Chapter 2

DIMENSIONAL AND SUBTYPE MODELS OF OCD

Steven Taylor

Although obsessive-compulsive disorder (OCD) is recognized in *DSM-IV* as a unitary syndrome (American Psychiatric Association [APA], 2000), clinical investigators have increasingly come to regard it as a heterogeneous condition (eg, Pato, Pato, & Pauls, 2002). Some regard OCD as being composed of sets of dimensions, with each dimension corresponding to a distinct set of mechanisms. A dimension may be defined by an aggregate of causal factors that incrementally influence the risk for a particular set of obsessive-compulsive (OC) symptoms (eg, contamination obsessions and washing compulsions).

A different approach to understanding OCD holds that there are discrete subtypes (categories or taxa) of the disorder. Subtypes are defined on the basis of being, in some way, more homogenous than OCD in general. A subgroup can be defined, for example, by whether or not OCD is associated with tic disorders. Tic-related OCD is a more homogenous collection of symptoms than OCD in general. By identifying homogenous phenotypes, researchers hope to identify discrete sets of mechanisms.

The purpose of this chapter is to consider the merits of dimensional and subtype approaches to understanding OCD, with particular attention to the most widely used or innovative approaches. We will consider the relative advantages and disadvantages of the various approaches, with the goal of identifying the most promising ways of conceptualizing and investigating OCD.

The dimension versus subtype distinction has important implications for theory and research (Strube, 1989). A categorical (subtype) variable implies a different set of causes than a continuous variable. Subtypes presumably arise from a small set of causal factors (eg, the presence versus absence of an agent damaging the brain circuits implicated in OCD). In comparison, dimensional variables are probably the result of a multitude of factors. For example, numerous, additive genetic factors, with each making a small but important contribution to the risk of OCD. Dimensional approaches are consistent with current thinking about the role of genes in psychiatric disorders; investigators are increasingly interested in identifying numerous genes that each make only tiny (eg, 1–2%) contributions to phenotypic variance (Plomin, Defries, Craig, & McGuffin, 2003). Thus, the assumption about whether OCD is dimensional

focuses research efforts differently than does the assumption that OCD is composed of subtypes.

Conceptually, typologies lead us to expect that disorders have an all-or-nothing state, with no intermediaries. That is, either the person has an OCD subtype or does not. Typologies imply that treatments should have a similar effect on the disorder; once the critical mechanism is addressed the disorder should rapidly remit. Change may be difficult to initiate with a class variable, but once initiated should be more complete and dramatic (Strube, 1989). In comparison, dimensional approaches assume both a continuum of disorder severity (ranging from absent to very severe) and a continuum of treatment effectiveness (ranging from weak to very strong interventions).

DIMENSIONAL APPROACHES

Factor Analytic Studies

Dimensional approaches to OCD arose from the observation that OC symptoms vary in severity, ranging from very mild (eg, the so-called "normal" obsessions and compulsions: Rachman & de Silva, 1978) to very severe. Scales measuring OC symptoms were developed to capture this range of severity. Factor analyses of these scales suggest that OC symptoms can be decomposed into a small number of dimensions (eg, Baer, 1994; Goodman et al., 1989; Leckman et al., 1997; Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999; Summerfeldt, Richter, Antony, & Swinson, 1999; Taylor, 1995). Factor solutions have varied to some extent from study to study, depending on the nature of the sample, the scale used to assess OC symptoms, and the factor analytic techniques. Even so, a number of consistencies have emerged, suggesting that OC symptoms can be partitioned into what may eventually emerge as a set of reliable (replicable) dimensions. Currently, one of the best supported factor solutions is that reported by Leckman et al. (1997), which had been replicated in the author's original samples and by Summerfeldt et al. (1999). This solution, which is similar to many other factor analytic solutions, consisted of four dimensions:

- Obsessions (aggressive, sexual, religious, or somatic) and checking compulsions.
- Symmetry obsessions and ordering, counting, and repeating compulsions.
- Contamination obsessions and cleaning compulsions.
- Hoarding obsessions and collecting compulsions.

Factor analytic studies have typically not assessed cognitive compulsions in much detail, so the factor solutions may change to some extent when a broader range of OC symptoms is assessed.

The dimensions identified in factor analytic studies tend to be naturally correlated with one another, although the correlations are typically not large ($r < .50$). Even so, the correlations suggest that many of these factors probably load on a higher-order factor. The assumption underlying factor analysis is that each factor corresponds to a distinct set of mechanisms (Cattell, 1978). The finding that dimensions are often correlated suggests that OCD may arise from a combination of general factors (ie,

those influencing OCD in general, and possibly other disorders), and specific factors (corresponding to a given set of symptoms).

Evaluating Dimensional Models

Factor analytic studies do not *prove* that OCD is dimensional. Factor analysis creates dimensions, just like cluster analysis creates categories. Taxometric statistical procedures (Waller & Meehl, 1998) can be used to determine whether a variable is dimensional or categorical, however, these procedures have yet to be applied to OCD. Accordingly, the question of dimensions versus categories must be addressed by considering the relative strengths and limitation of these approaches.

