

Obsessive Compulsive Related Disorders: A New Classification for the DSM-V

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There is concern surrounding the classification of obsessive compulsive disorder (OCD) for the next edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). The DSM-V workgroup for OCD related disorders and researchers alike suggest removing OCD from the anxiety disorders category of the DSM, and placing it under the heading of obsessive compulsive related disorders (OCD). This paper provides a review of current literature examining three popular candidates (body dysmorphic disorder, trichotillomania, and Tourette's disorder) for inclusion in the OCD category. Recommendations for future research are made.

The classification of obsessive compulsive disorder (OCD) has undergone considerable change since the initial compilation of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM). In the DSM-II, OCD was categorized as "obsessive-compulsive neurosis," while obsessive compulsive disorder received a new classification in the DSM-III as an anxiety disorder. Currently, OCD remains classified as an anxiety disorder in the DSM-IV-TR (Montgomery, 1993); however, the classification of OCD continues to be a topic of controversy for the DSM-V.

Researchers have recently suggested that the current classification of OCD is mainly symptom based. Specifically, obsessive thoughts increase anxiety which is moderated by compulsive behaviors (Bartz & Hollander, 2006). As such, anxiety presents as a byproduct of the disorder and not an underlying feature (Mataix-Cols, Pertusa, & Leckman, 2007). Additionally, the current classification fails to account for the differences among OCD and the remaining anxiety disorders, mainly in the sphere of repetitive behaviors and an inability to resist impulses. Several other disorders (e.g., body dysmorphic disorder, trichotillomania, and Tourette's disorder) that are not included in the anxiety disorders category also display the compulsive and impulsive behaviors present in OCD. Thus, the Research Planning Agenda for the DSM-V Workgroup on obsessive compulsive-related disorders suggests removing OCD from the anxiety disorders category and placing it under the title of obsessive compulsive related disorders (OCDs) (Hollander, Braun, & Simeon, 2008).

Other recent work has proposed a multidimensional model of OCD that recognizes the heterogeneity among causes and presenting symptoms across OCD and related disorders (Castle & Phillips, 2006; Mataix-Cols, 2007). It is suggested that OCD and related disorders be classified along a continuum for future editions of the DSM. The OCD spectrum is subdivided into three distinct clusters; (1) preoccupations with body sensations or image (e.g., body

dysmorphic disorder), (2) impulse disorders (e.g., trichotillomania), and (3) neurological based disorders (e.g., Tourette's disorder). These seemingly unrelated disorders display consistency across (1) symptomology (e.g., compulsivity and impulsivity), (2) neurobiology (e.g., fMRI data), and (3) responses to psychosocial (e.g., cognitive behavioral therapy) and pharmacological (selective serotonin reuptake inhibitors) treatments (Castle & Phillips, 2006; Rossi, 2006). Further, Hollander (1993) suggests a "compulsive-impulsive" dimension, with compulsive disorders (e.g., OCD) on one end of the spectrum (compulsive) and impulse control disorders (e.g., trichotillomania) at the other end of the spectrum (impulsive). In this case, compulsivity reflects harm avoidance, whereas impulsivity reflects risk seeking.

Obsessive compulsive symptoms are present in various disorders besides OCD. These disorders include Tourette's disorder (TD), body dysmorphic disorder (BDD), and trichotillomania (TMM) (Castle & Phillips, 2006). While these disorders are primary candidates for OCDs, eating disorders, autism, and depersonalization disorder, along with certain neurological disorders such as Parkinson's and Sydenham's chorea, are also under consideration for inclusion within the OCD domain (Castle & Phillips, 2006; Hollander, 1993). What is more, recent literature revealed that the DSM-V Workgroup on obsessive compulsive-related disorders labored over whether or not to include behavioral addictions, such as pathological gambling, kleptomania, and pyromania within the OCDs. The workgroup concluded these behavioral addictions will not be classified as OCDs, but rather will be included in a parallel category of behavioral and substance addictions. The behavioral and substance addictions category will include existing as well as new (compulsive buying, internet addiction, and compulsive sexual behavior) impulse/control disorders as well as substance addictions (Bartz & Hollander, 2006).

This paper aims to elaborate on the process of the new classification proposed for the OCDs by examining the

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three contending disorders (body dysmorphic disorder, trichotillomania, and, Tourette's disorder) with regard to the symptom domain, the neurobiological and genetic domain, and the treatment domain. Suggestions for future research are recommended as well.

