder's comorbidity with the other. In one study that compared children with Tourette's disorder alone or with OCD alone,<sup>[207]</sup> neither group demonstrated the pattern of memory deficits expected of frontal-striatal dysfunction (encoding deficits with intact recognition) and reported in the adult OCD literature.<sup>[208]</sup> In another study, direct comparisons between Tourette's disorder and OCD subjects found significantly greater deficits for recognition memory latency and Go/No-go reversal for OCD subjects, and quality of decision-making for Tourette's disorder subjects.<sup>[207, 209]</sup> Like Tourette's disorder patients, OCD patients performed significantly worse than controls in the inhibition of cognitive interference (Stroop test).<sup>[210]</sup> Finally, there is evidence that both TS and OCD are associated with procedural, as opposed to declarative, memory deficits.<sup>[209, 211, 212]</sup>

**Treatment Response**—Tics respond well to neuroleptics (typicals or atypicals), whereas OCD selectively responds to SRIs.<sup>[33, 213]</sup> Nevertheless, serotonergic pathways may play a role in the physiology of Tourette's disorder,<sup>[214, 215]</sup> especially in patients with concomitant sensory phenomena.<sup>[216]</sup> Neuroleptic augmentation is effective for treatment-resistant OCD, especially OCD patients with comorbid tics,<sup>[217–220]</sup> indicating some overlap between Tourette's disorder and OCD treatments. Dopaminergic receptor density (D2) may be decreased<sup>[221]</sup> and the dopaminergic transporter may be increased in the basal ganglia of OCD patients.<sup>[222]</sup> Also, serotonergic neurons produce inhibitory tonus on dopaminergic

neurons in the basal ganglia, suggesting that SRIs can also affect dopaminergic mechanisms.<sup>[39]</sup>

Exposure and response prevention (ERP) is the therapy of choice for OCD.<sup>[223, 224]</sup> ERP is also emerging as a promising treatment for Tourette's disorder.<sup>[225]</sup> The most studied and best-established behavioral technique for Tourette's disorder and other tic disorders is habit reversal training, which consists of awareness training, self-monitoring, relaxation training, competing response training, and contingency management.<sup>[226, 227]</sup> However, there are similarities between ERP and HRT, such as encouraging patients to resist engaging in the compulsion or tic behavior.

Taken together, treatment findings suggest a large number of differences in treatment approaches for OCD and Tourette's disorder, although some elements are similar.

**Clinical Utility, Conclusions, and Preliminary Recommendations**—The above data suggest both similarities and differences between Tourette's disorder and OCD. Certain validators indicate that OCD and Tourette's disorder are closely related. Perhaps the most compelling data comes from family-genetic studies. Comorbidity studies also indicate that these disorders may be related. However, they do have some clinically important differences – e.g., treatment response.

From the perspective of clinical utility, including tic disorders in an OCSD grouping may be useful in encouraging clinicians to assess OCD in Tourette's disorder patients and to assess tics in those with OCD. However, because there are some differences between OCD and Tourette's disorder, and given the early onset of Tourette's disorder and chronic tic disorder, it would also be reasonable for these disorders to remain in a section of disorders usually first diagnosed in infancy, childhood, or adolescence. If the latter category is not included in DSM-V, then a reasonable option would be to include tic disorders in an OCSD grouping, although alternative names for this category would need to be considered, and the text would need to clarify the important distinctions between tic disorders and OCD.

### **Trichotillomania (TTM) and Other Grooming Disorders**

DSM-IV classifies trichotillomania (TTM) as an impulse control disorder (ICD) not otherwise classified. Options for DSM-V include 1) continued inclusion of TTM in a section on ICDs and 2) inclusion of TTM as an OCSD. (This issue is also addressed in a separate review on TTM.<sup>[29]</sup> The relevant evidence base addressing validators for comparisons of TTM and ICDs, and of TTM with OCD and other anxiety disorders, is, however, relatively sparse.

**Symptom Similarity**—A number of studies have undertaken detailed phenomenological comparisons of patients with TTM and with OCD.<sup>[228, 229]</sup> In general, these studies indicate that there are some symptom similarities. For example, TTM involves repetitive behaviors and has important compulsive aspects, including various rituals preceding and following hair-pulling (e.g., mouthing of the hair, biting of the root). However, TTM and OCD have some differences; for example, patients with TTM do not describe obsessions preceding

hair-pulling, and they often obtain a sense of gratification from their behavior. Lochner et al reported that in contrast to OCD, all of their TTM subjects had good insight.<sup>[229]</sup>

Although TTM is currently classified as an ICD, as discussed in more detail in a separate review<sup>[29]</sup> not all patients with TTM describe impulsive hair-pulling (i.e., they do not meet TTM criteria B and C), and meeting criteria B and C is not predictive of increased psychological symptoms, pulling severity, or functional impairment.

Comparisons of subjects with TTM and other body-focused repetitive behavioral symptoms, such as skin-picking, indicate similarity in symptom phenomenology.<sup>[230, 231]</sup>

**Comorbidity**—Several studies indicate that OCD is more prevalent in individuals with TTM than in controls, and, conversely, that TTM is more prevalent in individuals with OCD than in controls<sup>[5, 49, 232]</sup> (Bienvenu et al, unpublished data), although one study did not find this.<sup>[6]</sup> Both TTM and OCD are often comorbid with mood and anxiety disorders, as well as a range of axis II disorders.<sup>[232]</sup> Nevertheless, there are distinctions in comorbidity patterns between the two disorders. For example, Lochner et al reported that in 130 patients with OCD and 49 patients with TTM, lifetime prevalence of a range of anxiety and putative OCDSDs was similar, but depression was more prevalent in OCD (66.9%) than TTM (49.0%), and OCD was more likely to be associated with other psychiatric disorders in general than was TTM.<sup>[229]</sup>

Richter et al compared lifetime rates of TTM in 98 OCD subjects, 86 panic disorder subjects, and 93 social phobia subjects, finding that TTM was significantly more common in OCD (9%) than in the other disorders (1–2%), suggesting that they may be related.<sup>[5]</sup> Skin-picking and tic-related conditions were also significantly more prevalent in OCD than in panic disorder patients.

Lifetime rates of TTM may be elevated in some ICDs such as compulsive sexual behavior (6%)<sup>[233]</sup> and kleptomania (10%),<sup>[234]</sup> although systematic comparisons with control groups are needed. However, TTM has relatively low comorbidity with other ICDs, particularly substance use disorders.<sup>[229, 232]</sup> Furthermore, research on other ICDs (e.g., pathological gambling, pyromania, intermittent explosive disorder) has found little if any co-occurrence with TTM.<sup>[235, 236]</sup> TTM is often comorbid with stereotypic behaviors, including skin-picking.<sup>[232, 237]</sup>

**Course of Illness**—Both TTM and OCD may have early onset (e.g., childhood or adolescence) and extended course.<sup>[238]</sup> TTM's mean age of onset is, however, relatively narrow, with the large majority of cases beginning around puberty.<sup>[239]</sup> In contrast, age at onset in OCD has a broader distribution; OCD may begin prepubertally as well as at other times in the life course (e.g., during or after pregnancy).<sup>[240]</sup> For example, in a sample of similarly recruited subjects, mean age of onset in OCD was 19.3 and for TTM was 11.8.<sup>[229]</sup> Age of onset for most ICDs is generally later than that for TTM (adolescence or early adulthood).<sup>[241]</sup> There are few data on long-term outcomes in TTM, but there is some evidence that, as in OCD, these are also somewhat variable.<sup>[238]</sup>

**Familiarity**—Family studies indicate a familial relationship between OCD and TTM, although neither condition appears very common in first-degree relatives of probands with the other condition. For example, a study of 16 TTM patients without OCD found that 5% of 65 case relatives had OCD, compared to 0% of 90 relatives of 18 normal controls.<sup>[242]</sup> Similar and statistically significant findings were reported in a family history study of 22 TTM patients.<sup>[243]</sup>

The few data that exist may also suggest a relationship between TTM and other disorders. Schlosser et al reported that first-degree relatives of TTM probands were significantly more likely to have substance use disorders (21.6% alcohol use disorders and 14.7% drug use disorders) than relatives of non-ill comparison subjects (7.7% alcohol use disorders and 2.2% drug use disorders).<sup>[243]</sup>

Bienvenu et al found an increased rate of any grooming disorder (TTM, skin-picking, or nail-biting) in probands with OCD compared with controls, as well as in first-degree relatives of OCD probands compared with controls; TTM was uncommon in the sample.<sup>[6]</sup> Bienvenu et al. (unpublished data) recently replicated these results using a much larger sample of families; although TTM was not common, the prevalence of TTM was higher in relatives of OCD-affected probands than control relatives, independent of TTM in probands. Skin picking was a more common condition, affecting substantially more OCD case probands than control probands; skin picking was also more common in case than control relatives, and this condition “ran in the families” independently of skin picking in probands and OCD in relatives.

**Genetic and Environmental Risk Factors**—Genetic and environmental risk factors for TTM are not well elucidated. As in OCD, there may be greater vulnerability after early exposure to adversity or trauma,<sup>[230]</sup> but with little evidence that this association is specific. Patients with the same rare gene variants may present with TTM, OCD, or Tourette’s disorder.<sup>[244–246]</sup> Such variants have not been well explored in ICDs or other body-focused repetitive behaviors. It has been hypothesized that common gene variants also share some overlap in OCD and TTM,<sup>[247]</sup> but the evidence-base is too thin to go beyond the speculative.

**Neural Substrates**—OCD has been associated with altered findings in cortico-striatal-thalamic circuitry.<sup>[248]</sup> These circuits also play a role in TTM, but there are fewer studies, and findings have been somewhat inconsistent. For example, left putamen volume was significantly smaller in TTM subjects than in healthy controls in a small study,<sup>[249]</sup> but another study using a similar design did not confirm this finding.<sup>[250]</sup> Additionally, there is limited evidence that TTM involves areas (e.g., amygdalo-hippocampal formation, cerebellum) that have not been strongly implicated in OCD.<sup>[251, 252]</sup>

**Biomarkers**—There have been few studies of biomarkers in TTM and none that directly compare TTM and OCD. Ninan et al found no differences in CSF metabolites in TTM and healthy controls,<sup>[253]</sup> in contrast to a number of OCD studies.<sup>[254]</sup>



**Temperamental Antecedents**—There has been little work on temperament in TTM. A comparative study of the Temperament and Character Inventory in subjects with TTM and OCD showed higher harm avoidance and lower novelty seeking in OCD, although both groups had high harm avoidance compared to published norms.<sup>[229]</sup>

**Cognitive and Emotional Processing**—Several studies have compared neuropsychological findings in OCD and TTM. Although not all data are consistent, findings have often emphasized differences in the pattern of deficits.<sup>[255]</sup> For example, Chamberlain et al reported that impairment in inhibition of motor responses was worse in trichotillomania than in OCD, whereas patients with OCD but not trichotillomania had deficits in cognitive flexibility.<sup>[255]</sup> It is possible that impaired inhibition of motor responses cuts across a number of OCSDs including tic disorders, whereas other neurocognitive deficits are more specific to each of these conditions, but further work is needed to address fully such a hypothesis.

**Treatment Response**—An early trial found that clomipramine (CMI) was more effective than desipramine in TTM, mirroring findings in OCD.<sup>[195, 256]</sup> There is also a small literature suggesting that augmentation of SRIs with dopamine blockers may be useful in TTM, as in OCD.<sup>[257, 258]</sup> Nevertheless, there appear also to be important differences in pharmacotherapy response, including inconsistent evidence in TTM for efficacy of the SRIs, and less evidence of maintained response to either clomipramine or SSRIs.<sup>[259]</sup> It is possible that TTM, and perhaps certain body-focused repetitive behaviors (such as skin picking), respond to agents that are ineffective in OCD – e.g., low-dose atypical neuroleptics as monotherapy or n-acetylcysteine, although data are not definitive.<sup>[260, 261]</sup> TTM may also respond to some interventions used for other ICDs (e.g., naltrexone),<sup>[258]</sup> although available data are too limited to draw definitive conclusions. ERP is efficacious for OCD, whereas habit reversal is efficacious for TTM, Tourette's disorder, and body-focused repetitive behavioral problems such as skin picking<sup>[226, 262]</sup> (although, as noted earlier, habit reversal and ERP have some overlapping features).

**Conclusions, Clinical Utility, and Preliminary Recommendations**—As discussed above, there is some degree of overlap between OCD and TTM on a number of validators, although this overlap is partial at best. For example, there is evidence of partial overlap (as well as some distinctions) in the neurocircuitry, family history, and neurogenetics of OCD and TTM (although data are still limited for TTM). In contrast, TTM does not appear closely related to other ICDs or behavioral addictions, such as pathological gambling. For example, there is little evidence of comorbidity of TTM with other ICDs or overlap in underlying psychobiology.

Although more research is needed, TTM may overlap phenomenologically and psychobiologically more with other body-focused repetitive behavioral disorders, such as skin-picking disorder, than with OCD. If body-focused repetitive behaviors are not classified in a separate category in DSM-V, then there is some evidence that they should be categorized as motoric OCSDs.

From the viewpoint of clinical utility there are both advantages and disadvantages of classifying TTM as an OCSD. The DSM-V text should clarify that TTM is not simply OCD, as there are some differences in these disorders' core features, assessment, and treatment.

### Hypochondriasis (HYP)

This review examines hypochondriasis (HYP) and its relationship to OCD and, to a lesser extent, to somatization disorder (SD) and panic disorder. We include SD because DSM-IV classifies both SD and HYP as somatoform disorders, and some studies suggest that they are related. We also discuss panic disorder, as both HYP and panic disorder involve health-related anxiety, and some literature suggests that they have similarities.

**Symptom Similarity**—Several studies have compared HYP and OCD and/or panic disorder. Neziroglu et al (n=16 subjects with HYP vs 22 subjects with OCD) found that while both groups had similar levels of obsessionality, anxiety, and depression, HYP subjects had less compulsivity, less insight, more somatic fear, and greater avoidance.<sup>[263]</sup> Abramowitz et al (n=21 subjects with HYP vs 18 subjects with OCD) found that while the two groups had similar beliefs about the probability of becoming ill, HYP patients had greater health anxiety and more catastrophic beliefs about disease.<sup>[264]</sup> Deacon et al compared HYP (n=23), OCD (n=21), and panic disorder (n=50).<sup>[265]</sup> Compared to OCD, HYP subjects had comparable levels of bodily vigilance and intolerance of uncertainty but greater health anxiety and fewer OC symptoms. Compared to panic disorder, the HYP patients had comparably low levels of OC symptoms and high bodily vigilance but differed in having elevated health anxiety and worse intolerance of uncertainty. The symptom focus for panic disorder was on arousal symptoms leading to feared immediate consequences, whereas the focus for HYP encompassed a broader symptom range related to longer-term consequences. On balance, these studies indicate that while there are areas of overlap between HYP and OCD, particularly in intolerance of uncertainty, there are also important differences. Few studies have directly compared HYP and SD (or subthreshold SD), but evidence indicates that HYP and SD patients share multiple medically unexplained symptoms, but they differ in that, compared to non-somatoform controls and to SD patients, HYP patients have significantly higher levels of fear and conviction of disease.<sup>[266]</sup>

**Comorbidity**—General population studies of current DSM-IV HYP reveal prevalence rates of 0.05%–0.4%.<sup>[267–271]</sup> Lifetime rates of HYP among patients with OCD vary from 8.2% (n=85 OCD) to 15% (n=80) of OCD patients.<sup>[6, 272, 273]</sup> Comorbid HYP is even more common in other disorders, however, with a prevalence of 20% in one primary care study of SD<sup>[274]</sup> and 25–51%<sup>[275–277]</sup> in studies of panic disorder.

In primary care samples, while the prevalence of current OCD among patients with HYP was notably low -- less than 1% -- in three studies,<sup>[278–280]</sup> lifetime comorbidity of OCD in HYP appears higher -- 9.5%.<sup>[280]</sup> OCD is not the most common comorbid disorder among HYP patients. Three primary care studies<sup>[274, 278, 280]</sup> in patients with HYP found increased comorbidity among HYP patients compared to primary care patients without HYP as follows: SD (5.5–20 x increased), panic disorder (6.4 x), OCD (3.7 x), any depressive disorder (2.8 x), GAD (2.6 x), pain disorder (2.5 x), and any anxiety disorder (2.0–2.8 x). Of

note, in three primary care studies (which would render a bias toward somatic presentations),<sup>[274, 278, 280]</sup> comorbidity in comparison to the control sample was consistently far more increased for SD than for any other disorder.

These studies therefore indicate that non-OCD Axis 1 comorbidity is far more common than OCD comorbidity in HYP patients and that among the comorbid disorders the increase in somatization disorder is greatest compared to primary care base rates.

**Course of Illness**—Approximately one-third of patients with HYP no longer meet diagnostic criteria after 5 years.<sup>[281]</sup> While OCD has generally been considered a more chronic disorder, with only a minority of patients (<6%) attaining prolonged remission,<sup>[282–284]</sup> a more favorable long-term outcome was suggested by a naturalistic follow-up of 144 patients with OCD, which found that 48% no longer met diagnostic criteria after 40 years.<sup>[284]</sup> Age of onset is similar for HYP and OCD – a median age of 20 for HYP from one primary care study of DSM-IV HYP<sup>[278]</sup> and 19–23 for OCD from two community studies in the United States.<sup>[92, 285]</sup> Median age of onset for panic disorder is slightly older (age 24 from one community study),<sup>[92]</sup> while that for SD is younger (age 15 in the Epidemiologic Catchment Area study in the U.S.).<sup>[286]</sup> However, studies from clinical OCD samples reveal a bimodal distribution for age of onset (ages 12–14 and ages 20–22).<sup>[287]</sup> Childhood-onset OCD has been reported as having important phenotypic differences from adult-onset OCD -- for example, more comorbidity with tics, somatoform, and eating disorders.<sup>[175]</sup> A bimodal pattern has not been described for age of onset in HYP.