Dimensional models, such as those identified by factor analysis of OC symptom scales, have the advantage of being consistent with the fact that OC symptoms vary in severity. Longitudinal studies suggest that OC symptoms tend to be stable in adults but not in children (Mataix-Cols et al., 2002; Rettew, Swedo, Leonard, Lenane, & Rapoport, 1992). In adults, changes in symptoms tend to occur within rather than between symptom dimensions; shifts from one dimension to another are rare (Mataix-Cols et al., 2002). In other words, if OCD symptoms change in adults, the changes tend to consist of movement up or down the symptom dimensions. Rettew et al. (1992) similarly suggested that in children, the observed changes had actually occurred within rather than between symptom categories, although their design did not allow them to test this. In summary, the available research is consistent with the idea that the dimensions of OC symptoms tend to be stable over time. Changes tend to be within dimensions, which is what one would expect if discrete sets of mechanisms were being modified over time (eg, with treatment).

The merits or usefulness of dimensional models can be further gauged by whether they have meaningful correlates, such as correlations with other symptoms, biometric variables associated with OCD, or treatment response. A number of such findings have emerged. For example, the extent to which OCD runs in families also varies across the symptom dimensions; aggression, sexual, and symmetry OC symptoms have a familial component, whereas hoarding and contamination symptoms do not (Alsobrook, Leckman, Goodman, Rasmussen, & Pauls, 1999). Scores on a dimension assessing counting and repeating compulsions, but not other OC dimensions, tend to be associated with an insertion/deletion polymorphism in the promoter region of the serotonin transporter gene (5-HTTLPR; Cavallini et al., 2002). Scores on the hoarding dimension are correlated with poor response to selective serotonin reuptake inhibitors and to behavior therapy (Abramowitz, Franklin, Schwartz, & Furr, 2003; Alonso et al., 2001; Black, Monahan, Gable, Blum, Clancy, & Baker, 1998; Mataix-Cols et al., 1999).

Comment

To summarize, dimensional models, in which OC symptoms are regarded as arising from a small number of dimensions, shows promise for understanding OCD. Future research, using taxometric methods (Waller & Meehl, 1998), is needed to investigate whether the dimensions are truly continua, or whether they are better conceptualized as categories. Additional research, using expanded assessments of OC symptoms, is also needed to firmly establish the best-fitting dimensional model.

SUBTYPING APPROACHES

General Approaches to Subtyping

Obsessive-compulsive disorder subtyping research, like *DSM-IV*, is couched in the idea that psychiatric disorders can be usefully partitioned into categories. The categorical approach works best "when all members of a diagnostic class are homogeneous, when there are clear boundaries between classes, and when the different classes are mutually exclusive" (APA, 2000, p. xxxi).

As with the *DSM-IV* approach to defining psychiatric disorders, OCD subtyping efforts have been based, to a greater or lesser extent, on the framework laid out in the classic paper by Robins and Guze (1970). These authors proposed that advances in understanding and treating psychiatric disorders are most likely to occur if we study homogeneous groups.

> "Homogeneous diagnostic grouping provides the soundest base for studies of etiology, pathogenesis, and treatment. The roles of heredity, family interactions, intelligence, education, and sociological factors are most simply, directly, and reliably studied when the group studied is as homogeneous as possible." (p. 984).

To identify and validate such groups, Robins and Guze outlined five phases, which interact with one another so that new findings in any one of the phases may lead to modifications in one or more of the other phases. The entire process is therefore one of continuing self-rectification and increasing refinement leading to more homogeneous diagnostic grouping. The five phases are as follows.

1. *Clinical description*. The clinical description of a proposed diagnostic syndrome (or subtype) may be based on some striking clinical feature, or on a combination of features that are thought to be associated with one another. The clinical description need not simply be based on signs and symptoms; it can include demographic features (eg, age, sex, and ethnicity), age of onset, precipitating factors, and any other descriptive features that can define the clinical picture most precisely.
2. *Laboratory studies*. These include chemical, physiological, radiological (eg, neuroimaging), and anatomical (biopsy and autopsy) findings. Psychological studies (eg, tests of cognitive processing) may also be included. When laboratory tests are consistent with the defined clinical picture, they permit a more refined classification.
3. *Exclusion of other disorders.* Exclusionary criteria (including criteria for discriminating subtypes) are developed on the basis of clinical descriptions and laboratory findings. The criteria should permit exclusion of borderline or doubtful cases so that the index group may be as homogeneous as possible.
4. *Follow-up studies.* These studies can be used to determine whether the diagnostic category or subtype is stable over time. Do patients with one putative OC subtype, for example, tend to switch to another subtype over time? Follow-up studies can also investigate whether members from a putative homogeneous group differ in their course of disorder or treatment response. A group may not be a homogenous disorder if it can be clearly divided into patients with good versus poor prognosis.

> "Marked differences in outcome, such as between complete recovery and chronic illness, suggest that the group is not homogeneous. . . . The same illness may have a variable prognosis, but until we know more about the fundamental nature of the common psychiatric illnesses marked differences in outcome should be regarded as a challenge to the validity of the original diagnosis." (Robins & Guze, 1970, p. 984).