Body Dysmorphic Disorder and OCD

The Symptom Domain

Body dysmorphic disorder is currently classified as a somatoform disorder, and is marked by an excessive preoccupation with a perceived body defect which is usually non-existent or minimal (American Psychiatric Association, 2000; McKay, Neziroglu, & Yaryura-Tobias, 1997). The experienced preoccupations are believed to be similar to the obsessions experienced in OCD; obsessions in OCD and BDD tend to be intrusive and persistent. Additionally, individuals with these disorders recognize the obsessions as excessive. However, unlike individuals with OCD, individuals with BDD do not view their obsessions as unreasonable (Hollander, Braun, & Simeon, 2008). This may be due to the lack of global insight often seen in individuals with BDD as compared to those with OCD who tend to have more insight and recognize their obsessive thoughts as excessive and harmful (Phillips et al., 2007).

As evidenced in OCD, individuals with BDD engage in compulsive behaviors (e.g., mirror checking) and reassurance seeking in attempts to reduce the stress surrounding the preoccupation. The repetitive behaviors often are expressed in a ritualized form and may follow a certain sequence. Furthermore, this sequence is repeated if interrupted or until a desired aim is achieved (Phillips, McElroy, Keck, Pope, & Hudson, 1994). In both groups, the compulsions are not inherently gratifying or pleasurable, but rather distressing and socially debilitating. While both individuals with OCD and BDD engage in compulsive behaviors, these activities are less likely to reduce anxiety, behaviors, or compulsions in individuals with BDD (Hollander, Braun, & Simeon, 2008), which might be attributed to the poorer insight observed in individuals with BDD.

McKay, Neziroglu, and Yaryura-Tobias (1997) examined the overlapping symptom domains of OCD and BDD. A sample of 22 OCD outpatients and 23 BDD outpatients were administered a variety of psychological measures assessing OCD, BDD, overvalued ideation, depression, physical symptoms of anxiety, and cognitive symptoms of anxiety. The researchers found that BDD is a more severe variation of OCD. Participants with BDD reported higher levels of overvalued ideas when compared to those with OCD. Additionally, the BDD participants exhibited more severe obsessive and compulsive symptoms than the OCD participants. The two groups were statistically equivalent on other measures of depression and physical symptoms of anxiety; however, there was a significant difference between the two groups on the measure of

cognitive symptoms of anxiety, with the OCD participants reporting higher incidences. These results suggest a difference in the experience of anxiety associated with each disorder, on the one hand, while, on the other, that those with OCD and BDD similarly experience obsessive and compulsive symptoms. The relatedness between the two disorders in the symptom domain provides evidence in favor of an OCD spectrum.

More recently, Phillips and colleagues (2007) conducted a comparison study of clinical features of OCD and BDD. The researchers administered the structured clinical interview and a variety of psychological measures assessing symptoms of OCD, BDD, beliefs, and depression with 210 OCD participants, 45 BDD participants, and 40 comorbid OCD/BDD participants. A comparison of subjects' characteristics showed OCD and BDD did not significantly differ in regard to demographic traits such as age, race, gender, age of onset, illness duration, most functioning measures, and most comorbidity. Findings suggest that subjects with BDD were more likely to suffer from lifetime major depression or another mood disorder. However, the symptom severity of these disorders did not differ significantly among the groups, suggesting that BDD may be a good candidate for the OCDs within the symptom domain.

The Neurological and Genetic Domain

The brain circuitry within OCD patients is characterized by hyperactivity in the orbital frontal cortex, caudate nucleus, thalamus, and the anterior cingulate nucleus (Bartz & Hollander, 2006). Researchers have noted that the prefrontal regions of the brain, specifically, the frontal-striatal region including the basal ganglia, have been linked with the intrusive thoughts associated with OCD (Stein, 2000). Unfortunately, few neuroimaging studies in BDD are available for comparison (Mataix-Cols & van den Heuvel, 2006). Researchers conducted a morphometric study with 8 women with BDD and 8 female controls, and results showed a significantly different asymmetry in the caudate nucleus with a leftward shift in the BDD group. The activity in the caudate nucleus suggests that a similar brain region is active in both OCD and BDD (Mataix-Cols & van den Heuvel, 2006); however, research in this area is sparse and requires replication.

Bienvenu et al. (2000) examined the relationship among OCD and prospective spectrum disorders. The researchers found that cases of BDD were significantly higher in case probands than controls. BDD was also shown to be transmitted in families of patients with OCD, while BDD rates were higher in families with patients of OCD. The researchers suggest developing phenotypic definitions for future genetic research of OCD spectrum disorders (Bienvenu et al., 2000).