**Familiality**—Noyes et al interviewed 72 first-degree relatives of 19 HYP probands and 97 first-degree relatives of 24 non-HYP probands.<sup>[288]</sup> The prevalence of HYP, HYP symptoms, or OCD was not increased in relatives of HYP probands, although somatization disorder and GAD were increased. Bienvenu et al evaluated 343 first-degree relatives of 80 OCD probands and 300 relatives of 73 non-patient control probands<sup>[6]</sup> and found no significant difference in the prevalence of HYP in the first-degree relatives of the OCD probands vs controls' relative (3% vs 1%). However, in a recent larger family study from the same research group, HYP was significantly more common among first-degree relatives of OCD versus control probands (4% vs 1%) (Bienvenu, unpublished data). In the only twin study of somatoform disorders, only one of the co-twins of the six probands with HYP had a psychiatric disorder (major depression), and none had somatization disorder or an anxiety disorder, including OCD.<sup>[289]</sup>

**Genetic and Environmental Risk Factors**—No molecular genetic studies of HYP have been reported. While the prevalence of early childhood trauma (particularly physical and sexual abuse) is high in the somatoform disorders (hypochondriasis, SD),<sup>[290–292]</sup> it is also high in PD and OCD,<sup>[293]</sup> and no study has directly compared these risk factors in somatoform disorders and anxiety disorders.

**Neural Substrates**—Van den Heuvel et al examined neurocognitive and neuroanatomical correlates of attentional bias in 16 OCD patients, 13 HYP patients, 15 panic disorder patients, and 19 controls using fMRI.<sup>[294]</sup> The HYP group differed from the OCD group (but not the panic group) in cortical activation patterns while completing a Stroop task. HYP and

panic patients' cortical activation pattern was widespread (frontal, striatal, temporal) in response to both OCD- and panic-related words, which is consistent with a more generalized attentional bias in response to threat in HYP and in panic compared to OCD and healthy controls. Only the OCD group activated posterior and ventrolateral brain regions. Comparable challenge or resting functional imaging studies with SD have not been conducted.

**Biomarkers**—No data are available in this domain.

**Temperamental Antecedents**—No published studies have used comparable measures to enable comparisons on temperamental factors.

**Cognitive and Emotional Processing**—No studies have reported broad neurocognitive profiles of patients with HYP. One study suggests attentional factors may differentiate HYP from OCD.<sup>[294]</sup> Compared to 19 healthy controls, Van der Heuvel found that performance on the cognitive Stroop task was unimpaired for HYP (n=14) and panic disorder (n=15) but impaired for OCD (n=18). On an emotional Stroop task, HYP and panic disorder groups both showed greater impairment of performance for panic-related words vs neutral words compared to OCD and healthy controls; no across-group difference was seen for OCD words. On attentional tasks, therefore, HYP and panic disorder patients appear to have more similarities than do HYP and OCD patients. There are no cognitive studies directly comparing HYP and SD to other disorders. A small study of 10 SD patients vs 10 non-SD controls revealed that SD patients scored more poorly on memory, visual-spatial, and attentional tasks.<sup>[295]</sup>

**Treatment Response**—CBT and SRIs are moderately effective for both HYP and OCD.<sup>[33, 296–305]</sup> (Similarities in treatment response need not imply similarity of underlying psychopathology or pathogenesis, however, as both CBT and SRIs are efficacious for a wide variety of psychiatric disorders.) It is unclear whether SRIs are preferentially efficacious for HYP (as is the case for OCD), as some studies suggest that HYP may respond to a broader range of antidepressant medications, although data on this issue are quite limited;<sup>[298]</sup> in particular, no studies have directly compared an SRI to a non-SRI medication in patients with HYP. One long-term double-blind study of fluoxetine for HYP suggested that patients may sustain their response after fluoxetine is discontinued.<sup>[306]</sup> If replicated in larger samples, this would distinguish OCD from HYP, as improvement among patients with OCD has been shown to decline rapidly after medication is discontinued.<sup>[307]</sup> The placebo response rate may also distinguish HYP from OCD (although data are limited for HYP). In the fluoxetine HYP study, 33% of patients given placebo were responders,<sup>[306]</sup> similar to the placebo responder rate in depressive disorders.<sup>[308]</sup> The response rate to placebo in OCD using the same scale is often much lower, closer to 10%,<sup>[309, 310]</sup> suggesting that the pathophysiologic mechanisms that perpetuate each of these disorders may differ. Similar to HYP and OCD, CBT has been shown to be efficacious for syndromal and subsyndromal SD, although specific strategies vary by disorder.<sup>[301, 311]</sup> There are no controlled pharmacologic studies of SD, but controlled studies of somatization using a less restrictive set of criteria (multisomatiform disorder) yielded favorable results for escitalopram,<sup>[312]</sup> opipramol,<sup>[313]</sup>

and St. John's wort,<sup>[314]</sup> whereas in a controlled study venlafaxine was not efficacious in reducing the total score for somatic symptoms.<sup>[315]</sup> Although no study has directly compared the relative responsiveness to treatment of patients with HYP vs somatization disorder, there are controlled studies using either CBT or SSRI therapy among patients with hypochondriasis that suggest that improvement is substantially greater on measures of health anxiety than on measures of somatization.<sup>[297, 306, 316]</sup>

**Clinical Utility, Conclusions, and Preliminary Recommendations**—HYP is a heterogeneous disorder characterized by varying amounts of fear, bodily preoccupation, obsessive thoughts, and disease conviction.<sup>[317, 318]</sup> The relative weight of each of these components guides treatment decisions and helps to determine whether a patient appears to have a disorder within a broader anxiety spectrum, a more specific OC spectrum, a somatization spectrum, or a depressive spectrum.<sup>[318]</sup> One research limitation needs emphasis. All studies that have directly compared HYP and OCD have been done in general psychiatric or anxiety clinics. These studies risk being biased toward an overemphasis on the anxious HYP patient who seeks treatment in a psychiatric setting and may not reflect HYP patients seen in primary care settings, who may be more fixated on the somatic symptoms of HYP. Large epidemiologic community studies are needed to clarify the relative contribution of somatic symptoms and illness anxiety among patients with HYP.

In conclusion, this review suggests that while HYP and OCD have some phenomenologic similarities, the substantial differences between them on almost all of the above validators leads to the conclusion that there may be some overlap between HYP and several anxiety disorders, but there does not appear to be preferential relationship between HYP and OCD. Whether HYP bears a closer relationship to somatization than to anxiety disorders can only be addressed by studies that compare HYP, somatization disorder, and specific anxiety disorders directly; these studies have not been done. Another limitation is the paucity of studies comparing HYP to SD directly, so whether there are more similarities on these validators for HYP and SD than for HYP and anxiety disorders remains an open question. Given these deficits in the literature, the best positioning of HYP in DSM remains uncertain.

Finally, the diagnostic criteria for HYP have been criticized as being overly restrictive and not representative of the larger number of patients with health anxiety.<sup>[278]</sup> If HYP is reconceptualized as not requiring misinterpretation of bodily symptoms, and if greater emphasis is placed on ruminative and fear dimensions, as proposed by some and for which there are emerging data,<sup>[278, 319]</sup> then placing a diagnosis of “Health Anxiety Disorder” in the Anxiety Disorders section would seem heuristically reasonable. At this time, however, given the current DSM-IV criteria for HYP, on balance there appears to be insufficient evidence to justify moving this disorder out of the somatoform section of DSM.

## Hoarding

In DSM-IV-TR, hoarding is one of the eight diagnostic criteria for OCPD, but it can sometimes be considered a symptom of OCD “when hoarding is extreme.”<sup>[164]</sup> As discussed elsewhere,<sup>[11]</sup> however, available data indicate that in a majority of cases problematic hoarding cannot be better accounted for by OCD or OCPD. This has led to the suggestion

that hoarding may be better conceptualized as a separate disorder.<sup>[11]</sup> At the time of this writing, it is unclear whether hoarding will be added to DSM-V as a new disorder (tentatively called hoarding disorder) and, if so, whether it will be in the main part of DSM or an Appendix of Criteria Sets Provided for Further Study. Because these options are being considered, we include hoarding in this review and ask where hoarding might be classified. We review the relationship between hoarding and other ‘near neighbor’ disorders, including OCD, depression, anxiety, personality disorders, and ICDs.

**Symptom Similarity**—As reviewed elsewhere,<sup>[11]</sup> hoarding resembles OCD obsessions in that these patients fear losing important items that they feel they may need in the future or feel emotionally attached to, and the excessive acquisition seen in many patients may resemble OCD compulsions. The avoidance of discarding items is similar to other avoidant behaviors seen in many anxiety disorders. If other people touch or move possessions without permission, this typically provokes distress, which may be similar to and overlap with some symmetry-related obsessions in OCD. Measures of hoarding symptoms are moderately correlated with measures of OCD symptoms in non-clinical and clinical hoarding samples.<sup>[320]</sup> However, hoarding symptoms are as strongly correlated with non-OCD symptoms, like depression and anxiety,<sup>[321, 322]</sup> suggesting a non-specific link with emotional disorders in general.

There are also phenomenological links with ICDs – in particular, compulsive buying, which is widely considered a type of ICD NOS. The ego-syntonic and sometimes pleasurable nature of excessive acquisition in hoarding resembles ICDs.<sup>[320]</sup> Many hoarders feel compelled to collect or acquire free items, and to buy excessively.<sup>[323]</sup> Nearly 75% of hoarders excessively buy, while just over half excessively acquire free things.<sup>[323]</sup> However, not everyone with hoarding problems reports excessive acquisition. On balance, these data indicate that hoarding shares some features with OCD but also with other emotional disorders and ICDs.

**Comorbidity**—Hoarding is relatively frequent in patients with OCD; approximately 20–40% endorse hoarding symptoms, but these symptoms are seldom clinically significant.<sup>[173, 324]</sup> In OCD clinics, severe hoarding occurs in about 5% of cases,<sup>[173, 324]</sup> although it is unknown in how many of these cases hoarding is a consequence of other OCD symptoms, such as contamination or harm fears (i.e., an OCD compulsion) vs. a separate comorbid condition. Conversely, in community-solicited samples of severe hoarders, OCD is present in 17–25% of cases, suggesting a link between hoarding and OCD.<sup>[325–327]</sup> However, other emotional disorders such as depression and anxiety disorders may be even more frequently comorbid with hoarding than OCD is. For example, among 217 patients from the community with a significant hoarding problem, 18% had concurrent OCD (based on non-hoarding symptoms), while 36%, 20%, and 24% had concurrent MDD, social phobia, and GAD, respectively,<sup>[327]</sup> although these latter disorders have higher base rates than OCD, and thus higher rates are expected.

Regarding a possible link between hoarding and ICDs, a high prevalence (about 25–40%) of hoarding has been described in samples of compulsive buyers.<sup>[328]</sup> A recent epidemiological study<sup>[329]</sup> reported significant correlations between measures of hoarding and compulsive

buying, and about two-thirds of those with hoarding also had compulsive buying. Preliminary data also suggest a link with other ICDs. For example, one study found high levels of hoarding symptoms among pathological gamblers.<sup>[330]</sup> Another study<sup>[331]</sup> found a greater frequency of TTM and skin picking among OCD patients with hoarding compared to non-hoarding OCD patients (although TTM and skin picking may be better conceptualized as OCSDs than as ICDs).<sup>[41]</sup>

Hoarding (regardless of whether it co-occurs with OCD) is also frequently comorbid with several personality disorders.<sup>[326, 331–334]</sup> The association with OCPD could be explained to some extent by the overlapping content in diagnostic criteria, although the evidence is mixed.<sup>[11, 335]</sup> Only about a third of hoarders meet criteria for OCPD,<sup>[334]</sup> and hoarders have been found to have no more OCPD traits than controls once the OCPD hoarding criterion is excluded.<sup>[326, 333]</sup> Hoarding severity does not correlate with OCPD severity.<sup>[336]</sup> On balance, there is comorbidity between hoarding, OCD, and other putative OCSDs; other anxiety, mood, personality, and certain other ICDs are at least as likely to be comorbid with hoarding.

**Course of Illness**—Although no longitudinal cohort studies have been done, available data suggest that hoarding typically has a chronic and progressive course.<sup>[336–338]</sup> Several retrospective studies suggest that, like OCD, other anxiety disorders, and ICDs, hoarding symptoms usually first emerge in childhood or early adolescence.<sup>[336–338]</sup> However, unlike these other disorders, hoarding symptoms may only become distressing and need treatment later in life, around the mid-thirties.<sup>[326, 337, 338]</sup> The average age of consultation for hoarding is 45–50.<sup>[335]</sup> Also unlike OCD, where symptom intensity can wax and wane, hoarding appears to have a very stable course.<sup>[338]</sup>

**Familiality**—Hoarding appears to run in families, with about 50% of severe hoarders reporting a relative with hoarding problems.<sup>[336, 339]</sup> In a family study, hoarding and tics were more frequent in first-degree relatives of hoarding than non-hoarding OCD probands.<sup>[331]</sup> In a case-control study, 25.6% of severe hoarders who did not meet diagnostic criteria for OCD had a positive (self-reported) family history of OCD,<sup>[326]</sup> suggesting a potential familial link between hoarding and OCD. Other disorders were not examined, so it is unknown how specific the link with OCD is.

**Genetic and Environmental Risk Factors**—The degree of genetic similarity between hoarding and other disorders is unclear. Genetic studies of hoarding have so far been conducted in patients with other disorders, such as Tourette's disorder or OCD.<sup>[340, 341]</sup> Results have been inconsistent but suggest that hoarding may be etiologically distinct from these disorders.<sup>[335]</sup> The literature on environmental risk factors is very limited. Three retrospective studies reported an increased prevalence of traumatic life events in patients with hoarding, compared with non-hoarding OCD patients.<sup>[342–344]</sup> However, these studies remain inconclusive until samples of severe hoarders who do not meet criteria for OCD are studied.

**Neural Substrates**—A handful of neuroimaging studies of hoarding symptoms in patients with OCD or predominantly without OCD have been conducted.<sup>[345–348]</sup> Hoarding (with or

without OCD) appears to be mediated by fronto-limbic circuits involving the cingulate cortex, ventromedial prefrontal cortex, and limbic structures,<sup>[345–347]</sup> whereas non-hoarding OCD is characterized by involvement of specific orbitofronto-striatal-pallidal-thalamic circuits.<sup>[248]</sup> Based on this preliminary evidence, hoarding appears to have a distinct neural substrate from OCD<sup>[348]</sup> and might share neural substrates with a wide range of emotional disorders, such as PTSD or phobias.<sup>[350]</sup> Comparisons with ICDs are not possible, as very few studies have been done.

**Biomarkers**—No data are available in this domain.

**Temperamental Antecedents**—No longitudinal studies have investigated temperamental antecedents of hoarding. Several studies examined the personality profile of compulsive hoarders who met criteria for OCD, finding that it seems broadly similar to that of patients with a variety of emotional disorders, including OCD, although there are no data directly comparing hoarding as a disorder vs. other disorders.<sup>[335]</sup> As reviewed elsewhere,<sup>[11]</sup> hoarders share some characteristics with OCD, such as increased sense of responsibility (but limited to possessions, rather than harm) and high levels of indecisiveness and perfectionism.<sup>[336, 351]</sup> On the other hand, compulsive hoarding and compulsive buying share certain features, including decision-making difficulties, emotional attachment to objects, and erroneous beliefs about possessions.<sup>[352]</sup>

**Cognitive and Emotional Processing**—A few neuropsychological studies have been conducted in hoarding, suggesting deficits in executive functioning, attention, memory, and categorization,<sup>[353–355]</sup> but it is currently unclear to what extent these deficits are specific to hoarding disorder or shared with other disorders.<sup>[335]</sup>

**Treatment Response**—Hoarding has rarely been studied alone in treatment trials.<sup>[349, 356, 357]</sup> In patients with OCD, hoarding symptoms tend to be less responsive than other OCD symptoms to evidence-based treatments for OCD, that is, ERP<sup>[358, 359]</sup> and SRIs.<sup>[324, 360]</sup> However, some preliminary evidence from uncontrolled trials suggest that community-solicited hoarders may respond to SRIs<sup>[349]</sup> and to adapted CBT protocols, which contain ERP elements plus other CBT techniques not typically used in OCD, such as motivational interviewing and skills training, as well as hoarding-specific cognitive restructuring.<sup>[356, 357]</sup>

**Clinical Utility, Conclusions, and Preliminary Recommendations**—If hoarding becomes a separate disorder in DSM-V, it is unclear where it may be best classified, because it shares features with OCD, other anxiety disorders, and ICDs, particularly compulsive buying. Given the phenomenological and historical link between OCD and hoarding, it may make sense to provisionally list it as an OCS until more research is done.

### **Obsessive-Compulsive Personality Disorder (OCPD)**

We focus primarily on OCPD's relationship to OCD and to other personality disorders (PDs).