The Treatment Domain

Obsessive compulsive disorder and body dysmorphic disorder both respond to psychosocial and psychopharmacological treatments (Hollander et al., 2008). The leading psychosocial treatments for OCD are cognitive and behavioral-based therapies that focus on exposure and response prevention (ERP). ERP is considered the preferred psychosocial treatment for OCD (Castle & Phillips, 2006; Wilhelm et al., 2005). One study suggests that 63% of OCD patients respond positively to ERPs; however, 20% to 30% remain resistant or refuse ERP due to the anxiety provoking exposure (Wilhelm et al., 2005).

While OCD responds favorably to ERP alone, the cognitive component of cognitive behavioral therapy (CBT) has also been efficacious (Castle & Phillips, 2006). Wilhelm and colleagues (2005) examined the effects of a purely cognitive treatment for individuals with OCD. Participants received 12 weekly 50-60 minute individual sessions of cognitive therapy. The cognitive therapy included psychoeducation, procedures according to Beck's cognitive model (e.g., Socratic dialogue and identification of cognitive errors), and relapse prevention strategies. The researchers found that all participants improved across OCD symptoms as well anxiety symptoms and maladaptive beliefs. While results support the efficacy of cognitive treatment alone, this was the first study to examine purely cognitive techniques with an OCD sample, and further work is needed to make more conclusive statements regarding this therapeutic approach.

The treatment approach for BDD is based on the treatment of OCD (Neziroglu & Khemlani-Patel, 2003), such as psychosocial interventions that include a cognitive component as well as exposure and response prevention treatment to lessen anxiety and ritualistic behaviors (Buhlmann, Reese, Renaud, & Wilhelm, 2008; Castle & Phillips, 2006). While the literature on BDD remains fairly limited, the available research suggests that BDD responds to similar psychosocial treatments as OCD (Neziroglu, 2008). Neziroglu, McKay, and Yaryura-Tobias (1996) presented a case series in which 17 patients with BDD received daily 90-minute CBT sessions over the course of 1 month. Results showed a 50% reduction in symptoms in more than half the participants ($n = 12$). Veale and colleagues (1996) conducted a randomized study assessing the effectiveness of CBT with individuals with BDD. Nineteen individuals with BDD were randomly assigned to either the treatment group or the waitlist control group. Participants in the treatment group attended weekly 1-hour sessions of CBT for 12 weeks, and those that received treatment improved significantly more than those in the waitlist condition. In a 6-week intensive treatment of BDD using exposure response and prevention (without a cognitive component), 10 participants received five 90-minute sessions a week. The results indicated that participants improved on measures of BDD symptoms, avoidance, depression, and

anxiety, while follow-up data showed that improvements remained stable over a 6-week period (McKay et al., 1997).

With regard to psychopharmacological treatments, OCD and BDD respond favorably to selective serotonin reuptake inhibitors (SSRIs) (Castle & Phillips, 2006; Hollander et al., 2008). In a meta-analysis of treatments employing long-term medication for OCD, researchers found that SSRIs were, overall, effective over time and also helpful in relapse prevention. Data suggest that paroxetine, escitalopram, and fluoxetine are the most effective SSRIs in relapse prevention of OCD (Fineberg, Pampaloni, Pallanti, Ipser, & Stein, 2007). Studies to date often report that SSRIs are also efficacious for BDD (Phillips, Didie, Feusner, & Wilhelm, 2008). In a 12-week double-blind study ($N = 67$), fluoxetine was significantly more efficacious than placebo for BDD beginning at 8 weeks and continuing at 10 and 12 weeks and with participants receiving a relatively high dose of the medication (77.7 ± 8.0 mg/d) (Phillips, Albertini, & Rasmussen, 2002). This finding is in accordance with previous reports that psychopharmacological treatment of BDD often has a longer delay in SSRI response and requires higher SSRI doses (Neziroglu & Khemlani-Patel, 2003). Literature shows similar information for the delay in SSRI response and effective treatment dose with OCD (Castle & Phillips, 2006; Hollander et al., 2008).

Trichotillomania

The Symptom Domain

Trichotillomania (TTM) is currently classified as an impulse control disorder characterized by repetitive hair pulling (American Psychiatric Association, 2000). While TTM is not distinctly marked by obsessions, they are sometimes present in the disorder. When obsessions are present, they often are not associated with an intrusive thought, but rather with the compulsive desire to pluck one's hair, and this urge is recognized as unreasonable as intrusive thoughts are to individuals with OCD (Hollander et al., 2008). The ritualized and repetitive hair pulling associated with TTM is parallel to compulsions expressed in OCD. Much like compulsive behaviors exhibited in OCD, the compulsive hair plucking often reduces anxiety and is influenced by an individual's need for symmetry in the hair pattern (Hollander et al., 2008). Often the compulsive behavior is rigid and repeated until the desired outcome is achieved; hair plucking is a tension reducing response to the urge to pluck one's hair. Furthermore, hair plucking is distressing and socially debilitating as seen in OCD, and, while both hair pulling and OCD compulsions reduce anxiety, hair pulling is gratifying (Castle & Phillips, 2006).

To the author's knowledge, few studies exist directly comparing observable behavior of OCD to TTM in terms of symptomology. Bohne, Savage, Deckersbach, Keuthen, and Wilhelm (2008) compared motor inhibition abilities in TTM and OCD patients. Reaction times were measured using a *GoNogo* task among 25 TTM, 21 OCD, and 26 control

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participants. The researchers reported no significant difference in reaction times among the TTM, OCD, and healthy control groups; however, there was a small portion of the TTM group that performed either “fast and inaccurate” or “slow and accurate.” This portion of the TTM group was associated with an earlier age of onset, suggesting a subgroup of TTM sufferers that experience increased motor inhibition. With that said, overall, findings do not represent a significant difference in reactions between individuals with TTM and OCD, and further work exploring motor inhibition in TTM and OCD is warranted.

The Neurological and Genetic Domain

The neuroimaging data on TTM is limited (Mataix-Cols & van den Heuvel, 2006; Stein, 2000). Researchers suggest the caudate nucleus, a brain structure implicated in OCD, is not significantly involved in trichotillomania. Stein and colleagues (1997) compared caudate nucleus volumes in 13 OCD participants, 17 TTM participants, and 12 healthy controls. Results showed no significant differences among the groups. In a study with 10 women with TTM, researchers found clomipramine (a tricyclic antidepressant often used in OCD) was negatively correlated with anterior cingulate and orbitofrontal metabolism. Additionally, findings suggest that symptom severity of hair pulling is correlated with decreased perfusion in the frontal, parietal, and striatum brain regions (Stein et al., 2002). These findings agree with prior neuroimaging work on OCD, suggesting that both TTM and OCD involve activity in similar brain regions, mainly the frontal regions (Mataix-Cols & van den Heuvel, 2006; Stein et al., 2002).

Bienvenu et al. (2000) found that cases of TTM were significantly higher in case probands than controls. TTM was shown to be transmitted in families of patients with OCD, and the disorder was the most prevalent in patients whose family members exhibited OCD. The researchers provided a lifetime prevalence of OC spectrum disorders for patients with OCD; TTM (4%) exhibited one of the highest lifetime prevalences (Hollander, 1993).

The Treatment Domain

The leading psychosocial and psychopharmacological interventions for TTM are habit reversal therapy (HR), SSRIs, and clomipramine (Bloch et al., 2007). Bloch and colleagues (2007) conducted a meta-analysis assessing the individual effectiveness of HR for TTM participants as compared to controls. The researchers found a significant difference between the HR and waitlist/control group. These results are consistent with current literature concerning psychosocial treatment for TTM (Hollander et al., 2008). In another study, 22 participants with TTM were involved in an open trial of individual CBT with an emphasis on relapse prevention. Treatment was administered in two phases. The first phase consisted of active treatment in which individuals were provided with psychoeducation, strategies of

competing responses training, and cognitive restructuring. This phase included CBT once a week for 8 weeks. The second phase consisted of four bi-weekly sessions focusing on relapse prevention. Results showed that 77% of participants were classified as “treatment responder” and 32% were classified as “excellent responders.” Results remained consistent at a 6-week follow up, with 66% of participants classified as “treatment responders,” and 32% considered “excellent responders” (Tolin, Franklin, Diefenbach, Anderson, & Meunier, 2007). The results revealed that TTM, like OCD, responds positively to cognitive and behavioral treatments.

In terms of psychopharmacological treatment, SSRIs appear to be an effective treatment for TTM. In a 12-week *open-label* trial of treatment with escitalopram, (SSRI), 20 women were administered 10-30 mg/d of the medication. Researchers found a 50% reduction from baseline in symptom severity (Gadde, Wagner II, Connor, & Foust, 2007). However, based on a meta-analysis, Bloch and colleagues (2007) suggest that there is little evidence for the efficacy of SSRIs for TTM when compared to controls. Additionally, these researchers examined the effectiveness of clomipramine (a medication often used in the treatment of OCD) when compared to placebo control groups. Results suggest that clomipramine is more effective when compared to the placebo control group. On whole, research has yielded mixed results regarding the similarities and differences of TTM and OCD in their response to SSRI treatments.