**Symptom Similarity**—DSM-IV OCPD is defined as preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency, as manifested by eight specific criteria (of which four or more are needed to make the diagnosis): preoccupation with details, orderliness, and rules; rigidity; perfectionism; excessive work devotion; reluctance to delegate; hypermorality; miserliness; and hoarding. Some of the abnormal cognitions considered to underpin OCD, which may be the focus of CBT, such as preoccupation with orderliness, perfectionism, scrupulosity, and behavioral (need for control) or cognitive (stubbornness) rigidity,<sup>[44, 361, 362]</sup> overlap with OCPD criteria. Thus, DSM-IV warns that the two disorders may be confused.<sup>[164]</sup>

OCPD is differentiated from OCD by the absence of strictly defined obsessions and compulsions.<sup>[164, 363]</sup> However, behavioral manifestations of OCPD traits can have a compulsive quality (i.e., intentional, repetitive, time-consuming, difficult to resist or control, not pleasurable and associated with distress).<sup>[164, 361]</sup> In OCD, obsessions are intrusive, distressing and generally *ego-dystonic*. In contrast, OCPD traits and symptomatic behaviors are considered *ego-syntonic*, as they are viewed as correct.<sup>[364]</sup> Arguably, OCPD may be less impairing than OCD,<sup>[365]</sup> although the full impact of OCPD is not well understood, as the disorder has not received much scientific attention.<sup>[363, 366]</sup>

Although not the DSM-IV definition, personality disorder (PD) may be conceptualized, broadly speaking, as the failure to develop an adaptive self-concept and interpersonal relations.<sup>[365]</sup> Within this framework, OCPD could be considered to represent such a rigid self-concept that the ability to respond adaptively to environmental contingencies, such as unexpected change in routines or the need to prioritize timeliness over perfection, is impaired. Like other PDs, OCPD features are traits rather than “states,” but chronic symptoms, as often seen in OCD, can have a trait-like quality. However, OCPD differs in some respects from other PDs, and in factor analyses forms its own factor.<sup>[367]</sup> In some studies, OCPD has been associated with less functional impairment than other PDs,<sup>[365]</sup> and adaptive aspects have been recognized.<sup>[368]</sup> Furthermore, among some researchers, OCPD increasingly is conceptualized as primarily a disorder of neurocognitive function (see section on cognitive and emotional processing) rather than personality.<sup>[364]</sup>

**Comorbidity**—Compared to population norms, OCPD rates are elevated in individuals with OCD (25–32%)<sup>[369–371]</sup> and BDD (14–28%).<sup>[78, 83]</sup> Although OCPD is not uniquely or preferentially associated with OCSDs (e.g., it is comorbid with MDD (31%),<sup>[372, 373]</sup> panic disorder (17%),<sup>[374]</sup> and eating disorders (20–61%)<sup>[375–378]</sup>), this comorbidity profile resembles that of OCD and OCSDs. Moreover, compared to other PDs, OCPD has been found to be the most commonly comorbid PD with anorexia nervosa-restricting type, binge eating disorder,<sup>[379]</sup> and bipolar disorder,<sup>[372, 380]</sup> disorders that are highly comorbid with OCD.<sup>[381, 382]</sup> Conversely, the prevalence of OCD (20%) is elevated in patients with OCPD, although the majority of OCPD patients do not have comorbid OCD.<sup>[383]</sup> This extensive comorbidity does not favor any one of the many theoretical models proposed to explain the overlap between OCPD and Axis I disorders (reviewed in Shea et al.<sup>[384]</sup>).

OCPD also shares substantial comorbidity with other PDs, at least in clinical samples. In one study,<sup>[385]</sup> a high proportion of OCPD patients (77%) had concurrent PDs, but only

comorbidity with paranoid PD (23%) was significantly higher than expected. Moreover, in a meta-analysis of 33 factor analyses of PD comorbidity, OCPD formed its own factor.<sup>[367]</sup> Thus, comorbidity data are equivocal regarding the placement of OCPD.

**Course of Illness**—Although OCPD personality traits are typically stable over time, DSM-IV OCPD per se may not be (e.g., only 42% of patients diagnosed with OCPD at baseline in one study remained above threshold for the diagnosis at 2-year follow-up<sup>[386]</sup>). There are some similarities between the course of OCPD and OCD, which typically both emerge relatively early in life<sup>[387]</sup> and have a chronic course<sup>[388]</sup> that fluctuates in severity, although a subsample with episodic OCD has also been described.<sup>[228]</sup> A retrospective, long-term study comparing first psychiatric admissions diagnosed with OCD or OCPD showed similar levels of diagnostic stability over up to eight years of follow-up.<sup>[389]</sup> Another study<sup>[384]</sup> failed to find a clear longitudinal association between the course of comorbid anxiety disorder (including OCD) and OCPD, suggesting that the two disorders may share only surface phenomenological similarity rather than common underlying substrates, or that the expression of such substrates is complex and varies over time.

**Familiarity/Genetic and Environmental Risk Factors**—Twin studies suggest high heritability for OCPD.<sup>[200, 390–392]</sup> In one study, OCPD was the only PD to co-occur significantly more often in relatives of OCD probands than in relatives of controls, suggesting a specific shared heritability between OCPD and OCD.<sup>[370]</sup> Another study<sup>[393]</sup> reported a higher incidence of DSM-IV OCPD in parents of pediatric OCD probands compared to parents of healthy children, even after parents with OCD were excluded. Similarly, Bienvenu et al. (unpublished data) found a raised prevalence of OCPD in first-degree relatives of OCD-affected probands, independent of OCPD in probands; notably, OCPD also appeared to “run in” these families independent of OCD itself. However, OCPD traits also may constitute a specific familial risk factor for anorexia nervosa.<sup>[394]</sup> OCPD runs together with tics and OCD in families with PANDAS, hinting at a shared familial vulnerability to post-streptococcal psychiatric sequelae.<sup>[395]</sup> In contrast, statistical modeling of genetic and familial factors found limited shared genetic (11%) and environmental (15%) variance between OCPD and the other cluster C personality disorders.<sup>[386]</sup> Thus, on balance, these data, although not specific to OCD, offer fairly strong support for a familial relationship between OCPD and OCD.

There are very limited data examining the contribution of environmental factors to the development of OCPD. One study<sup>[396]</sup> found that patients with OCPD reported significantly lower levels of parental care and significantly higher levels of overprotection compared to healthy controls and to other psychiatric outpatients, similar to OCD.<sup>[397, 398]</sup>

**Neural Substrates**—No brain-imaging studies have specifically investigated OCPD.

**Biomarkers**—Blunted fenfluramine-mediated prolactin responses have been reported for OCPD and also for depression, anorexia nervosa, and binge-eating disorder,<sup>[399–401]</sup> suggesting similarities in serotonergic processing. Blunted fenfluramine responses have also been reported for OCD, but increased responses have also been reported (reviewed in Fineberg et al.<sup>[402]</sup>). Failure to screen for comorbid OCPD and depression in some of these

studies makes interpretation difficult. In summary, there are only limited data on this topic that do not allow clear conclusions about the relatedness of OCPD to OCD or other disorders.

**Temperamental Antecedents**—High neuroticism scores on the five-factor model of personality (e.g., anxiety, self-consciousness, and vulnerability to stress) characterize family members of individuals with OCD and OCPD, even in the absence of symptoms of mental disorder, suggesting shared temperamental factors for OCD and OCPD.<sup>[370]</sup> However, high neuroticism is a non-specific dimension that exemplifies virtually all psychopathology.<sup>[403]</sup>

**Cognitive and Emotional Processing**—DSM-IV OCPD is characterized by inability to respond flexibly to environmental contingencies. A controlled study (n=20) suggests that patients with OCD and comorbid OCPD have greater cognitive inflexibility on a set-shift paradigm (extra-dimensional set-shift; CANTAB),<sup>[404]</sup> thought to be mediated by prefrontal cortex and associated subcortical brain-circuitry than patients with OCD without comorbid OCPD.<sup>[364]</sup> This abnormality is also present in OCD probands and their unaffected first-degree relatives<sup>[405]</sup> as well as patients with schizo-OCD<sup>[406]</sup> and may represent a neurocognitive endophenotype for disorders sharing neurocircuitry with OCD.

**Treatment Response**—A small randomized placebo-controlled trial suggests OCPD may respond to SRIs.<sup>[407]</sup> In a study of patients with OCD, clomipramine was more efficacious than imipramine in improving scores on a self-rated OCPD inventory, suggesting that SRIs might be preferentially efficacious for OCPD traits,<sup>[408]</sup> however, the results were not clinically confirmed and were based on a completer-analysis rather than the intention-to-treat sample. Borderline PD has also been shown to respond to SRIs.<sup>[409]</sup>

**Conclusions, Clinical Utility, and Preliminary Recommendations**—Integration of OCPD into a broadly defined grouping of OCSDs is supported by some evidence from the above validators, including its comorbidity and relatively specific heritability within families of OCD probands, its possible response to SRIs (although treatment data are very limited, and SRI response is not necessarily specific to OCPD among PDs), and its neurocognitive profile implicating frontostriatal neurocircuitry similar to that in OCD (although data are preliminary). Evidence suggesting notable differences between OCPD and other PDs include the separation of dimensional measures of OCPD from other PDs on factor analysis, and heritability patterns that differentiate OCPD from other cluster C disorders. On the other hand, OCPD differs in some ways from OCD. For example, whereas OCPD is characterized by behaviors that have some phenomenological similarities to OCD compulsions, they lack the ‘ego-dystonic’ quality generally considered a key characteristic of OCD.

While more systematic research would be desirable to fully justify moving OCPD from the PD to an OCSD grouping, we consider that moving OCPD to a grouping of OCSDs in DSM-V, while controversial, would probably improve the clinical recognition and endophenotypic evaluation of this neglected and potentially treatable disorder, and may thereby enable development of effective treatments. However, if PD is conceptualized as a single disorder in DSM-V, as is preliminarily recommended by the Personality and Personality Disorders Work Group, it is not clear how this would be effected. One

possibility is that personality traits commonly considered to comprise OCPD (e.g., perfectionism, rigidity) may be risk factors within the OC spectrum, although this possibility needs to be studied.

## CONCLUSIONS AND PRELIMINARY RECOMMENDATIONS FOR DSM-V

From the perspectives of clinical utility, face validity, and evidence from various validators, we conclude that, on balance, a number of disorders discussed in this review appear more closely related to OCD than to other near-neighbor disorders, and that there is merit to including an OC spectrum group of disorders in DSM-V. Our preliminary recommendation is to include within this category several cognitively focused (“higher-order”) disorders – OCD and BDD -- characterized by both compulsive behaviors and obsessional thoughts. If hoarding is added as a new disorder to the main part of DSM-V (rather than an Appendix), this review suggests that it would be reasonable to categorize it with other OCSDs. We also recommend including motorically focused (“lower-order”) disorders characterized by stereotypic behaviors but not obsessions, which would include TTM and possibly tic disorders, particularly if disorders of childhood/adolescent onset are not retained. Hypochondriasis and OCPD could potentially be included with OCSDs, as they have some similarities to OCD, although evidence for their relatedness to OCD is more mixed and less persuasive. Placement of these two disorders requires further consideration in collaboration with relevant DSM-V Work Groups (Somatic Distress and Personality and Personality Disorders).

These preliminary recommendations are highly congruent with recommendations from the previously noted worldwide survey of OCD experts<sup>[28]</sup> and fairly congruent with those from the DSM-V Research Planning Conference on OCSDs.<sup>[12, 26]</sup> Other ICDs and substance use disorders were not the focus of this review, but we concur with prior recommendations from these sources that these disorders should not be included in an OCSD group of disorders.<sup>[13, 26]</sup>

Several disorders that are not a focus of this review merit comment. Stereotypic movement disorder (SMD), discussed in another review,<sup>[29]</sup> is classified in DSM-IV’s section of disorders usually first diagnosed in infancy, childhood, or adolescence. If DSM-V retains this section, there may be no compelling reasons to move SMD elsewhere. If DSM-V does not retain this section, there may be merit to categorizing SMD as a motoric OCSD. This issue requires further discussion with other relevant DSM-V Work Groups. Skin picking disorder and olfactory reference syndrome are not included in DSM-IV but are candidates for inclusion in DSM-V.<sup>[29, 410]</sup> These conditions have many similarities to cognitive OCSDs (in the case of ORS) or motoric OCSDs (in the case of skin picking disorder).

It should be acknowledged that some disorders preliminarily recommended for inclusion in an OCSD category may not be closely related to one another, although this is probably also the case for other current and potential groupings of disorders in DSM. It should also be acknowledged that while neuroimaging and neurocognitive data support a heuristic focus on frontostriatal circuitry with respect to the pathophysiology of candidate OCSDs, the case is empirically well established only for OCD and Tourette’s disorder. Moreover, there are

some apparent differences in neuroimaging findings across some OCSDs, although these findings could be considered to have some convergence in that they involve various striatal subterritories (motoric vs. cognitive vs. affective) and their corresponding corticostriatal networks. However, such neuroanatomical hypotheses pertaining to OCSDs need further testing.

We believe that a category of OCSDs will increase the diagnostic validity and clinical utility of the classification system. From the perspective of clinical utility, there are advantages to grouping these disorders together – e.g., for purposes of diagnosis and differential diagnosis, given similarities in their clinical features (obsessions/preoccupations and/or driven repetitive behaviors). The repetitive behaviors can involve complex compulsions or simple motoric behaviors, which are often associated with anxiety reduction and/or the regulation of arousal. A category of OCSDs would potentially remind clinicians to rigorously assess the range of repetitive symptoms and obsessional thoughts that can occur in these disorders (this is important, because of high comorbidity among some of them), and to consider similar assessment procedures (for example, standardized symptom severity scales for a number of these disorders have been modeled after the Yale-Brown Obsessive-Compulsive Scale for OCD<sup>[72, 73, 411–413]</sup>).

This approach to classification, however, should not imply that OCSDs are simply subtypes or variants of OCD. Indeed, as noted throughout this paper, candidate OCSDs have some clinically important differences from OCD and from one another. Thus they need to be individually identified in clinical settings and when selecting treatment and monitoring treatment response. The potential for confusion about this point is perhaps the most important possible disadvantage of including OCSDs as a category. It would be essential to clarify this issue in the DSM-V text, to ensure appropriate diagnosis and treatment of OCSDs.

If OCSDs are grouped together in DSM-V, what should this category be called? Many in the field have adopted the term “obsessive-compulsive spectrum disorders,” as it emphasizes the disorders’ core features of obsessions/preoccupations and/or compulsions/repetitive behaviors. However, some might consider this name too “OCD-centric” (although, of note, this name does not include the name of the disorder OCD). Another potential problem is that some DSM users might misinterpret this name to mean that all OCSDs are simply types of OCD rather than being likely related but distinct disorders. Alternative names could be considered; we recommend that any alternative names capture these disorders’ key features and be relatively brief.

Another important question is, if DSM-V includes a group of OCSDs, should they be in their own separate chapter, or should they be classified in the same chapter as another group of disorders -- for example, in a chapter of “anxiety and obsessive-compulsive spectrum disorders”? Supporting the former view, motoric OCSDs appear to have relatively little in common with anxiety disorders. Supporting the latter view, OCD is classified as an anxiety disorder in DSM-IV and has much in common with other anxiety disorders.<sup>[30, 41]</sup> While less researched, BDD, too, appears to have much in common with anxiety disorders, especially social phobia. A combined “anxiety and obsessive-compulsive spectrum chapter”

has the advantage of emphasizing established links between anxiety disorders and certain OCSDs while also emphasizing some important distinctions between them. (The categorization of PTSD is discussed in a separate review [Spiegel et al, in preparation].) The majority of the authors of this review consider the option of a combined, supraordinate category of “anxiety and obsessive-compulsive spectrum disorders” most satisfactory, especially given that the overall number of categories in DSM-V will likely be limited. We further recommend that this supraordinate category contain two subcategories -- one for anxiety disorder and one for OCSDs. This approach has some similarities to that in ICD-10, where OCD and anxiety disorders are separate sub-categories within the same larger, overarching, supraordinate category (“neurotic, stress-related, and somatoform disorders”).<sup>[363]</sup>

This review, and the status of the field’s knowledge, have some limitations. For some disorders, little or no research evidence is available for certain validators, and, for some validators, findings are inconsistent. Direct comparison studies of putative OCSDs to one another and to other disorders are still limited. Many studies do not report effect sizes or another metric reflecting degree of similarity between disorders,<sup>[414]</sup> and this review was qualitatively based (formal meta-analyses were not done). Furthermore, the field does not have clear indicators for how similar disorders must be across validating domains to be considered closely related, whether all validators should be weighted equally, or whether similarities in particular validating domains should be required for spectrum membership.<sup>[415]</sup> The selected validators may not be perfect, and during the DSM-V process, thinking about which validators to employ has evolved. These limitations are relevant to decisions about groupings of disorders across DSM (not just the OC spectrum); some may also be relevant to certain non-psychiatric medical disorders.

There is no perfect way to categorize disorders in DSM-V; the field is not at the point where nature can be carved perfectly at its joints. Ultimately, knowledge of disorders’ etiology and pathophysiology may advance the field to the point where categories with greater validity and utility are possible.<sup>[415, 416]</sup> In the meantime, many disorders across DSM share certain features and also have meaningful differences. Arguments could be made for alternative classification schemes.<sup>[417]</sup> Decisions about how to group disorders in DSM-V must also consider constraints on the overall number of categories that can be included in the manual.