Tourette’s Disorder

The Symptom Domain

Currently, Tourette’s disorder (TD) is classified based on diagnosis in infancy, childhood, or adolescence. TD is marked by multiple motor and/or one or more vocal tics (American Psychiatric Association, 2000). The most familiar associated symptoms of TD are obsessions and compulsions. Obsessions are not present in all diagnosis of TD; however, when present, obsessions tend to be similar to OCD, and might include obsessions of symmetry or exactness. Compulsions are more persistent and, as in OCD, they are not pleasurable, but rather, are experienced as distressing and socially debilitating (American Psychological Association, 2000; Hollander et al., 2008). In addition to a shared symptomology, motor stereotypy, a defining feature of TD, is frequently also manifested in OCD (Korff, Stein, & Harvey, 2008).

The Neurological and Genetic Domain

The frontal-striatal cortex has been indicated in various disorders, including OCD and other perspective OCDs, such as TD (Mataix-Cols & van den Heuvel, 2006; Stein, 2000). The basal-ganglia, caudate nucleus, and thalamus have been implicated in OCD and TD (Bartz & Hollander,

2006; Mataix-Cols & van den Heuvel, 2006). Peterson and colleagues (2003) measured basal ganglia volumes using structural MRIs in 154 participants with TD, including individuals with comorbid OCD under the condition that TD preceded OCD, and 130 healthy controls. The researchers assessed regional specificity and abnormal asymmetries in the basal ganglia. Results showed reduced volume in the caudate nucleus of the basal ganglia in participants with TD when compared to controls. Furthermore, individuals with comorbid TD and OCD exhibited even smaller caudate nucleus volume than those with TD alone. Participants with comorbid TD and OCD also showed an increase in tic symptom severity, suggesting that a similar brain region may be active in both OCD and TD. Using structural MRIs, Peterson and colleagues (2001), examined prefrontal, parieto-occipital, and inferior occipital brain regions in 155 TD and 131 health control participants. Results were consistent with the previously mentioned study, showing decreased volume in the prefrontal region of the brain in participants with TD. Additionally, increased symptom severity was correlated with decreased volume in these regions.

One of the most pertinent comparisons of TD and OCD may be with respect to the neurochemistry of these disorders. Research often points to the effectiveness of SSRIs and the role of the serotonin neurotransmitter system in OCD, however, the dopamine neurotransmitter system offers insight into the relationship between TD and OCD, especially in regard to stereotypic behaviors (Stein, 2000). Korff, Stein, and Harvey (2008) examined stereotypic behaviors in deer mice to assess the role of the serotonin and dopamine neurotransmitter systems in OCD. The stereotypic behaviors were observed and baseline data was recorded. Deer mice were then administered high and low doses of fluoxetine (an SSRI). Dopamine D₂ receptor agonists were administered as well. Results showed a significant decrease in stereotypic behavior in deer mice that received both high and low doses of fluoxetine; the dose of the SSRI was positively correlated with the decrease in symptom severity. Additionally, states of spontaneous stereotypic behavior were lessened by dopamine D₂ receptor agonists. On whole, results indicate that both the serotonin and dopamine neurotransmitter system may have an effect on the stereotypic behavior expressed in OCD and often in TD. These results may also aid in psychopharmacological treatment of these disorders.

Tourette's disorder is genetically transmitted through families (American Psychiatric Association, 2000). Stein (2000) makes note that there is a strong genetic overlap between OCD and TD. Tics are seen more frequently in families of OCD probands, and OCD is more common in families with individuals with TD than in control subjects. Another study presents three case studies in which individuals with comorbid OCD and TD expressed a similar chromosome 18 breakpoint; the breakpoint localized to the same chromosomal band in each of the cases presented (Cuker, State, King, Davis, & Ward, 2003). While the sample

size (18) used in this study was small, the data provide a promising start, and further research with individuals with comorbid OCD and TD seems appropriate.