Despite these limitations, research on this issue has substantially advanced relevant knowledge since DSM-IV was published 16 years ago. The challenge for DSM-V is whether it can improve upon the disorder groupings in DSM-IV, which were created before much of the research discussed in this review had been done. An important question is, how problematic is the current placement of putative OCSDs? In other words, how compelling is the need to move them to a different category? BDD appears to have little in common with the somatoform disorders, and thus it seems problematic to leave it there. Hypochondriasis as defined in DSM-IV seems more closely related to other somatoform disorders (or, at least, somatization disorder), and thus keeping it in that group seems less problematic. Similarly, leaving OCPD in the personality disorder section is a reasonable alternative. Tourette’s disorder and tic disorders could remain in a childhood chapter, but it is currently unclear whether this chapter will remain in DSM-V; good options (other than OCSDs) for

classifying tic disorders are not apparent. Leaving TTM with other ICDs is problematic insofar as TTM differs from them in a number of ways and requires a different assessment and treatment approach (although its treatment also differs somewhat from OCD's).

DSM-V's final grouping scheme will have disadvantages as well as advantages, and the text can be helpful here. For example, if BDD is included in a category of Anxiety and Obsessive-Compulsive Spectrum Disorders, the text could remind readers of the overlap with mood disorders and, in some patients, with eating disorders. Even more important, as in DSM-IV, the text should indicate key differences between disorders and their "nearest neighbors," so disorders with some similar features are not confused with one another and misdiagnosed. A good description of differential diagnosis in the text will enhance clinical care by facilitating accurate clinical assessment and, in turn, selection of appropriate treatment.

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## References

1. Stein, DJ.; Hollander, E. Obsessive-compulsive related disorders. Washington, DC: American Psychiatric Press; 1993. The spectrum of obsessive-compulsive related disorders.
2. Castle DJ, Phillips KA. Obsessive-compulsive spectrum of disorders: a defensible construct? *Aust N Z J Psychiatry*. 2006; 40:114–120. [PubMed: 16476128]
3. Stein DJ. Neurobiology of the obsessive-compulsive spectrum disorders. *Biol Psychiatry*. 2000; 47:296–304. [PubMed: 10686264]
4. Hollander E, Stein DJ, Decaria CM, et al. Disorders related to OCD--neurobiology. *Clin Neuropharmacol*. 1992; 15 (suppl):259A–260A.
5. Richter MA, Summerfeldt LJ, Antony MM, Swinson RP. Obsessive-compulsive spectrum conditions in obsessive-compulsive disorder and other anxiety disorders. *Depress Anxiety*. 2003; 18:118–127. [PubMed: 14625876]
6. Bienvenu OJ, Samuels JF, Riddle MA, et al. The relationship of obsessive-compulsive disorder to possible spectrum disorders: results from a family study. *Biol Psychiatry*. 2000; 48:287–293. [PubMed: 10960159]
7. Phillips KA. The obsessive compulsive spectrums. *Psychiatr Clin North Am*. 2002; 25:791–809. [PubMed: 12462861]
8. Denys D. Pharmacotherapy of obsessive-compulsive disorder and obsessive-compulsive spectrum disorders. *Psychiatr Clin North Am*. 2006; 29:553–584. [PubMed: 16650723]
9. Hollander E. Obsessive-compulsive spectrum disorders: an overview. *Psychiatr Ann*. 1994; 23:355–358.
10. Ravindran AV, da Silva TL, Ravindran LN, et al. Obsessive-compulsive spectrum disorders: a review of the evidence-based treatments. *Can J Psychiatry*. 2009; 54:331–343. [PubMed: 19497165]
11. Mataix-Cols D, Frost RO, Pertusa A, et al. Compulsive hoarding: a new disorder for DSM V? *Depress Anxiety*. in press.
12. Hollander, E.; Zohar, J.; Sirovatka, P.; Regier, D., editors. *Obsessive Compulsive Spectrum Disorders: Refining the Research Agenda for DSM-V*. Washington, DC: American Psychiatric Publishing; in press
13. Hollander E, Kim S, Zohar J. OCSDs in the forthcoming DSM-V. *CNS Spectr*. 2007; 12:320–323. [PubMed: 17585430]

14. Hollander E, Braun A, Simeon D. Should OCD leave the anxiety disorders in DSM-V? The case for obsessive compulsive-related disorders. *Depress Anxiety*. 2008; 25:317–3629. [PubMed: 18412058]
15. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 1. Washington, DC: American Psychiatric Association; 1952.
16. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 2. Washington, DC: American Psychiatric Association; 1968.
17. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3. Washington, DC: American Psychiatric Association; 1980.
18. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 3. Washington, DC: American Psychiatric Association; 1987. revised
19. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4. Washington, DC: American Psychiatric Association; 1994.
20. Kendler KS, Gardner CO, Prescott CA. Toward a comprehensive developmental model for major depression in women. *Am J Psychiatry*. 2002; 159:1133–1145. [PubMed: 12091191]
21. Robins E, Guze SB. Establishment of diagnostic validity in psychiatric illness: its application to schizophrenia. *Am J Psychiatry*. 1970; 126:983–987. [PubMed: 5409569]
22. First MB, Pincus HA, Levine JB, et al. Clinical utility as a criterion for revising psychiatric diagnoses. *Am J Psychiatry*. 2004; 161:946–954. [PubMed: 15169680]
23. Krueger RF. The structure of common mental disorders. *Arch Gen Psychiatry*. 1999; 56:921–926. [PubMed: 10530634]
24. Wittchen H-U, Beesdo K, Gloster AT, et al. The structure of mental disorders reexamined: is it developmentally stable and robust against additions? *Int J Methods Psychiatr Res*. 2009; 18:189–203. [PubMed: 20033884]
25. Wittchen HU, Beesdo K, Gloster AT. The position of anxiety disorders in structural models of mental disorders. *Psychiatr Clin North Am*. 2009; 32:465–481. [PubMed: 19716987]
26. <http://www.psych.org/MainMenu/Research/DSMIV/DSMV/DSMRevisionActivities/ConferenceSummaries/ObsessiveCompulsive.SpectrumDisordersConference.aspx>.
27. Leckman J, Denys D, Simpson H, et al. Obsessive-compulsive disorder: a review of the diagnostic criteria and possible subtypes and dimensional specifiers for DSM-V. *Depress Anxiety*. in press.
28. Mataix-Cols D, Pertusa A, Leckman JF. Issues for DSM-V: how should obsessive-compulsive and related disorders be classified? *Am J Psychiatry*. 2007; 164:1313–1314. [PubMed: 17728412]
29. Stein DJ, Grant JE, Franklin ME, et al. Trichotillomania (hair pulling disorder), skin picking disorder, and stereotypic movement disorder: towards DSM-V. *Depress Anxiety*. in press.
30. Storch EA, Abramowitz J, Goodman WK. Where does obsessive-compulsive disorder belong in DSM-V? *Depress Anxiety*. 2008; 25:336–347. [PubMed: 18412060]
31. Hollander E, Zohar J. Beyond refractory obsessions and anxiety states: toward remission. *J Clin Psychiatry*. 2004; 65 (Suppl 14):3–5. [PubMed: 15554781]
32. Stein DJ. Is disorder X in category or spectrum Y? General considerations and application to the relationship between obsessive-compulsive disorder and anxiety disorders. *Depress Anxiety*. 2008; 25:330–335. [PubMed: 18412059]
33. Koran LM, Hanna GL, Hollander E, et al. Practice guideline for the treatment of patients with obsessive-compulsive disorder. *Am J Psychiatry*. 2007; 164:5–53. [PubMed: 17849776]
34. Hollander E, Allen A, Kwon J, et al. Clomipramine vs desipramine crossover trial in body dysmorphic disorder: selective efficacy of a serotonin reuptake inhibitor in imagined ugliness. *Arch Gen Psychiatry*. 1999; 56:1033–1039. [PubMed: 10565503]
35. Phillips KA, Hollander E. Treating body dysmorphic disorder with medication: evidence, misconceptions, and a suggested approach. *Body Image: Int J Res*. 2008; 5:13–27.
36. Ipser JC, Sander C, Stein DJ. Pharmacotherapy and psychotherapy for body dysmorphic disorder. *Cochrane Database Syst Rev*. 2009:CD005332. [PubMed: 19160252]
37. National Collaborating Centre for Mental Health. Core interventions in the treatment of obsessive compulsive disorder and body dysmorphic disorder (a guideline from the National Institute for



Health and Clinical Excellence, National Health Service). <http://www.nice.org.uk/page.aspx?o=289817>

38. Stein DJ, Lochner C. Obsessive-compulsive spectrum disorders: a multidimensional approach. *Psychiatr Clin North Am.* 2006; 29:343–351. [PubMed: 16650712]
39. Joel, D.; Stein, DJ.; Schreiber, R. Animal models of obsessive-compulsive disorder: from bench to bedside via endophenotypes and biomarkers. In: McArthur, RA.; Borsini, F., editors. *Animal and translational models for CNS drug discovery, volume 1: psychiatric disorders.* Amsterdam: Elsevier; 2008.
40. Feusner J, Hembacher E, Phillips KA. The mouse who couldn't stop washing: pathologic grooming in animals and humans. *CNS Spectr.* 2009; 14:503–513. [PubMed: 19890232]
41. Stein DJ, Fineberg NA, Bienvenu OJ, et al. Should OCD be classified as an anxiety disorder in DSM-V? *Depress Anxiety.* in press.
42. Potenza MN, Koran LM, Pallanti S. The relationship between impulse-control disorders and obsessive-compulsive disorder: a current understanding and future research directions. *Psychiatry Res.* 2009; 170:22–31. [PubMed: 19811840]
43. Grant JE, Brewer JA, Potenza MN. The neurobiology of substance and behavioral addictions. *CNS Spectr.* 2006; 11:924–930. [PubMed: 17146406]
44. Chamberlain SR, Blackwell AD, Fineberg NA, et al. The neuropsychology of obsessive compulsive disorder: the importance of failures in cognitive and behavioural inhibition as candidate endophenotypic markers. *Neurosci Biobehav Rev.* 2005; 29:399–419. [PubMed: 15820546]
45. Abramowitz, J.; Deacon, BJ. Obsessive-compulsive disorders: essential phenomenology and overlap with other anxiety disorders. In: Abramowitz, J.; Houts, AC., editors. *Concepts and controversies in obsessive-compulsive disorder.* New York, N.Y: Springer; 2005.
46. Brady KT, Austin L, Lydiard RB. Body dysmorphic disorder: the relationship to obsessive-compulsive disorder. *J Nerv Ment Dis.* 1990; 178:538–540. [PubMed: 2380703]
47. Cohen, L.; Hollander, E. Obsessive-compulsive spectrum disorders. In: Stein, DJ., editor. *Obsessive-compulsive disorders.* New York: Marcel Dekker; 1997.
48. Goldsmith, T.; Shapira, NA.; Phillips, KA.; McElroy, SL. Conceptual foundations of obsessive-compulsive spectrum disorders. In: Antony, MM.; Rachman, S.; Richer, MA.; Swinson, RP., editors. *Obsessive-compulsive disorder: theory, research and treatment.* New York: Guilford Press; 1998.
49. Jaisoorya TS, Janardhan Reddy YC, Srinath S. The relationship of obsessive-compulsive disorder to putative spectrum disorders: results from an Indian study. *Compr Psychiatry.* 2003; 44:317–323. [PubMed: 12923710]
50. Phillips, KA. *Understanding body dysmorphic disorder: an essential guide.* New York: Oxford University Press; 2009.
51. Coles ME, Phillips KA, Menard W, et al. Body dysmorphic disorder and social phobia: cross-sectional and prospective data. *Depress Anxiety.* 2006; 23:26–33. [PubMed: 16278832]
52. Hrabosky JI, Cash TF, Veale D, et al. Multidimensional body image comparisons among patients with eating disorders, body dysmorphic disorder, and clinical controls: a multisite study. *Body Image: Int J Res.* 2009; 6:155–163.
53. Phillips KA. Body dysmorphic disorder and depression: theoretical considerations and treatment strategies. *Psychiatr Q.* 1999; 70:313–331. [PubMed: 10587987]
54. Phillips KA, Gunderson CG, Mallya G, et al. A comparison study of body dysmorphic disorder and obsessive-compulsive disorder. *J Clin Psychiatry.* 1998; 59:568–575. [PubMed: 9862601]
55. Phillips KA, Pinto A, Menard W, et al. Obsessive-compulsive disorder versus body dysmorphic disorder: a comparison study of two possibly related disorders. *Depress Anxiety.* 2007; 24:399–409. [PubMed: 17041935]
56. Frare F, Perugi G, Ruffolo G, Toni C. Obsessive-compulsive disorder and body dysmorphic disorder: a comparison of clinical features. *Eur Psychiatry.* 2004; 19:292–298. [PubMed: 15276662]

57. McKay D, Neziroglu F, Yaryura-Tobias JA. Comparison of clinical characteristics in obsessive-compulsive disorder and body dysmorphic disorder. *J Anxiety Disord.* 1997; 11:447–454. [PubMed: 9276787]
58. Buhlmann U, Wilhelm S, McNally RJ, et al. Interpretive biases for ambiguous information in body dysmorphic disorder. *CNS Spectr.* 2002; 7:441–443.
59. Saxena S, Winograd A, Dunkin JJ, et al. A retrospective review of clinical characteristics and treatment response in body dysmorphic disorder versus obsessive-compulsive disorder. *J Clin Psychiatry.* 2001; 62:67–72. [PubMed: 11235937]
60. Didie ER, Walters MM, Pinto A, et al. A comparison of quality of life and psychosocial functioning in obsessive-compulsive disorder and body dysmorphic disorder. *Ann Clin Psychiatry.* 2007; 19:181–186. [PubMed: 17729020]
61. Marazziti D, Dell'Osso L, Presta S, et al. Platelet [3H]paroxetine binding in patients with OCD-related disorders. *Psychiatry Res.* 1999; 89:223–228. [PubMed: 10708268]
62. Buhlmann U, Etcoff NL, Wilhelm S. Facial attractiveness ratings and perfectionism in body dysmorphic disorder and obsessive-compulsive disorder. *J Anxiety Disord.* 2008; 22:540–547. [PubMed: 17624717]
63. Buhlmann U, McNally RJ, Wilhelm S, Florin I. Selective processing of emotional information in body dysmorphic disorder. *J Anxiety Disord.* 2002; 16:289–298. [PubMed: 12214814]
64. Buhlmann U, Etcoff NL, Wilhelm S. Emotion recognition bias for contempt and anger in body dysmorphic disorder. *J Psychiatr Res.* 2006; 40:105–111. [PubMed: 15904932]
65. Rosen JC, Ramirez E. A comparison of eating disorders and body dysmorphic disorder on body image and psychological adjustment. *J Psychosom Res.* 1998; 44:441–449. [PubMed: 9587886]
66. Phillips KA, Kaye WH. The relationship of body dysmorphic disorder and eating disorders to obsessive-compulsive disorder. *CNS Spectr.* 2007; 12:347–358. [PubMed: 17514080]
67. Phillips, KA.; Wilhelm, S.; Koran, LM., et al. Body dysmorphic disorder: some key issues for DSM-V. in press
68. Phillips, KA. *The Broken Mirror: Understanding and Treating Body Dysmorphic Disorder.* New York: Oxford University Press; 2005. (revised and expanded ed.)
69. Phillips KA, McElroy SL, Keck PE Jr, et al. Body dysmorphic disorder: 30 cases of imagined ugliness. *Am J Psychiatry.* 1993; 150:302–308. [PubMed: 8422082]
70. Phillips KA, Diaz SF. Gender differences in body dysmorphic disorder. *J Nerv Ment Dis.* 1997; 185:570–577. [PubMed: 9307619]
71. Phillips KA, Menard W, Fay C, Weisberg R. Demographic characteristics, phenomenology, comorbidity, and family history in 200 individuals with body dysmorphic disorder. *Psychosomatics.* 2005; 46:317–325. [PubMed: 16000674]
72. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale II: validity. *Arch Gen Psychiatry.* 1989; 46:1012–1016. [PubMed: 2510699]
73. Phillips KA, Hollander E, Rasmussen SA, et al. A severity rating scale for body dysmorphic disorder: development, reliability, and validity of a modified version of the Yale-Brown Obsessive Compulsive Scale. *Psychopharmacol Bull.* 1997; 33:17–22. [PubMed: 9133747]
74. Eisen JL, Phillips KA, Coles ME, Rasmussen SA. Insight in obsessive compulsive disorder and body dysmorphic disorder. *Compr Psychiatry.* 2004; 45:10–15. [PubMed: 14671731]
75. Phillips KA, Menard W, Pagano ME, et al. Delusional versus nondelusional body dysmorphic disorder: clinical features and course of illness. *J Psychiatr Res.* 2006; 40:95–104. [PubMed: 16229856]
76. Mancuso S, Knoesen N, Castle DJ. Delusional vs nondelusional body dysmorphic disorder. *Compr Psychiatry.* 2010; 51:177–182. [PubMed: 20152299]
77. Veale D, Riley S. Mirror, mirror on the wall, who is the ugliest of them all? the psychopathology of mirror gazing in body dysmorphic disorder. *Behav Res Ther.* 2001; 39:1381–1393. [PubMed: 11758697]
78. Veale D, Boocock A, Gournay K, et al. Body dysmorphic disorder: a survey of fifty cases. *Br J Psychiatry.* 1996; 169:196–201. [PubMed: 8871796]

79. Hofmann, S.; Barlow, DH. Social phobia (social anxiety disorder). In: Barlow, DH., editor. *Anxiety and its disorders*. 2. New York: Guilford Press; 2002.
80. Beck, J., editor. *Cognitive therapy, basics and beyond*. New York: Guilford Press; 2005.
81. Pinto A, Phillips KA. Social anxiety in body dysmorphic disorder. *Body Image: Int J Res*. 2005; 2:401–405.
82. Kelly MM, Walters C, Phillips KA. Social anxiety and its relationship to functional impairment in body dysmorphic disorder. *Behav Ther*. in press.
83. Phillips KA, McElroy SL. Personality disorders and traits in patients with body dysmorphic disorder. *Compr Psychiatry*. 2000; 41:229–236. [PubMed: 10929788]
84. Phillips KA, Pinto A, Jain S. Self-esteem in body dysmorphic disorder. *Body Image: Int J Res*. 2004; 1:385–390.
85. Orth U, Robins RW, Trzesniewski KH, et al. Low self-esteem is a risk factor for depressive symptoms from young adulthood to old age. *J Abnorm Psychol*. 2009; 118:472–478. [PubMed: 19685945]
86. Posternak MA, Zimmerman M. Symptoms of atypical depression. *Psychiatry Res*. 2001; 104:175–181. [PubMed: 11711170]
87. Phillips KA, Nierenberg AA, Brendel G, Fava M. Prevalence and clinical features of body dysmorphic disorder in atypical major depression. *J Nerv Ment Dis*. 1996; 184:125–129. [PubMed: 8596110]
88. Phillips KA, Siniscalchi JM, McElroy SL. Depression, anxiety, anger, and somatic symptoms in patients with body dysmorphic disorder. *Psychiatr Q*. 2004; 75:309–320. [PubMed: 15563049]
89. Didie ER, Kuniega-Pietrzak T, Phillips KA. Body image in patients with body dysmorphic disorder: evaluations of and investment in appearance, health/illness, and fitness. *Body Image: Int J Res*. 2010; 7:66–69.
90. Gunstad J, Phillips KA. Axis I comorbidity in body dysmorphic disorder. *Compr Psychiatry*. 2003; 44:270–276. [PubMed: 12923704]
91. Demyttenaere K, Bruffaerts R, Posada-Villa J, et al. Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health Surveys. *JAMA*. 2004; 291:2581–2590. [PubMed: 15173149]
92. Kessler RC, Berglund P, Demler O, et al. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2005; 62:593–602. [PubMed: 15939837]
93. Simeon D, Hollander E, Stein DJ, et al. Body dysmorphic disorder in the DSM-IV field trial for obsessive-compulsive disorder. *Am J Psychiatry*. 1995; 152:1207–1209. [PubMed: 7625473]
94. Diniz JB, Rosario-Campos MC, Shavitt RG, et al. Impact of age at onset and duration of illness on the expression of comorbidities in obsessive-compulsive disorder. *J Clin Psychiatry*. 2004; 65:22–27. [PubMed: 14744164]
95. Brawman-Mintzer O, Lydiard RB, Phillips KA, et al. Body dysmorphic disorder in patients with anxiety disorders and major depression: a comorbidity study. *Am J Psychiatry*. 1995; 152:1665–1667. [PubMed: 7485632]
96. Hollander E, Cohen LJ, Simeon D. Body dysmorphic disorder. *Psychiatr Ann*. 1993; 23:359–364.
97. Wilhelm S, Otto MW, Zucker BG, Pollack MH. Prevalence of body dysmorphic disorder in patients with anxiety disorders. *J Anxiety Disord*. 1997; 11:499–502. [PubMed: 9407269]
98. Perugi G, Akiskal HS, Lattanzi L, et al. The high prevalence of “soft” bipolar (II) features in atypical depression. *Compr Psychiatry*. 1998; 39:63–71. [PubMed: 9515190]
99. Nierenberg AA, Phillips KA, Petersen TJ, et al. Body dysmorphic disorder in outpatients with major depression. *J Affect Disord*. 2002; 69:141–148. [PubMed: 12103460]
100. Grant JE, Kim SW, Eckert ED. Body dysmorphic disorder in patients with anorexia nervosa: prevalence, clinical features, and delusionalities of body image. *Int J Eat Disord*. 2002; 32:291–300. [PubMed: 12210643]
101. Steinhausen HC, Weber S. The outcome of bulimia nervosa: findings from one-quarter century of research. *Am J Psychiatry*. 2009; 166:1331–1341. [PubMed: 19884225]

102. Hudson JI, Hiripi E, Pope HG Jr, Kessler RC. The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biol Psychiatry*. 2007; 61:348–358. [PubMed: 16815322]
103. Phillips KA, Pagano ME, Menard W, Stout RL. A 12-month follow-up study of the course of body dysmorphic disorder. *Am J Psychiatry*. 2006; 163:907–912. [PubMed: 16648334]
104. Mataix-Cols D, Rauch SL, Baer L, 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. [PubMed: 11823269]
105. Keller MB. Social anxiety disorder clinical course and outcome: review of Harvard/Brown Anxiety Research Project (HARP) findings. *J Clin Psychiatry*. 2006; 67 (Suppl 12):14–19. [PubMed: 17092191]
106. Herzog DB, Dorer DJ, Keel PK, et al. Recovery and relapse in anorexia and bulimia nervosa: a 7.5-year follow-up study. *J Am Acad Child Adolesc Psychiatry*. 1999; 38:829–837. [PubMed: 10405500]
107. Keller MB, Lavori PW, Mueller TI, et al. Time to recovery, chronicity, and levels of psychopathology in major depression. A 5-year prospective follow-up of 431 subjects. *Arch Gen Psychiatry*. 1992; 49:809–816. [PubMed: 1417434]
108. Phillips KA, Stout RL. Associations in the longitudinal course of body dysmorphic disorder with major depression, obsessive compulsive disorder, and social phobia. *J Psychiatr Res*. 2006; 40:360–369. [PubMed: 16309706]
109. Zohar J, Kennedy JL, Hollander E, Koran LM. Serotonin-1D hypothesis of obsessive-compulsive disorder: an update. *J Clin Psychiatry*. 2004; 65 (Suppl 14):18–21. [PubMed: 15554784]
110. Didie ER, Tortolani CC, Pope CG, et al. Childhood abuse and neglect in body dysmorphic disorder. *Child Abuse Negl*. 2006; 30:1105–1015. [PubMed: 17005251]
111. Neziroglu F, Khemlani-Patel S, Yaryura-Tobias JA. Rates of abuse in body dysmorphic disorder and obsessive-compulsive disorder. *Body Image: Int J Res*. 2006; 3:189–193.
112. Jacobi C, Hayward C, de Zwaan M, et al. Coming to terms with risk factors for eating disorders: application of risk terminology and suggestions for a general taxonomy. *Psychol Bull*. 2004; 130:19–65. [PubMed: 14717649]
113. Gibb BE, Chelminski I, Zimmerman M. Childhood emotional, physical, and sexual abuse, and diagnoses of depressive and anxiety disorders in adult psychiatric outpatients. *Depress Anxiety*. 2007; 24:256–263. [PubMed: 17041933]
114. Rauch SL, Phillips KA, Segal E, et al. A preliminary morphometric magnetic resonance imaging study of regional brain volumes in body dysmorphic disorder. *Psychiatry Res*. 2003; 122:13–19. [PubMed: 12589879]
115. Atmaca M, Bingol I, Aydin A, et al. Brain morphology of patients with body dysmorphic disorder. *J Affect Disord*. 2009 Epub ahead of print.
116. Feusner JD, Townsend J, Bystritsky A, et al. Regional brain volumes and symptom severity in body dysmorphic disorder. *Psychiatry Res*. 2009; 172:161–167. [PubMed: 19328661]
117. 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. [PubMed: 18952675]
118. Carey P, Seedat S, Warwick J, et al. SPECT imaging of body dysmorphic disorder. *J Neuropsychiatry Clin Neurosci*. 2004; 16:357–359. [PubMed: 15377744]
119. Feusner JD, Townsend J, Bystritsky A, Bookheimer S. Visual information processing of faces in body dysmorphic disorder. *Arch Gen Psychiatry*. 2007; 64:1417–1425. [PubMed: 18056550]
120. Feusner JD, Moody T, Hembacher E. Abnormalities of visual processing and fronto-striatal systems in body dysmorphic disorder. *Arch Gen Psychiatry*. 2010; 67:197–205. [PubMed: 20124119]
121. Rotge JY, Guehl D, Dilharreguy B, et al. Provocation of obsessive-compulsive symptoms: a quantitative voxel-based meta-analysis of functional neuroimaging studies. *J Psychiatry Neurosci*. 2008; 33:405–412. [PubMed: 18787662]
122. Saxena S, Feusner JD. Toward a neurobiology of body dysmorphic disorder. *Prim Psychiatry*. 2006; 13:41–48.

123. Stein MB, Goldin PR, Sareen J, et al. Increased amygdala activation to angry and contemptuous faces in generalized social phobia. *Arch Gen Psychiatry*. 2002; 59:1027–1034. [PubMed: 12418936]
124. Ashby JS, Kottman T, Schoen E. Perfectionism and eating disorders reconsidered. *J Ment Health Couns*. 1998; 20:261–271.
125. Kimbrel NA. A model of the development and maintenance of generalized social phobia. *Clin Psychol Rev*. 2008; 28:592–612. [PubMed: 17959288]
126. Bienvenu OJ, Samuels JF, Costa PT, et al. Anxiety and depressive disorders and the five-factor model of personality: a higher- and lower-order personality trait investigation in a community sample. *Depress Anxiety*. 2004; 20:92–97. [PubMed: 15390211]
127. Cuijpers P, van Straten A, Donker M. Personality traits of patients with mood and anxiety disorders. *Psychiatry Res*. 2005; 133:229–237. [PubMed: 15740998]
128. Petersen T, Bottonari K, Alpert JE, et al. Use of the five-factor inventory in characterizing patients with major depressive disorder. *Compr Psychiatry*. 2001; 42:488–493. [PubMed: 11704941]
129. Bienvenu OJ, Nestadt G, Samuels JF, et al. Phobic, panic, and major depressive disorders and the five-factor model of personality. *J Nerv Ment Dis*. 2001; 189:154–161. [PubMed: 11277351]
130. Pavan C, Vindigni V, Semenzin M, et al. Personality, temperament and clinical scales in an Italian plastic surgery setting: what about body dysmorphic disorder? *Int J Psychiatry Clin Pract*. 2006; 10:91–96.
131. Pfohl B, Black D, Noyes R Jr, et al. A test of the tridimensional personality theory: association with diagnosis and platelet imipramine binding in obsessive-compulsive disorder. *Biol Psychiatry*. 1990; 28:41–46. [PubMed: 2165423]
132. Lyoo IK, Lee DW, Kim YS, et al. Patterns of temperament and character in subjects with obsessive-compulsive disorder. *J Clin Psychiatry*. 2001; 62:637–641. [PubMed: 11561937]
133. Faytout M, Tignol J, Swendsen J, et al. Social phobia, fear of negative evaluation and harm avoidance. *Eur Psychiatry*. 2007; 22:75–79. [PubMed: 17101266]
134. Abrams KY, Yune SK, Kim SJ, et al. Trait and state aspects of harm avoidance and its implication for treatment in major depressive disorder, dysthymic disorder, and depressive personality disorder. *Psychiatry Clin Neurosci*. 2004; 58:240–248. [PubMed: 15149288]
135. Cassin SE, von Ranson KM. Personality and eating disorders: a decade in review. *Clin Psychol Rev*. 2005; 25:895–916. [PubMed: 16099563]
136. Deckersbach T, Savage CR, Phillips KA, et al. Characteristics of memory dysfunction in body dysmorphic disorder. *J Int Neuropsychol Soc*. 1999; 6:673–681. [PubMed: 11011514]
137. Deckersbach T, Otto MW, Savage CR, et al. The relationship between semantic organization and memory in obsessive-compulsive disorder. *Psychother Psychosom*. 2000; 69:101–107. [PubMed: 10671831]
138. Savage CR, Deckersbach T, Wilhelm S, et al. Strategic processing and episodic memory impairment in obsessive compulsive disorder. *Neuropsychology*. 2000; 14:141–151. [PubMed: 10674806]
139. Sherman BJ, Savage CR, Eddy KT, et al. Strategic memory in adults with anorexia nervosa: are there similarities to obsessive compulsive spectrum disorders? *Int J Eat Disord*. 2006; 39:468–476. [PubMed: 16715489]
140. Amir N, Foa EB, Coles ME. Automatic activation and strategic avoidance of threat-relevant information in social phobia. *J Abnorm Psychol*. 1998; 107:285–290. [PubMed: 9604557]
141. Buhlmann U, McNally RJ, Etkoff NL, et al. Emotion recognition deficits in body dysmorphic disorder. *J Psychiatr Res*. 2004; 38:201–206. [PubMed: 14757335]
142. Phillips KA, Menard W. A prospective pilot study of levetiracetam for body dysmorphic disorder. *CNS Spectr*. 2009; 14:252–260. [PubMed: 19407724]
143. Allen A, Hadley SJ, Kaplan A, et al. An open-label trial of venlafaxine in body dysmorphic disorder. *CNS Spectr*. 2008; 13:138–144. [PubMed: 18227745]
144. Phillips KA. Placebo-controlled study of pimozide augmentation of fluoxetine in body dysmorphic disorder. *Am J Psychiatry*. 2005; 162:377–379. [PubMed: 15677604]

145. Phillips KA. Olanzapine augmentation of fluoxetine in body dysmorphic disorder (letter). *Am J Psychiatry*. 2005; 162:1022–1023. [PubMed: 15863817]
146. Westenberg HG. Recent advances in understanding and treating social anxiety disorder. *CNS Spectr*. 2009; 14:24–33. [PubMed: 19238127]
147. Yager, J.; Devlin, M.; Halmi, KA., et al. Practice guideline for the treatment of patients with eating disorders. 3. Washington, DC: American Psychiatric Association; 2006.
148. Karasu, T.; Gelenberg, A.; Merriam, A.; Wang, P. Practice guideline for the treatment of patients with major depressive disorder. 2. Washington, DC: American Psychiatric Association; 2000.
149. Claudino AM, Hay P, Lima MS, et al. Antidepressants for anorexia nervosa. *Cochrane Database Syst Rev*. 2006:CD004365. [PubMed: 16437485]
150. Neziroglu F, Khemlani-Patel S. A review of cognitive and behavioral treatment for body dysmorphic disorder. *CNS Spectr*. 2002; 7:464–471. [PubMed: 15107768]
151. Abramowitz JS. Effectiveness of psychological and pharmacological treatments for obsessive-compulsive disorder: a quantitative review. *J Consult Clin Psychol*. 1997; 65:44–52. [PubMed: 9103733]
152. Gould RA, Buckminster S, Pollack MH, et al. Cognitive-behavioral and pharmacological treatment for social phobia: a meta-analysis. *J Clin Psychol*. 1997; 4:291–306.
153. Feske U, Chambless DL. Cognitive behavioral versus exposure only treatment for social phobia: a meta-analysis. *Behav Ther*. 1995; 26:695–720.
154. van Balkom AJLM, van Oppen P, Vermeulen AWA, et al. A meta-analysis on the treatment of obsessive compulsive disorder: a comparison of antidepressants, behavior, and cognitive therapy. *Clin Psychol Rev*. 1994; 14:359–381.
155. Fedoroff IC, Taylor S. Psychological and pharmacological treatments of social phobia: a meta-analysis. *J Clin Psychopharmacol*. 2001; 21:311–324. [PubMed: 11386495]
156. Rosa-Alcazar AI, Sanchez-Meca J, Gomez-Conesa A, Marin-Martinez F. Psychological treatment of obsessive-compulsive disorder: a meta-analysis. *Clin Psychol Rev*. 2008; 28:1310–1325. [PubMed: 18701199]
157. Singh JS, Hope DA. Cognitive-behavioral approaches to the treatment of social anxiety disorder. *Isr J Psychiatry Relat Sci*. 2009; 46:62–69. [PubMed: 19728574]
158. Wilhelm, S.; Phillips, KA.; Steketee, G. A cognitive-behavioral treatment manual for body dysmorphic disorder: Guilford Publications. in press
159. Janet, P. *Les Obsessions et la Psychasthenie*. Paris: Felix Alcan; 1903.
160. Morselli E. Sulla dismorfofobia e sulla tafefobia. *Bollettino della R Accademia di Genova*. 1891; 6:110–119.
161. Phillips KA. Body dysmorphic disorder: the distress of imagined ugliness. *Am J Psychiatry*. 1991; 148:1138–1149. [PubMed: 1882990]
162. Stekel, W. *Compulsion and doubt*. Gutheil, EA., translator. New York: Liveright; 1949.
163. Suzuki K, Takei N, Kawai M, et al. Is taijin kyofusho a culture-bound syndrome? *Am J Psychiatry*. 2003; 160:1358. [PubMed: 12832264]
164. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4. Washington, DC: American Psychiatric Association; 2000. text revision
165. Leckman JF, Peterson BS, Pauls DL, Cohen DJ. Tic disorders. *Psychiatr Clin North Am*. 1997; 20:839–861. [PubMed: 9443353]
166. Ferrao YA, Miguel EC, Stein DJ. Tourette’s syndrome, trichotillomania, and obsessive-compulsive disorder: how closely are they related? *Psychiatry Res*. 2009; 170:32–42. [PubMed: 19801170]
167. Leckman JF, Walker DE, Goodman WK, et al. “Just right” perceptions associated with compulsive behavior in Tourette’s syndrome. *Am J Psychiatry*. 1994; 151:675–680. [PubMed: 8166308]
168. Rosario MC, Prado HS, Borcato S, et al. Validation of the University of Sao Paulo Sensory Phenomena Scale: initial psychometric properties. *CNS Spectr*. 2009; 14:315–323. [PubMed: 19668122]