The Treatment Domain

While psychopharmacological treatments have been shown effective and are most often employed in the treatment of TD, many patients refuse or discontinue medication (Wilhelm et al., 2003). Researchers have examined the effectiveness of psychosocial therapeutic approach such as habit reversal therapy. A randomized controlled study assessed the effectiveness of habit reversal therapy versus supportive therapy. Participants with TD were randomly assigned to 14 sessions of either habit reversal therapy ($n = 16$) of supportive therapy ($n = 13$). Habit reversal contained awareness training, self-monitoring, relaxation, competing response training, and contingency management. All participants received weekly individual therapy sessions for the first 8 weeks, while the remaining sessions were administered bi-monthly. HR patients exhibited significantly lower tic severity scores than supportive therapy patients; tic symptoms in the supportive therapy group remained largely unchanged, while symptom severity was significantly reduced in the HR group. Furthermore, HR participants showed significantly less functional impairment at 10-month follow-up compared to the supportive therapy group (Wilhelm et al., 2003). In a replication of this study, Deckersbach and colleagues (2006) found results similar to the initial experiment, further evidencing the effectiveness of HR for individuals with TD. While HR is not explicitly employing all the components of CBT, it does implement behavioral strategies (e.g., self-monitoring) utilized in CBT and ERP. Thus, both TD and OCD respond well to treatments with a behavioral component.

The leading psychopharmacological treatment for TD is dopamine agonists, including haloperidol and pimozide; however, other dopamine antagonists, such as metoclopramide have proven efficacious in double-blind studies as well (Nicolson, Craven-Thuss, Smith, McKinlay, & Castellanos, 2005). More recently, researchers examined the effects of aripiprazole, a partial dopamine agonist. In a pilot study, 14 children and adolescents with TD were administered aripiprazole over the course of 8 weeks starting with a dose of 5 mg and increasing the dose by 5 mg every 1 to 2 weeks. Results showed that more than half of the participants experienced at least a 30% improvement in tic severity, providing a positive foundation for future work which might also include other age groups. As mentioned above (TD-The Neurobiological Domain), the stereotypic behaviors displayed in OCD appear to be related to the dopamine neurotransmitter system, and it does respond well to dopamine agonists, especially in the reduction of TD like behaviors.

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Discussion

The proposal of an Obsessive Compulsive Related Disorders category for the next edition of the DSM has significant implications for treatment and classification (Castle & Phillips, 2006). There exists a systematic approach in examining the proposed disorders in relation to OCD, mainly assessing similarities and differences in the (1) symptom domain, (2) the neurobiological domain, (3) and the treatment domain. While this approach elucidates similarities among OCD and the prospective OCRDs, conclusive research is lacking.

While ample research ties together the similarities between OCD, BDD, TTM, and TD, as outlined above, findings rely heavily on observable symptoms. A variety of disorders not included in the prospective OCRDs (e.g., impulse control disorder) display obsessions, compulsions, or both, and yet, are not under consideration for OCD classification (Hollander et al., 2008). Similarly, as noted above, two of the more popular contenders for the OCRDs, TMM and TD, rarely include obsessions in their clinical presentation. Additionally, demographic parameters, such as comorbidity and age of onset, may lack sufficient strength as a defining OCD criterion. While OCD shares these features with many of the perspective OCRDs, they are present with other anxiety disorders as well (Bartz & Hollander, 2006; Hollander, et al., 2008).

Neurobiological studies lend to the comprehensive review of OCD and related disorders. However, a variety of brain regions are affected in OCD, and while OCD shows brain region abnormalities similar to BDD, TTM, and TD, it shares abnormalities with existing anxiety disorders as well. For example, both OCD and social phobia express abnormalities in the limbic region of the brain (Mataix-Cols & van den Heuvel, 2006). Similarly, effective treatments for OCD such as CBT, ERP, and SSRIs, have also proven to be efficacious for other anxiety disorders (Bartz & Hollander, 2006).

The future classification OCRDs proposes a spectrum model. Yet, a more productive approach may be to consider symptomology, neurobiology, and treatment on a continuum as opposed to categories in which similarities and differences with OCD are dichotomously placed (Castle & Phillips, 2006). The current method of studying OCRDs is to examine the similarities and differences between OCD and another disorder, and place the results into a “fix” or “no fix” category. If a continuum of symptomology, neurobiology, and treatment responses were created prior to conducting further research studies, perhaps data on the OCRDs for the DSM-V would be more conclusive.

Conclusion

The classification of OCD has undergone considerable changes over the past editions of the DSM. In fact, classification remains a concern for future editions of the DSM. Researchers have offered evidence both in support of

and against a new classification of this disorder (Mataix-Cols et al., 2007). However, the majority of current research is heavily focused on the prospective OCRDs. In order to conclusively and confidently remove OCD from the anxiety disorders category, research comparing and contrasting OCD to both anxiety disorders and OCRDs is warranted.

Further research on the classification of OCD as a spectrum disorder is recommended, especially since the literature remains preliminary and inconclusive. Researchers have found similarities among OCD and the proposed OC spectrum disorders across various domains. While OCD and BDD appear to share the most putative symptoms, there still lacks conclusive evidence for the inclusion of this and other disorders such as, trichotillomania and Tourette’s disorder in the OC spectrum. To develop a spectrum of disorders based on the symptom domain alone appears to be an oversimplification which neglects other possibilities.