169. The Tourette Syndrome Classification Study Group. Definitions and classification of tic disorders. *Arch Neurol*. 1993; 50:1013–1016. [PubMed: 8215958]
170. Leckman JF, Walker DE, Cohen DJ. Premonitory urges in Tourette's syndrome. *Am J Psychiatry*. 1993; 150:98–102. [PubMed: 8417589]
171. Kurlan R, Lichter D, Hewitt D. Sensory tics in Tourette's syndrome. *Neurology*. 1989; 39:731–734. [PubMed: 2710364]
172. Cohen AJ, Leckman JF. Sensory phenomena associated with Gilles de la Tourette's syndrome. *J Clin Psychiatry*. 1992; 53:319–323. [PubMed: 1517194]
173. Foa EB, Kozak MJ, Goodman WK, et al. DSM-IV field trial: obsessive-compulsive disorder. *Am J Psychiatry*. 1995; 152:90–96. [PubMed: 7802127]
174. Miguel EC, Ferrao YA, Rosario MC, et al. The Brazilian Research Consortium on Obsessive-Compulsive Spectrum Disorders: recruitment, assessment instruments, methods for the development of multicenter collaborative studies and preliminary results. *Revista Brasileira de Psiquiatria Brazilian Journal of Psychiatry*. 2008; 30:185–196.
175. de Mathis MA, do Rosario MC, Diniz JB, et al. Obsessive-compulsive disorder: influence of age at onset on comorbidity patterns. *Eur Psychiatry*. 2008; 23:187–194. [PubMed: 18329252]
176. Pauls DL, Raymond CL, Stevenson JM, Leckman JF. A family study of Gilles de la Tourette syndrome. *Am J Hum Gene*. 1991; 48:154–163.
177. van de Wetering BJ, Heutink P. The genetics of the Gilles de la Tourette syndrome: a review. *J Lab Clin Med*. 1993; 121:638–645. [PubMed: 8478592]
178. 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. [PubMed: 11504325]
179. 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. [PubMed: 15180774]
180. Bloch MH, Craiglow BG, Landeros-Weisenberger A, et al. Predictors of early adult outcome in pediatric-onset obsessive-compulsive disorder. *Pediatrics*. 2009; 124:1085–1093. [PubMed: 19786445]
181. Stewart SE, Illmann C, Geller DA, et al. A controlled family study of attention-deficit/hyperactivity disorder and Tourette's disorder. *J Am Acad Child Adolesc Psychiatry*. 2006; 45:1354–1362. [PubMed: 17075358]
182. Pauls DL, Alsobrook JP 2nd, Goodman W, et al. A family study of obsessive-compulsive disorder. *Am J Psychiatry*. 1995; 152:76–84. [PubMed: 7802125]
183. 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. [PubMed: 11690590]
184. 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. [PubMed: 15892140]
185. Reddy PS, Reddy YC, Srinath S, et al. A family study of juvenile obsessive-compulsive disorder. *Can Journal Psychiatry*. 2001; 46:346–351.
186. Fyer AJ, Lipsitz JD, Mannuzza S, et al. A direct interview family study of obsessive-compulsive disorder: I. *Psychol Med*. 2005; 35:1611–1621. [PubMed: 16219119]
187. Olson S. Neurobiology: making sense of Tourette's. *Science*. 2004; 305:1390–1392. [PubMed: 15353772]
188. Lin H, Katsovich L, Ghebremichael M, et al. Psychosocial stress predicts future symptom severities in children and adolescents with Tourette syndrome and/or obsessive-compulsive disorder. *J Child Psychol Psychiatry*. 2007; 48:157–166. [PubMed: 17300554]
189. Verkerk AJ, Cath DC, van der Linde HC, et al. Genetic and clinical analysis of a large Dutch Gilles de la Tourette family. *Mol Psychiatry*. 2006; 11:954–964. [PubMed: 16894393]
190. Price RA, Kidd KK, Cohen DJ, et al. A twin study of Tourette syndrome. *Arch Gen Psychiatry*. 1985; 42:815–820. [PubMed: 3860194]

191. Bloch MH, Leckman J, Peterson BS. Basal ganglia volumes and future tic severity in children with Tourette syndrome. *Neurology*. 2005; 65:1253–1258. [PubMed: 16247053]
192. Peterson BS, Thomas P, Kane MJ, et al. Basal ganglia volumes in patients with Gilles de la Tourette syndrome. *Arch Gen Psychiatry*. 2003; 60:415–424. [PubMed: 12695320]
193. Eidelberg D, Moeller JR, Antonini A, et al. The metabolic anatomy of Tourette's syndrome. *Neurology*. 1997; 48:927–934. [PubMed: 9109879]
194. Stern E, Silbersweig DA, Chee KY, et al. A functional neuroanatomy of tics in Tourette syndrome. *Arch Gen Psychiatry*. 2000; 57:741–748. [PubMed: 10920461]
195. Swedo SE, Schapiro MB, Grady CL, et al. Cerebral glucose metabolism in childhood-onset obsessive-compulsive disorder. *Arch Gen Psychiatry*. 1989; 46:518–523. [PubMed: 2786402]
196. Saxena S, Brody AL, Schwartz JM, Baxter LR. Neuroimaging and frontal-subcortical circuitry in obsessive-compulsive disorder. *Br J Psychiatry*. 1998; (Suppl):26–37.
197. Baxter LR Jr. Positron emission tomography studies of cerebral glucose metabolism in obsessive compulsive disorder. *J Clin Psychiatry*. 1994; 55 (Suppl):54–59. [PubMed: 7961533]
198. 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. [PubMed: 11288777]
199. Horesh N, Zimmerman S, Steinberg T, et al. Is onset of Tourette syndrome influenced by life events? *J Neural Transm*. 2008; 115:787–793. [PubMed: 18217190]
200. Lochner C, Hemmings SM, Kinnear CJ, et al. Cluster analysis of obsessive-compulsive spectrum disorders in patients with obsessive-compulsive disorder: clinical and genetic correlates. *Compr Psychiatry*. 2005; 46:14–19. [PubMed: 15714189]
201. Simpson H, Rosen W, Huppert J, et al. Are there reliable neuropsychological deficits in obsessive-compulsive disorder. *J Psychiatr Res*. 2006; 40:247–257. [PubMed: 15950242]
202. 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. [PubMed: 18061263]
203. Chamberlain S, Menzies L, Hampshire A, et al. Orbitofrontal dysfunction in patients with obsessive-compulsive disorder and their unaffected relatives. *Science*. 2008; 321:421–422. [PubMed: 18635808]
204. 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. [PubMed: 16846259]
205. 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. [PubMed: 17988651]
206. 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. [PubMed: 19221910]
207. Chang SW, McCracken JT, Piacentini JC. Neurocognitive correlates of child obsessive compulsive disorder and Tourette syndrome. *J Clin Exp Neuropsychol*. 2007; 29:724–733. [PubMed: 17896198]
208. Savage CR, Baer L, Keuthen NJ, et al. Organizational strategies mediate nonverbal memory impairment in obsessive-compulsive disorder. *Biol Psychiatry*. 1999; 45:905–916. [PubMed: 10202579]
209. Watkins LH, Sahakian BJ, Robertson MM, et al. Executive function in Tourette's syndrome and obsessive-compulsive disorder. *Psychol Med*. 2005; 35:571–582. [PubMed: 15856727]
210. Penades R, Catalan R, Rubia K, et al. Impaired response inhibition in obsessive compulsive disorder. *Eur Psychiatry*. 2007; 22:404–410. [PubMed: 17127038]
211. Deckersbach T, Savage CR, Curran T, et al. A study of parallel implicit and explicit information processing in patients with obsessive-compulsive disorder. *Am J Psychiatry*. 2002; 159:1780–1782. [PubMed: 12359688]



212. Marsh R, Alexander GM, Packard MG, et al. Habit learning in Tourette syndrome: a translational neuroscience approach to a developmental psychopathology. *Arch Gen Psychiatry*. 2004; 61:1259–1268. [PubMed: 15583117]
213. Bloch MH. Emerging treatments for Tourette's disorder. *Curr Psychiatry Rep*. 2008; 10:323–330. [PubMed: 18627671]
214. Muller-Vahl KR, Meyer GJ, Knapp WH, et al. Serotonin transporter binding in Tourette syndrome. *Neurosci Lett*. 2005; 385:120–125. [PubMed: 15936877]
215. Heinz A, Knable MB, Wolf SS, et al. Tourette's syndrome: [I-123]beta-CIT SPECT correlates of vocal tic severity. *Neurology*. 1998; 51:1069–1074. [PubMed: 9781531]
216. Shavitt RG, Belotto C, Curi M, et al. Clinical features associated with treatment response in obsessive-compulsive disorder. *Compr Psychiatry*. 2006; 47:276–281. [PubMed: 16769302]
217. McDougle CJ, Goodman WK, Price LH, et al. Neuroleptic addition in fluvoxamine-refractory obsessive-compulsive disorder. *Am J Psychiatry*. 1990; 147:652–654. [PubMed: 1970224]
218. Rasmussen SA, Eisen JL. Treatment strategies for chronic and refractory obsessive-compulsive disorder. *J Clin Psychiatry*. 1997; 58 (Suppl 13):9–13. [PubMed: 9402914]
219. Ferrao YA, Diniz JB, Lopes AC, et al. Resistance and refractoriness in obsessive-compulsive disorder. *Revista Brasileira Psiquiatria Brazilian J Psychiatry*. 2007; 29 (Suppl 2):S66–S76.
220. Bloch MH, Landeros-Weisenberger A, Kelmendi B, et al. A systematic review: antipsychotic augmentation with treatment refractory obsessive-compulsive disorder. *Mol Psychiatry*. 2006; 11:622–632. [PubMed: 16585942]
221. Denys D, van der Wee N, Janssen J, et al. Low level of dopaminergic D2 receptor binding in obsessive-compulsive disorder. *Biol Psychiatry*. 2004; 55:1041–1045. [PubMed: 15121489]
222. van der Wee NJ, Stevens H, Hardeman JA, et al. Enhanced dopamine transporter density in psychotropic-naïve patients with obsessive-compulsive disorder shown by [123I]{beta}-CIT SPECT. *Am J Psychiatry*. 2004; 161:2201–2206. [PubMed: 15569890]
223. Volpato Cordioli A, Heldt E, Braga Bochi D, et al. Cognitive-behavioral group therapy in obsessive-compulsive disorder: a randomized clinical trial. *Psychother Psychosom*. 2003; 72:211–216. [PubMed: 12792126]
224. Anderson RA, Rees CS. Group versus individual cognitive-behavioural treatment for obsessive-compulsive disorder: a controlled trial. *Behav Res Ther*. 2007; 45:123–137. [PubMed: 16540080]
225. Verdellen CW, Hoogduin CA, Kato BS, et al. Habituation of premonitory sensations during exposure and response prevention treatment in Tourette's syndrome. *Behav Modif*. 2008; 32:215–227. [PubMed: 18285507]
226. Himle MB, Woods DW, Piacentini JC, Walkup JT. Brief review of habit reversal training for Tourette syndrome. *J Child Neurol*. 2006; 21:719–725. [PubMed: 16970874]
227. Azrin NH, Nunn RG. Habit-reversal: a method of eliminating nervous habits and tics. *Behav Res Ther*. 1973; 11:619–628. [PubMed: 4777653]
228. Tükel R, Keser V, Karali NT, et al. Comparison of clinical characteristics in trichotillomania and obsessive-compulsive disorder. *J Anxiety Disord*. 2001; 15:433–441. [PubMed: 11583075]
229. Lochner C, Seedat S, du Toit PL, et al. Obsessive-compulsive disorder and trichotillomania: a phenomenological comparison. *BMC Psychiatry*. 2005; 5:2. [PubMed: 15649315]
230. Lochner C, Simeon D, Niehaus DJ, Stein DJ. Trichotillomania and skin-picking: a phenomenological comparison. *Depress Anxiety*. 2002; 15:83–86. [PubMed: 11891999]
231. Odlaug BL, Grant JE. Trichotillomania and pathologic skin picking: clinical comparison with an examination of comorbidity. *Ann Clin Psychiatry*. 2008; 20:57–63. [PubMed: 18568576]
232. Christenson, GA.; Mansueto, CS. Trichotillomania: descriptive characteristics and phenomenology. In: Stein, DJ.; Christenson, GA.; Hollander, E., editors. *Trichotillomania*. Washington, D.C: American Psychiatric Press, Inc; 1999.
233. Black DW, Kehrberg LL, Flumerfelt DL, Schlosser SS. Characteristics of 36 subjects reporting compulsive sexual behavior. *Am J Psychiatry*. 1997; 154:243–249. [PubMed: 9016275]
234. McElroy SL, Pope HG Jr, Hudson JI, et al. Kleptomania: a report of 20 cases. *Am J Psychiatry*. 1991; 148:652–657. [PubMed: 2018170]

235. McElroy SL, Soutullo CA, Beckman DA, et al. DSM-IV intermittent explosive disorder: a report of 27 cases. *J Clin Psychiatry*. 1998; 59:203–210. [PubMed: 9590677]
236. Grant JE, Kim SW. Comorbidity of impulse control disorders in pathological gamblers. *Acta Psychiatr Scand*. 2003; 108:203–207. [PubMed: 12890275]
237. Stein DJ, Flessner CA, Franklin M, et al. Is trichotillomania a stereotypic movement disorder? An analysis of body-focused repetitive behaviors in people with hair-pulling. *Ann Clin Psychiatry*. 2008; 20:194–198. [PubMed: 19034750]
238. Keuthen NJ, Fraim C, Deckersbach T, et al. Longitudinal follow-up of naturalistic treatment outcome in patients with trichotillomania. *J Clin Psychiatry*. 2001; 62:101–107. [PubMed: 11247093]
239. Christenson GA, Mackenzie TB, Mitchell JE. Characteristics of 60 adult chronic hair pullers. *Am J Psychiatry*. 1991; 148:365–370. [PubMed: 1992841]
240. Williams KE, Koran LM. Obsessive-compulsive disorder in pregnancy, the puerperium, and the premenstruum. *J Clin Psychiatry*. 1997; 58:330–334. [PubMed: 9269260]
241. McElroy SL, Hudson JI, Pope H Jr, et al. The DSM-III-R impulse control disorders not elsewhere classified: clinical characteristics and relationship to other psychiatric disorders. *Am J Psychiatry*. 1992; 149:318–327. [PubMed: 1536268]
242. Lenane MC, Swedo SE, Rapoport JL, et al. Rates of obsessive-compulsive disorder in first degree relatives of patients with trichotillomania: a research note. *J Child Psychol Psychiatry*. 1992; 33:925–933. [PubMed: 1634595]
243. Schlosser S, Black DW, Blum N, Goldstein RB. The demography, phenomenology, and family history of 22 persons with compulsive hair pulling. *Ann Clin Psychiatry*. 1994; 6:147–152. [PubMed: 7881494]
244. Abelson JF, Kwan KY, O’Roak BJ, et al. Sequence variants in *SLITRK1* are associated with Tourette’s syndrome. *Science*. 2005; 310:317–320. [PubMed: 16224024]
245. Zuchner S, Cuccaro ML, Tran-Viet KN, et al. *SLITRK1* mutations in trichotillomania. *Mol Psychiatry*. 2006; 11:887–889. [PubMed: 17003809]
246. Zuchner S, Wendland JR, Ashley-Koch AE, et al. Multiple rare *SAPAP3* missense variants in trichotillomania and OCD. *Mol Psychiatry*. 2009; 14:6–9. [PubMed: 19096451]
247. Hemmings SM, Kinnear CJ, Lochner C, et al. Genetic correlates in trichotillomania: a case-control association study in the South African Caucasian population. *Isr J Psychiatry Relat Sci*. 2006; 43:93–101. [PubMed: 16910371]
248. Saxena S, Rauch SL. Functional neuroimaging and the neuroanatomy of obsessive-compulsive disorder. *Psychiatr Clin North Am*. 2000; 23:563–586. [PubMed: 10986728]
249. O’Sullivan RL, Rauch SL, Breiter HC, et al. Reduced basal ganglia volumes in trichotillomania measured via morphometric magnetic resonance imaging. *Biol Psychiatry*. 1997; 42:39–45. [PubMed: 9193740]
250. Stein DJ, Coetzer R, Lee M, et al. Magnetic resonance brain imaging in women with obsessive-compulsive disorder and trichotillomania. *Psychiatry Res*. 1997; 74:177–182. [PubMed: 9255863]
251. Keuthen NJ, Makris N, Schlerf JE, et al. Evidence for reduced cerebellar volumes in trichotillomania. *Biol Psychiatry*. 2007; 61:374–381. [PubMed: 16945351]
252. Chamberlain SR, Menzies LA, Fineberg NA, et al. Grey matter abnormalities in trichotillomania: morphometric magnetic resonance imaging study. *Br J Psychiatry*. 2008; 193:216–221. [PubMed: 18757980]
253. Ninan PT, Rothbaum BO, Stipetic M, et al. CSF 5-HIAA as a predictor of treatment response in trichotillomania. *Psychopharmacol Bull*. 1992; 28:451–455. [PubMed: 1284254]
254. Micallef J, Blin O. Neurobiology and clinical pharmacology of obsessive-compulsive disorder. *Clin Neuropharmacol*. 2001; 24:191–207. [PubMed: 11479390]
255. Chamberlain SR, Fineberg NA, Blackwell AD, et al. A neuropsychological comparison of obsessive-compulsive disorder and trichotillomania. *Neuropsychologia*. 2007; 45:654–662. [PubMed: 17005210]

256. Leonard H, Swedo SE, Rapoport JL, et al. Treatment of obsessive-compulsive disorder with clomipramine and desipramine in children and adolescents: a double-blind crossover comparison. *Arch Gen Psychiatry*. 1989; 46:1088–1092. [PubMed: 2686576]
257. Stein DJ, Hollander E. Low-dose pimozide augmentation of serotonin reuptake blockers in the treatment of trichotillomania. *J Clin Psychiatry*. 1992; 53:123–126. [PubMed: 1532960]
258. Chamberlain SR, Orlaug BL, Boulougouris V, et al. Trichotillomania: neurobiology and treatment. *Neurosci Biobehav Rev*. 2009; 33:831–842. [PubMed: 19428495]
259. Bloch MH, Landeros-Weisenberger A, Dombrowski P, et al. Systematic review: pharmacological and behavioral treatment for trichotillomania. *Biol Psychiatry*. 2007; 62:839–846. [PubMed: 17727824]
260. Grant JE, Orlaug BL, Kim SW. N-acetylcysteine, a glutamate modulator, in the treatment of trichotillomania: a double-blind, placebo-controlled study. *Arch Gen Psychiatry*. 2009; 66:756–763. [PubMed: 19581567]
261. Walsh KH, McDougle CJ. Pharmacological strategies for trichotillomania. *Expert Opin Pharmacother*. 2005; 6:975–984. [PubMed: 15952925]
262. Woods DW, Miltenberger RG. Habit reversal: a review of applications and variations. *J Behav Ther Exp Psychiatry*. 1995; 26:123–131. [PubMed: 7593685]
263. Neziroglu F, McKay D, Yaryura-Tobias JA. Overlapping and distinctive features of hypochondriasis and obsessive-compulsive disorder. *J Anxiety Disord*. 2000; 14:603–614. [PubMed: 11918094]
264. Abramowitz JS, Olatunji BO, Deacon BJ. Health anxiety, hypochondriasis, and the anxiety disorders. *Behav Therapy*. 2007; 38:86–94.
265. Deacon BJ, Abramowitz J. Is hypochondriasis related to obsessive-compulsive disorder, panic disorder, or both? An empirical evaluation. *J Cogn Psychother*. 2008; 22:115–127.
266. Leibbrand R, Hiller W, Fichter MM. Hypochondriasis and somatization: two distinct aspects of somatoform disorders? *J Clin Psychol*. 2000; 56:63–72. [PubMed: 10661369]
267. Lieb R, Zimmermann P, Friis RH, et al. The natural course of DSM-IV somatoform disorders and syndromes among adolescents and young adults: a prospective-longitudinal community study. *Eur Psychiatry*. 2002; 17:321–331. [PubMed: 12457742]
268. Looper KJ, Kirmayer LJ. Hypochondriacal concerns in a community population. *Psychol Med*. 2001; 31:577–584. [PubMed: 11352360]
269. Noyes R Jr, Carney CP, Hillis SL, et al. Prevalence and correlates of illness worry in the general population. *Psychosomatics*. 2005; 46:529–539. [PubMed: 16288132]
270. Bleichhardt G, Hiller W. Hypochondriasis and health anxiety in the German population. *Br J Health Psychol*. 2007; 12:511–523. [PubMed: 17931470]
271. Martin A, Jacobi F. Features of hypochondriasis and illness worry in the general population in Germany. *Psychosom Med*. 2006; 68:770–777. [PubMed: 17012532]
272. du Toit PL, van Kradenburg J, Niehaus D, Stein DJ. Comparison of obsessive-compulsive disorder patients with and without comorbid putative obsessive-compulsive spectrum disorders using a structured clinical interview. *Compr Psychiatry*. 2001; 42:291–300. [PubMed: 11458303]
273. Abramowitz JS, Brigidi BD, Foa EB. Health concerns in patients with obsessive-compulsive disorder. *J Anxiety Disord*. 1999; 13:529–539. [PubMed: 10600053]
274. Escobar JI, Gara M, Waitzkin H, et al. DSM-IV hypochondriasis in primary care. *Gen Hosp Psychiatry*. 1998; 20:155–159. [PubMed: 9650033]
275. Bach M, Nutzinger DO, Hartl L. Comorbidity of anxiety disorders and hypochondriasis considering different diagnostic systems. *Compr Psychiatry*. 1996; 37:62–67. [PubMed: 8770529]
276. Barsky AJ, Barnett MC, Cleary PD. Hypochondriasis and panic disorder; boundary and overlap. *Arch Gen Psychiatry*. 1994; 51:918–925. [PubMed: 7944880]
277. Furer P, Walker JR, Chartier MJ, Stein MB. Hypochondriacal concerns and somatization in panic disorder. *Depress Anxiety*. 1997; 6:78–85. [PubMed: 9451549]
278. Fink P, Ornbol E, Toft T, et al. A new, empirically established hypochondriasis diagnosis. *Am J Psychiatry*. 2004; 161:1680–1691. [PubMed: 15337660]

279. Noyes R Jr, Kathol RG, Fisher MM, et al. Psychiatric comorbidity among patients with hypochondriasis. *Gen Hosp Psychiatry*. 1994; 16:78–87. [PubMed: 8039697]
280. Barsky AJ, Wyshak G, Klerman GL. Psychiatric comorbidity in DSM-III-R hypochondriasis. *Arch Gen Psychiatry*. 1992; 49:101–108. [PubMed: 1550462]
281. Barsky AJ, Fama JM, Bailey ED, Ahern DK. A prospective 4- to 5-year study of DSM-III-R hypochondriasis. *Arch Gen Psychiatry*. 1998; 55:737–744. [PubMed: 9707385]
282. 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. [PubMed: 1503140]
283. Ross S, Fallon BA, Petkova E, et al. Long-term follow-up study of patients with refractory obsessive-compulsive disorder. *J Neuropsychiatry Clin Neurosci*. 2008; 20:450–457. [PubMed: 19196930]
284. Skoog G, Skoog I. A 40-year follow-up of patients with obsessive-compulsive disorder. *Arch Gen Psychiatry*. 1999; 56:121–127. [PubMed: 10025435]
285. Rasmussen SA, Eisen JL. Epidemiology of obsessive compulsive disorder. *J Clin Psychiatry*. 1990; 51 (Suppl):10–13. [PubMed: 2404965]
286. Robins, LN.; Regier, DA. Psychiatric disorders in America: the epidemiologic catchment area study. New York: The Free Press; 1991.
287. Rasmussen SA, Tsuang MT. Epidemiologic and clinical findings of significance to the design of neuropharmacologic studies of obsessive-compulsive disorder. *Psychopharmacol Bull*. 1986; 22:723–729. [PubMed: 3797577]
288. Noyes R Jr, Holt CS, Happel RL, et al. A family study of hypochondriasis. *J Nerv Ment Dis*. 1997; 185:223–232. [PubMed: 9114807]
289. Torgersen S. Genetics of somatoform disorders. *Arch Gen Psychiatry*. 1986; 43:502–505. [PubMed: 3964029]
290. Barsky AJ, Wool C, Barnett MC, Cleary PD. Histories of childhood trauma in adult hypochondriacal patients. *Am J Psychiatry*. 1994; 151:397–401. [PubMed: 8109648]
291. Noyes R Jr, Stuart S, Langbehn DR, et al. Childhood antecedents of hypochondriasis. *Psychosomatics*. 2002; 43:282–289. [PubMed: 12189253]
292. Morrison J. Childhood sexual histories of women with somatization disorder. *Am J Psychiatry*. 1989; 146:239–241. [PubMed: 2912266]
293. Caspi A, Vishne T, Sasson Y, et al. Relationship between childhood sexual abuse and obsessive-compulsive disorder: case control study. *Isr J Psychiatry Relat Sci*. 2008; 45:177–182. [PubMed: 19398821]
294. van den Heuvel OA, Veltman DJ, Groenewegen HJ, et al. Disorder-specific neuroanatomical correlates of attentional bias in obsessive-compulsive disorder, panic disorder, and hypochondriasis. *Arch Gen Psychiatry*. 2005; 62:922–933. [PubMed: 16061770]
295. Niemi PM, Portin R, Aalto S, et al. Cognitive functioning in severe somatization—a pilot study. *Acta Psychiatr Scand*. 2002; 106:461–463. [PubMed: 12392490]
296. Fallon BA, Mathew SJ. Biological therapies for obsessive-compulsive disorder. *J Psychiatr Pract*. 2000; 6:113–128. [PubMed: 15990479]
297. Barsky AJ, Ahern DK. Cognitive behavior therapy for hypochondriasis: a randomized controlled trial. *JAMA*. 2004; 291:1464–1470. [PubMed: 15039413]
298. Fallon BA. Pharmacotherapy of somatoform disorders. *J Psychosom Res*. 2004; 56:455–460. [PubMed: 15094032]
299. Greeven A, van Balkom AJ, Visser S, et al. Cognitive behavior therapy and paroxetine in the treatment of hypochondriasis: a randomized controlled trial. *Am J Psychiatry*. 2007; 164:91–99. [PubMed: 17202549]
300. Abramowitz JS. Hypochondriasis: conceptualization, treatment, and relationship to obsessive compulsive disorder. *Ann Clin Psychiatry*. 2005; 17:211–217. [PubMed: 16402753]
301. Kroenke K. Efficacy of treatment for somatoform disorders: a review of randomized controlled trials. *Psychosom Med*. 2007; 69:881–888. [PubMed: 18040099]

302. Looper KJ, Kirmayer LJ. Behavioral medicine approaches to somatoform disorders. *J Consult Clin Psychol.* 2002; 70:810–827. [PubMed: 12090385]
303. Kroenke K, Swindle R. Cognitive-behavioral therapy for somatization and symptom syndromes: a critical review of controlled clinical trials. *Psychother Psychosom.* 2000; 69:205–215. [PubMed: 10867588]
304. Foa, EB.; Franklin, ME. Psychotherapies for obsessive-compulsive disorder: a review. In: Crino, R.; Maj, M.; Sartorius, N.; Okasha, A.; Zohar, J., editors. *Obsessive compulsive disorder.* Chichester: Wiley; 2000. p. 93-115.
305. Jenike MA. An update on obsessive-compulsive disorder. *Bull Menninger Clin.* 2001; 65:4–25. [PubMed: 11280957]
306. Fallon BA, Petkova E, Skritskaya N, et al. A double-masked, placebo-controlled study of fluoxetine for hypochondriasis. *J Clin Psychopharmacol.* 2008; 28:638–645. [PubMed: 19011432]
307. Simpson HB, Liebowitz MR, Foa EB, et al. Post-treatment effects of exposure therapy and clomipramine in obsessive-compulsive disorder. *Depress Anxiety.* 2004; 19:225–233. [PubMed: 15274171]
308. Walsh BT, Seidman SN, Sysko R, Gould M. Placebo response in studies of major depression: variable, substantial, and growing. *JAMA.* 2002; 287:1840–1847. [PubMed: 11939870]
309. The Clomipramine Collaborative Study Group. Clomipramine in the treatment of patients with obsessive-compulsive disorder. *Arch Gen Psychiatry.* 1991; 48:730–738. [PubMed: 1883256]
310. Simpson HB, Huppert JD, Petkova E, et al. Response versus remission in obsessive-compulsive disorder. *J Clin Psychiatry.* 2006; 67:269–276. [PubMed: 16566623]
311. Allen LA, Woolfolk RL, Escobar JI, et al. Cognitive-behavioral therapy for somatization disorder: a randomized controlled trial. *Arch Intern Med.* 2006; 166:1512–1518. [PubMed: 16864762]
312. Muller JE, Wentzel I, Koen L, et al. Escitalopram in the treatment of multisomatoform disorder: a double-blind, placebo-controlled trial. *Int Clin Psychopharmacol.* 2008; 23:43–48. [PubMed: 18090507]
313. Volz HP, Moller HJ, Reimann I, Stoll KD. Opipramol for the treatment of somatoform disorders: results from a placebo-controlled trial. *Eur Neuropsychopharmacol.* 2000; 10:211–217. [PubMed: 10793324]
314. Muller T, Mannel M, Murck H, Rahlfs VW. Treatment of somatoform disorders with St. John's wort: a randomized, double-blind and placebo-controlled trial. *Psychosom Med.* 2004; 66:538–547. [PubMed: 15272100]
315. Kroenke K, Messina N 3rd, Benattia I, et al. Venlafaxine extended release in the short-term treatment of depressed and anxious primary care patients with multisomatoform disorder. *J Clin Psychiatry.* 2006; 67:72–80. [PubMed: 16426091]
316. Thomson AB, Page LA. Psychotherapies for hypochondriasis. *Cochrane Database Syst Rev.* 2007:CD006520. [PubMed: 17943915]
317. Pilowsky I. The concept of abnormal illness behavior. *Psychosomatics.* 1990; 31:207–213. [PubMed: 2184454]
318. Fallon BA, Qureshi AI, Laje G, Klein B. Hypochondriasis and its relationship to obsessive-compulsive disorder. *Psychiatr Clin North Am.* 2000; 23:605–616. [PubMed: 10986730]
319. Kroenke K, Sharpe M, Sykes R. Revising the classification of somatoform disorders: key questions and preliminary recommendations. *Psychosomatics.* 2007; 48:277–285. [PubMed: 17600162]
320. Steketee G, Frost R. Compulsive hoarding: current status of the research. *Clin Psychol Rev.* 2003; 23:905–927. [PubMed: 14624821]
321. Wu KD, Watson D. Hoarding and its relation to obsessive-compulsive disorder. *Behav Res Ther.* 2005; 43:897–921. [PubMed: 15896286]
322. Abramowitz JS, Wheaton MG, Storch EA. The status of hoarding as a symptom of obsessive-compulsive disorder. *Behav Res Ther.* 2008; 46:1026–1033. [PubMed: 18684434]
323. Frost RO, Tolin DF, Steketee G, et al. Excessive acquisition in hoarding. *J Anxiety Disord.* 2009; 23:632–639. [PubMed: 19261435]

324. 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. [PubMed: 10484953]
325. Frost, RO.; Steketee, G.; Tolin, DF.; Brown, T. *Eur Assoc Behav Cog Ther. Paris*: 2006. Diagnostic issues in compulsive hoarding.
326. Pertusa A, Fullana MA, Singh S, et al. Compulsive hoarding: OCD symptom, distinct clinical syndrome, or both? *Am J Psychiatry*. 2008; 165:1289–1298. [PubMed: 18483134]
327. Frost, R.; Steketee, G.; Tolin, D.; Glossner, K. *Diagnostic comorbidity in hoarding and OCD. World Congress of Behavioral and Cognitive Therapies; Boston, MA*. 2010.
328. Mueller A, Mueller U, Albert P, et al. Hoarding in a compulsive buying sample. *Behav Res Ther*. 2007; 45:2754–2763. [PubMed: 17868641]
329. Mueller A, Mitchell JE, Crosby RD, et al. The prevalence of compulsive hoarding and its association with compulsive buying in a German population-based sample. *Behav Res Ther*. 2009; 47:705–709. [PubMed: 19457476]
330. Frost RO, Meagher BM, Riskind JH. Obsessive-compulsive features in pathological lottery and scratch-ticket gamblers. *J Gamb Stud*. 2001; 17:5–19. [PubMed: 11705017]
331. Samuels J, Bienvenu OJ 3rd, Riddle MA, et al. Hoarding in obsessive compulsive disorder: results from a case-control study. *Behav Res Ther*. 2002; 40:517–528. [PubMed: 12043707]
332. 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; 102:199–202. [PubMed: 11008855]
333. Frost RO, Steketee G, Williams LF, Warren R. Mood, personality disorder symptoms and disability in obsessive compulsive hoarders: a comparison with clinical and nonclinical controls. *Behav Res Ther*. 2000; 38:1071–1081. [PubMed: 11060936]
334. Samuels JF, Bienvenu OJ 3rd, Pinto A, et al. Hoarding in obsessive-compulsive disorder: results from the OCD Collaborative Genetics Study. *Behav Res Ther*. 2007; 45:673–686. [PubMed: 16824483]
335. Pertusa A, Frost RO, Fullana MA, et al. Refining the diagnostic boundaries of compulsive hoarding: A critical review. *Clin Psychol Rev*. 2010
336. Frost RO, Gross RC. The hoarding of possessions. *Behav Res Ther*. 1993; 31:367–381. [PubMed: 8512538]
337. Grisham JR, Frost RO, Steketee G, et al. Age of onset of compulsive hoarding. *J Anxiety Disord*. 2006; 20:675–686. [PubMed: 16112837]
338. Ayers CR, Saxena S, Golshan S, Wetherell JL. Age at onset and clinical features of late life compulsive hoarding. *Int J Geriatr Psychiatry*. 2009; 25:142–149. [PubMed: 19548272]
339. Winsberg ME, Cassic KS, Koran LM. Hoarding in obsessive-compulsive disorder: a report of 20 cases. *J Clin Psychiatry*. 1999; 60:591–597. [PubMed: 10520977]
340. Zhang H, Leckman JF, Pauls DL, et al. Genomewide scan of hoarding in sib pairs in which both sibs have Gilles de la Tourette syndrome. *Am J Hum Gene*. 2002; 70:896–904.
341. Samuels J, Shugart YY, Grados MA, et al. Significant linkage to compulsive hoarding on chromosome 14 in families with obsessive-compulsive disorder: results from the OCD Collaborative Genetics Study. *Am J Psychiatry*. 2007; 164:493–499. [PubMed: 17329475]
342. Hartl TL, Duffany SR, Allen GJ, et al. Relationships among compulsive hoarding, trauma, and attention-deficit/hyperactivity disorder. *Behav Res Ther*. 2005; 43:269–276. [PubMed: 15629755]
343. Cromer KR, Schmidt NB, Murphy DL. Do traumatic events influence the clinical expression of compulsive hoarding? *Behav Res Ther*. 2007; 45:2581–2592. [PubMed: 17673166]
344. Samuels JF, Bienvenu OJ, Grados MA, et al. Prevalence and correlates of hoarding behavior in a community-based sample. *Behav Res Ther*. 2008; 46:836–844. [PubMed: 18495084]
345. An SK, Mataix-Cols D, Lawrence NS, et al. To discard or not to discard: the neural basis of hoarding symptoms in obsessive-compulsive disorder. *Mol Psychiatry*. 2009; 14:318–331. [PubMed: 18180763]