The existing research serves as a phenomenal starting point and needs to be built upon, especially in the areas of psychological and psychopharmacological treatment. For classification as a spectrum disorder, researchers need to replicate the studies that find biological, neurological, and treatment response similarities between OCD and the recommended OCD spectrum disorders. Currently, there are few research studies directly comparing OCD to an OCD. Rather, OCD and OCRDs are often indirectly connected through the comparison of separate research studies, and future work should employ samples consisting of both OCD and OCD patients. A consistent methodology would offer a more accurate comparison of OCD and OCRDs. Continued research is needed to explore the many aspects of OCD and how these features are expressed across other potential OCD spectrum disorders.

References

- American Psychiatric Association. (2000). *The diagnostic and statistical manual of mental disorders* (4th ed. text revision) Washington, DC: American Psychiatric Association.
- Bartz, J.A., & Hollander, E. (2006). Is obsessive-compulsive disorder an anxiety disorder? *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 30, 338-352.
- Bienvenu, O. J., Samuels, J. F., Riddle, M. A., Hoehn-Saric, R., Liang, K-Y., Cullen, B. A. M., et al. (2000). The relationship of obsessive-compulsive disorder to possible spectrum disorders: Results from a family study. *Biological Psychiatry*, 48, 287-293.
- Bloch, M. H., Landeros-Weisenberger, A., Dombrowski, P., Kelemendi, B., Wegner, R., Nudel, J., et al. (2007). Systematic review: Pharmacological and behavioral treatment for trichotillomania. *Biological Psychiatry*, 62, 839-846.
- Bohne, A., Savage, C. R., Deckersbach, T., Keuthen, N. J., & Wilhelm, S. (2008). Motor inhibition in trichotill-