346. Saxena S, Brody AL, Maidment KM, et al. Cerebral glucose metabolism in obsessive-compulsive hoarding. *Am J Psychiatry*. 2004; 161:1038–1048. [PubMed: 15169692]
347. Tolin DF, Kiehl KA, Worhunsky P, et al. An exploratory study of the neural mechanisms of decision making in compulsive hoarding. *Psychol Med*. 2009; 39:325–336. [PubMed: 18485263]
348. Saxena S. Neurobiology and treatment of compulsive hoarding. *CNS Spectr*. 2008; 13:29–36. [PubMed: 18849909]
349. Saxena S, Brody AL, Maidment KM, Baxter LR Jr. Paroxetine treatment of compulsive hoarding. *J Psychiatr Res*. 2007; 41:481–487. [PubMed: 16790250]
350. Cannistraro PA, Rauch SL. Neural circuitry of anxiety: evidence from structural and functional neuroimaging studies. *Psychopharmacol Bull*. 2003; 37:8–25. [PubMed: 15131515]
351. Steketee G, Frost R, Kyrios M. Cognitive aspects of compulsive hoarding. *Cog Ther Res*. 2003; 27:463–479.
352. Kyrios M, Frost RO, Steketee G. Cognitions in compulsive buying and acquisition. *Cognit Ther Res*. 2004; 28:241–258.
353. Wincze JP, Steketee G, Frost RO. Categorization in compulsive hoarding. *Behav Res Ther*. 2007; 45:63–72. [PubMed: 16530724]
354. Luchian SA, McNally RJ, Hooley JM. Cognitive aspects of nonclinical obsessive-compulsive hoarding. *Behav Res Ther*. 2007; 45:1657–1662. [PubMed: 17014824]
355. Hartl TL, Frost RO, Allen GJ, et al. Actual and perceived memory deficits in individuals with compulsive hoarding. *Depress Anxiety*. 2004; 20:59–69. [PubMed: 15390215]
356. Muroff J, Steketee G, Rasmussen J, et al. Group cognitive and behavioral treatment for compulsive hoarding: a preliminary trial. *Depress Anxiety*. 2009; 26:634–640. [PubMed: 19569229]
357. Tolin DF, Frost RO, Steketee G. An open trial of cognitive-behavioral therapy for compulsive hoarding. *Behav Res Ther*. 2007; 45:1461–1470. [PubMed: 17306221]
358. Mataix-Cols D, Marks IM, Greist JH, et al. Obsessive-compulsive symptom dimensions as predictors of compliance with and response to behaviour therapy: results from a controlled trial. *Psychother Psychosom*. 2002; 71:255–262. [PubMed: 12207105]
359. 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. [PubMed: 14622080]
360. Stein DJ, Carey PD, Lochner C, et al. Escitalopram in obsessive-compulsive disorder: response of symptom dimensions to pharmacotherapy. *CNS Spectr*. 2008; 13:492–498. [PubMed: 18567973]
361. Rheaume J, Freeston MH, Dugas MJ, et al. Perfectionism, responsibility and obsessive-compulsive symptoms. *Behav Res Ther*. 1995; 33:785–794. [PubMed: 7677716]
362. Nelson EA, Abramowitz JS, Whiteside SP, Deacon BJ. Scrupulosity in patients with obsessive-compulsive disorder: relationship to clinical and cognitive phenomena. *J Anxiety Disord*. 2006; 20:1071–1086. [PubMed: 16524696]
363. World Health Organization. ICD-10 classification of mental and behavioural disorders: diagnostic criteria for research. World Health Organization; Geneva: 1992.
364. Fineberg NA, Sharma P, Sivakumaran T, et al. Does obsessive-compulsive personality disorder belong within the obsessive-compulsive spectrum? *CNS Spectr*. 2007; 12:467–482. [PubMed: 17545957]
365. Skodol AE, Gunderson JG, McGlashan TH, et al. Functional impairment in patients with schizotypal, borderline, avoidant, or obsessive-compulsive personality disorder. *Am J Psychiatry*. 2002; 159:276–283. [PubMed: 11823271]
366. Blashfield RK, Intoccia V. Growth of the literature on the topic of personality disorders. *Am J Psychiatry*. 2000; 157:472–473. [PubMed: 10698831]
367. O'Connor BP. A search for consensus on the dimensional structure of personality disorders. *J Clin Psychol*. 2005; 61:323–345. [PubMed: 15468325]
368. Abraham, K. Contribution to the theory of anal character. In: Lewin, BD., editor. *On character and libido development*. New York, N.Y.: W.W. Norton and Co; 1966.

369. Pinto A, Mancebo MC, Eisen JL, et al. The Brown Longitudinal Obsessive Compulsive Study: clinical features and symptoms of the sample at intake. *J Clin Psychiatry*. 2006; 67:703–711. [PubMed: 16841619]
370. Samuels J, Nestadt G, Bienvenu OJ, et al. Personality disorders and normal personality dimensions in obsessive-compulsive disorder. *Br J Psychiatry*. 2000; 177:457–462. [PubMed: 11060001]
371. Coles ME, Pinto A, Mancebo MC, et al. OCD with comorbid OCPD: a subtype of OCD? *J Psychiatr Res*. 2008; 42:289–296. [PubMed: 17382961]
372. Rossi A, Marinangeli MG, Butti G, et al. Personality disorders in bipolar and depressive disorders. *J Affect Disord*. 2001; 65:3–8. [PubMed: 11426507]
373. Farmer R, Nelson-Gray RO. Personality disorders and depression: hypothetical relations, empirical findings, and methodological considerations. *Clin Psychol Rev*. 1990; 10:453–476.
374. Albert U, Maina G, Forner F, Bogetto F. DSM-IV obsessive-compulsive personality disorder: prevalence in patients with anxiety disorders and in healthy comparison subjects. *Compr Psychiatry*. 2004; 45:325–332. [PubMed: 15332194]
375. Nilsson EW, Gillberg C, Gillberg IC, Rastam M. Ten-year follow-up of adolescent-onset anorexia nervosa: personality disorders. *J Am Acad Child Adolesc Psychiatry*. 1999; 38:1389–1395. [PubMed: 10560225]
376. Anderluh MB, Tchanturia K, Rabe-Hesketh S, Treasure J. Childhood obsessive-compulsive personality traits in adult women with eating disorders: defining a broader eating disorder phenotype. *Am J Psychiatry*. 2003; 160:242–247. [PubMed: 12562569]
377. Grilo CM, McGlashan TH. Convergent and discriminant validity of DSM-IV axis II personality disorder criteria in adult outpatients with binge eating disorder. *Compr Psychiatry*. 2000; 41:163–166. [PubMed: 10834623]
378. Karwautz A, Troop NA, Rabe-Hesketh S, et al. Personality disorders and personality dimensions in anorexia nervosa. *J Personal Disord*. 2003; 17:73–85.
379. Sansone RA, Levitt JL, Sansone LA. The prevalence of personality disorders among those with eating disorders. *Eat Disord J Treat Prevent*. 2005; 13:7–21.
380. Altindag A, Yanik M, Nebioglu M. Comorbid personality disorders in subjects with bipolar I disorder. *Int J Psychiatry Clin Pract*. 2006; 10:33–37.
381. Altman SE, Shankman SA. What is the association between obsessive-compulsive disorder and eating disorders? *Clin Psychol Rev*. 2009; 29:638–646. [PubMed: 19744759]
382. Angst J, Gamma A, Endrass J, et al. Obsessive-compulsive syndromes and disorders: significance of comorbidity with bipolar and anxiety syndromes. *Eur Arch Psychiatry Clin Neurosci*. 2005; 255:65–71. [PubMed: 15711895]
383. Mancebo MC, Eisen JL, Grant JE, Rasmussen SA. Obsessive compulsive personality disorder and obsessive compulsive disorder: clinical characteristics, diagnostic difficulties, and treatment. *Ann Clin Psychiatry*. 2005; 17:197–204. [PubMed: 16402751]
384. Shea MT, Stout RL, Yen S, et al. Associations in the course of personality disorders and Axis I disorders over time. *J Abnorm Psychol*. 2004; 113:499–508. [PubMed: 15535783]
385. Hummelen B, Wilberg T, Pedersen G, Karterud S. The quality of the DSM-IV obsessive-compulsive personality disorder construct as a prototype category. *J Nerv Ment Dis*. 2008; 196:446–455. [PubMed: 18552621]
386. Reichborn-Kjennerud T, Czajkowski N, Neale MC, et al. Genetic and environmental influences on dimensional representations of DSM-IV cluster C personality disorders: a population-based multivariate twin study. *Psychol Med*. 2007; 37:645–653. [PubMed: 17134532]
387. Heyman I, Fombonne E, Simmons H, et al. Prevalence of obsessive-compulsive disorder in the British nationwide survey of child mental health. *Br J Psychiatry*. 2001; 179:324–329. [PubMed: 11581112]
388. Carballo JJ, Baca-Garcia E, Blanco C, et al. Stability of childhood anxiety disorder diagnoses: a follow-up naturalistic study in psychiatric care. *Eur Child Adolesc Psychiatry*. 2009
389. Thomsen PH, Jensen J. Obsessive-compulsive disorder: admission patterns and diagnostic stability, a case-register study. *Acta Psychiatr Scand*. 1994; 90:19–24. [PubMed: 7976444]



390. Light KJ, Joyce PR, Luty SE, et al. Preliminary evidence for an association between a dopamine D3 receptor gene variant and obsessive-compulsive personality disorder in patients with major depression. *Am J Med Genet B Neuropsychiatr Genet.* 2006; 141B:409–413. [PubMed: 16583407]
391. Perez M, Brown JS, Vrshek-Schallhorn S, et al. Differentiation of obsessive-compulsive-, panic-, obsessive-compulsive personality-, and non-disordered individuals by variation in the promoter region of the serotonin transporter gene. *J Anxiety Disord.* 2006; 20:794–806. [PubMed: 16303282]
392. Torgersen S, Lygren S, Oien PA, et al. A twin study of personality disorders. *Compr Psychiatry.* 2000; 41:416–425. [PubMed: 11086146]
393. Calvo R, Lazaro L, Castro-Fornieles J, et al. Obsessive-compulsive personality disorder traits and personality dimensions in parents of children with obsessive-compulsive disorder. *Eur Psychiatry.* 2009; 24:201–206. [PubMed: 19118984]
394. Lilenfeld LR, Kaye WH, Greeno CG, et al. A controlled family study of anorexia nervosa and bulimia nervosa: psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Arch Gen Psychiatry.* 1998; 55:603–610. [PubMed: 9672050]
395. Lougee L, Perlmutter SJ, Nicolson R, et al. Psychiatric disorders in first-degree relatives of children with pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS). *J Am Acad Child Adolesc Psychiatry.* 2000; 39:1120–1126. [PubMed: 10986808]
396. Nordahl HM, Stiles TC. Perceptions of parental bonding in patients with various personality disorders, lifetime depressive disorders, and healthy controls. *J Personal Disord.* 1997; 11:391–402.
397. Lennertz L, Grabe HJ, Ruhrmann S, et al. Perceived parental rearing in subjects with obsessive-compulsive disorder and their siblings. *Acta Psychiatr Scand.* 2009 epub ahead of print.
398. Yoshida T, Taga C, Matsumoto Y, Fukui K. Paternal overprotection in obsessive-compulsive disorder and depression with obsessive traits. *Psychiatry Clin Neurosci.* 2005; 59:533–538. [PubMed: 16194254]
399. Monteleone P, Brambilla F, Bortolotti F, et al. Prolactin response to d-fenfluramine is blunted in people with anorexia nervosa. *Br J Psychiatry.* 1998; 172:439–442. [PubMed: 9747408]
400. Monteleone P, Brambilla F, Bortolotti F, et al. Plasma prolactin response to d-fenfluramine is blunted in bulimic patients with frequent binge episodes. *Psychol Med.* 1998; 28:975–983. [PubMed: 9723152]
401. Stein DJ, Trestman RL, Mitropoulou V, et al. Impulsivity and serotonergic function in compulsive personality disorder. *J Neuropsychiatry Clin Neurosci.* 1996; 8:393–398. [PubMed: 9116474]
402. Fineberg NA, Roberts A, Montgomery SA, Cowen PJ. Brain 5-HT function in obsessive-compulsive disorder: prolactin responses to d-fenfluramine. *Br J Psychiatry.* 1997; 171:280–282. [PubMed: 9337985]
403. Clark LA. Temperament as a unifying basis for personality and psychopathology. *J Abnorm Psychol.* 2005; 114:505–521. [PubMed: 16351374]
404. [www.camcog.com](http://www.camcog.com).
405. Chamberlain SR, Fineberg NA, Menzies LA, et al. Impaired cognitive flexibility and motor inhibition in unaffected first-degree relatives of patients with obsessive-compulsive disorder. *Am J Psychiatry.* 2007; 164:335–338. [PubMed: 17267798]
406. Patel DD, Laws KR, Padhi A, et al. The neuropsychology of the schizo-obsessive subtype of schizophrenia: a new analysis. *Psychol Med.* 2009:1–13. [PubMed: 19335938]
407. Ansseau M. Serotonergic antidepressants in obsessive personality. *L'Encephale.* 1996; 22:309–310.
408. Volavka J, Neziroglu F, Yaryura-Tobias JA. Clomipramine and imipramine in obsessive-compulsive disorder. *Psychiatry Res.* 1985; 14:85–93. [PubMed: 3887445]
409. Rinne T, van den Brink W, Wouters L, van Dyck R. SSRI treatment of borderline personality disorder: a randomized, placebo-controlled clinical trial for female patients with borderline personality disorder. *Am J Psychiatry.* 2002; 159:2048–2054. [PubMed: 12450955]

410. Feusner JD, Phillips KA, Stein DJ. Olfactory reference syndrome: issues for DSM-V. *Depress Anxiety*. in press.
411. Keuthen NJ, Wilhelm S, Deckersbach T, et al. The Skin Picking Scale: scale construction and psychometric analyses. *J Psychosom Res*. 2001; 50:337–341. [PubMed: 11438115]
412. O’Sullivan RL, Keuthen NJ, Hayday CF, et al. The Massachusetts General Hospital (MGH) hairpulling scale: 2. reliability and validity. *Psychother Psychosom*. 1995; 64:146–148. [PubMed: 8657845]
413. Keuthen NJ, O’Sullivan RL, Ricciardi JN, et al. The Massachusetts General Hospital (MGH) hairpulling scale: 1. development and factor analyses. *Psychother Psychosom*. 1995; 64:141–145. [PubMed: 8657844]
414. McKay D, Neziroglu F. Methodological issues in the obsessive-compulsive spectrum. *Psychiatry Res*. 2009; 170:61–65. [PubMed: 19804912]
415. Phillips, KA.; Price, LH.; Greenberg, BD.; Rasmussen, SA. Should DSM’s diagnostic groupings be changed?. In: Phillips, KA.; First, MB.; Pincus, HA., editors. *Advancing DSM: dilemmas in psychiatric diagnosis*. Washington, DC: American Psychiatric Publishing Inc; 2003. p. 57-84.
416. Hyman, SE. Forward. In: Phillips, KA.; First, MB.; Pincus, HA., editors. *Advancing DSM: dilemmas in psychiatric diagnosis*. Washington, D.C: American Psychiatric Association; 2003. p. xi-xxi.
417. Andrews G, Goldberg DP, Krueger RF, et al. Exploring the feasibility of a meta-structure for DSM-V and ICD-11: could it improve utility and validity? *Psychol Med*. 2009; 39:1993–2000. [PubMed: 19796425]