- omania and obsessive-compulsive disorder. *Journal of Psychiatric Research*, 42, 141-150.
- Buhlmann, U., Reese, H. E., Renaud, S., & Wilhelm, S. (2008). Clinical considerations for the treatment of body dysmorphic disorder with cognitive-behavioral therapy. *Body Image*, 5, 39-49.
- Castle, D. J., & Phillips, K. A. (2006). Obsessive-compulsive spectrum of disorders: A defensible construct?. *Australian and New Zealand Journal of Psychiatry*, 40, 114-120.
- Cuker, A., State, M. W., King, R. A., Davis, N., & Ward, D. C. (2003). Candidate locus for Gilles de la Tourette syndrome/obsessive compulsive disorder/chronic tic disorder at 18q22. *American Journal of Medical Genetics*, 130A, 37-39.
- Deckersbach, T., Rauch, S., Buhlmann, U., & Wilhelm, S. (2006). Habit reversal versus supportive psychotherapy in Tourette's disorder: A randomized controlled trial and predictors of treatment responses. *Behaviour Research and Therapy*, 44, 1079-1090.
- Fineberg, N. A., Pampaloni, I., Pallanti, S., Ipser, J., & Stein, D. (2007). Sustained response versus relapse: The pharmacotherapeutic goal for obsessive-compulsive disorder. *International Clinical Psychopharmacology*, 22, 313-322.
- Gadde, K. M., Wagner II, H. R., Connor, K. M., & Foust, M. S. (2007). Escitalopram treatment of trichotillomania. *International Clinical Psychopharmacology*, 22, 39-42.
- Hollander, E. (1993). Obsessive compulsive spectrum disorders: An overview. *Psychiatric Annals*, 23, 355-358.
- Hollander, E., Braun, A., & Simeon, D. (2008). Should OCD leave the anxiety disorders in DSM-V? The case for obsessive compulsive-related disorders. *Depression and Anxiety*, 25, 317-329.
- Korff, S., Stein, D. J., & Harvey, B. H. (2008). Stereotypic behaviour in the deer mouse: Pharmacological validation and relevance for obsessive compulsive disorder. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, 32, 348-355.
- Mataix-Cols, D., Pertusa, A., & Leckman, J. F. (2007). Issues for DSM-V: How should obsessive-compulsive and related disorders be classified? *The American Journal of Psychiatry*, 164(9), 1313-1314.
- Mataix-Cols, D. & van den Heuvel, O. A. (2006). Common and distinct neural correlates of obsessive-compulsive and related disorders. *Psychiatric Clinics of North America*, 29, 391-410.
- McKay, D., Neziroglu, F., & Yaryura-Tobias, J. A. (1997). Comparison of clinical characteristics in obsessive-compulsive disorder and body dysmorphic disorder. *Journal of Anxiety Disorders*, 11, 447-454.
- Montgomery, S. A. (1993). Obsessive compulsive disorder is not an anxiety disorder. *International Clinical Psychopharmacology*, 8(S1), 57-62.
- Neziroglu, F. (2008). Body dysmorphic disorder: Causes, characteristics, and clinical treatments. *Body Image*, 5, 1-2.
- Neziroglu, F. & Khemlani-Patel, S. (2003). Therapeutic approaches to body dysmorphic disorder. *Brief Treatment and Crisis Intervention*, 3(3), 307-322.
- Neziroglu, F., McKay, D., Todara, J., & Yaryura-Tobias, J. (1996). Effect of cognitive behavior therapy on persons with body dysmorphic disorder and comorbid axis II diagnosis. *Behavior Therapy*, 27(1), 67-77.
- Nicolson, R., Craven-Thuss, B., Smith, J., McKinlay, B. D., & Castellanos, F. X. (2005). A randomized, double-blind, placebo-controlled trial of metoclopramide for the treatment of Tourette's disorder. *Journal of American Academy of Children and Adolescent Psychiatry*, 44(7), 640-646.
- Peterson, B. S., Staib, L., Scahill, L., Zhang, H., Anderson, C., Leckman, J. F., et al. (2001). Regional and ventricular volumes in Tourette's syndrome. *Archives of General Psychiatry*, 58 (5), 427-440.
- Peterson, B. S., Thomas, P., Kane, M. K., Scahill, L., Zhang, H., Bronen, R., et al. (2003). Basal ganglia volume in patients with Gilles de la Tourette syndrome. *Archives of General Psychiatry*, 60, 415-424.
- Phillips, K. A., Albertini, R. S., & Rasmussen, S. A. (2002). A randomized placebo-controlled trial of fluoxetine in body dysmorphic disorder. *Archives of General Psychiatry*, 59, 381-388.
- Phillips, K. A., Didie, E. R., Feusner, J., & Wilhelm, S. (2008). Body dysmorphic disorder: Treating an underrecognized disorder. *American Journal of Psychiatry*, 169(9), 1111-1118.
- Phillips, K.A., McElroy, S.L., Keck, P.E., Hudson, J.I., & Pope, H.G. (1994). A comparison of delusional and non-delusional body dysmorphic disorder in 100 cases. *Psychopharmacology Bulletin*, 30, 179-186.
- Phillips, K. A., Pinto, A., Menard, W., Eisen, J. L., Mancebo, M., & Rasmussen, S. A. (2007). Obsessive-compulsive disorder versus body dysmorphic disorder: A comparison study of two possibly related disorders. *Depression and Anxiety*, 24, 399-409.
- Rossi, L. (2006). Obsessive-compulsive disorder and related conditions. *Psychiatric Annals*, 36(7), 514-517.
- Stein, D. J. (2000). Neurobiology of the obsessive-compulsive spectrum disorders. *Biological Psychiatry*, 47, 296-304.
- Stein, D.J., Coetzer, R., Lee, M., Davids, B., & Bouwer, C. (1997). Magnetic resonance brain imaging with women with obsessive-compulsive disorder and trichotillomania. *Psychiatric Research*, 74 (3), 177-182.
- Stein, D.J., van Heerden, B., Hugo, C., van Kradenburg, J., Warwick, J., Zungu-Dirwayi, N et al. (2002). Functional brain imaging and pharmacotherapy in trichotillomania: Single photon emission computed tomography before and after treatment with the selective serotonin reuptake inhibitor citalopram. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 26 (5), 885-890.
- Tolin, D. F., Frankling, M. E., Diefenbach, G. J., Anderson, E., & Meunier, S. A. (2007). Pediatric trichotillomania:

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- Descriptive psychopathology and an open trial of cognitive behavioral therapy. *Cognitive Behaviour Therapy*, 36 (3), 129-144.
- Wilhelm, S., Deckersbach, T., Coffey, B. J., Bohné, A., Peterson, A. L. & Baer, L. (2003). Habit reversal versus supportive psychotherapy for tourette's disorder: A randomized controlled trial. *American Journal of Psychiatry*, 160, 1175-1177.
- Wilhelm, S., Steketee, G., Reilly-Harrington, N. A., Deckersbach, T., Buhlmann, U., & Baer, L. (2005). Effectiveness of cognitive therapy for obsessive-compulsive disorder: An open trial. *Journal of Cognitive Psychotherapy: An International Quarterly*, 19(5), 173-179.