5. *Family studies.* The validity of a proposed type or subtype of psychiatric disorder would be supported by showing that it runs in families, reflecting the effects of genetic or shared environmental factors.

OCD Subtype Models

Researchers interested in identifying OCD types have used some or sometimes many of these five phases. Some studies have focused primarily on clinical descriptions for identifying subtypes, while others have focused mainly on family studies or laboratory tests. Still others have attempted to examine all five phases to validate OC subtypes. As a result of these efforts, various subtyping schemes have been proposed, as reviewed in the following sections.

Subtyping by Clustering OC Symptoms

Cluster analyses of OC symptoms have yielded various cluster schemes, depending, in part, on range of symptoms assessed (eg, Abramowitz et al., 2003; Calamari et al., 1999; Khanna et al., 1990). Abramowitz et al. clustered the broadest sampling of OC symptoms, obtaining a five-cluster solution: Harming, contamination, hoarding, unacceptable thoughts, and symmetry. Poorest response to behavior therapy was found for among patients with hoarding symptoms, compared to other patients with other OC symptoms.

There are two major problems with such symptom-based subtyping schemes. First, subtypes (categories) defined by OC symptoms are unable to account for the fact that, phenotypically, OC symptoms vary along a continuum of severity. A subtype model leads one to expect that a person either falls into a subtype category or does not. The range of symptom severity, including so-called "normal" obsessions and compulsions is inconsistent with this notion. A further problem is that discrete, non-overlapping subtypes of OC symptoms are the exception rather than the rule. This problem was recently noted by Mataix-Cols et al. (2002).

> "The efforts based on categorical classification of patients with different OCD symptom subtypes (ie, washers, checkers, etc) have been relatively fruitless, in part because there are so few monosymptomatic patients; therefore, the recruitment of sufficient numbers of subjects with 'pure' OCD subtypes is impractical because such an approach excludes a majority of patients." (p. 263).

Calamari et al. (1999) acknowledged this problem in their cluster analysis: "The five subgroups were characterized by dominant symptom patterns and significant secondary concerns reflecting the symptom heterogeneity often seen in the clinical presentation of obsessional patients" (p. 113).

Autogenous Versus Reactive Obsessions

Lee and Kwon (2003) provided data and argument to distinguish between two types of obsessions—autogenous and reactive obsessions—which were said to be associated with distinct subtypes of OCD. People with autogenous obsessions are said to perceive the obsessions as ego-dystonic and irrational. The person attempts to expel or suppress the unwanted thoughts from consciousness, and frequently employs covert or superstitious compulsive behaviors (eg, counting, praying, undoing the thought with a more acceptable thought) to control the obsession. People with reactive obsessions, on the other hand, are said to believe the thoughts to be relatively rational and realistic, although they frequently or superficially describe their thoughts as being irrational and absurd to clinicians. People with reactive obsessions devote themselves to coping behaviors for preventing the unwanted possible consequences of the obsessions rather than from expelling the thoughts themselves. Thus, people with reactive obsessions resort to overt compulsive behaviors (eg, washing, checking, arranging, hoarding) for preventing the unwanted possible consequences of the obsessions. The compulsions are maintained by anxiety reduction and by the fact that compulsions block the opportunity for disconfirming the obsession.

Lee and Kwon's innovative subtyping scheme merits further investigation. One question worth investigating is whether the mechanisms underlying each type of obsession are categorical or dimensional. Given that obsessions vary along a range of continua (eg, intensity, duration, believability), a dimensional mechanism might be more appropriate. If so, then Lee and Kwon's formulation might provide a basis for understanding the various dimensions of OC symptoms, as identified in factor analytic studies.

Personality Traits

Researchers have attempted to subtype OCD according to personality traits, such as schizotypal personality features (Fals-Stewart & Lucente, 1993; Sobin et al., 2000). A problem with personality-based subtyping schemes is that they are not directly concerned with OCD; they are more appropriately viewed as subtype models of personality. Personality disorder traits co-occur with all kinds of Axis I disorders. Although personality pathology may be associated with poor treatment outcome for OCD (Fals-Stewart & Lucente, 1993), this tells us little about OCD per se; personality disorders predict poor outcome for OCD and other Axis I disorders (eg, panic disorder: Taylor, 2000).

Age at Onset, Tics, and Family History

Three features, early age at onset of OCD, history of tics, and family history of OCD or tics, have been used collectively or individually to define subtypes of OCD. There is suggestive evidence that OCD has a bimodal age of onset; most cases develop in adolescence or early adulthood, while a subgroup develop the disorder in childhood (Geller et al., 1998; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995). The latter are mostly males, while later-onset OCD is split evenly among genders, or contains more females (Geller et al., 2001a; Leonard et al., 1999).

Childhood-onset OCD is more likely to be comorbid with tic disorders, such as Tourette's disorder (Geller et al., 2001b; Leonard et al., 1992; Pauls et al., 1995). The

research generally suggests that aggressive, sexual, symmetry, and exactness obsessions are more common in OCD with comorbid tics (George, Trimble, & Ring, 1993; Holzer et al., 1994; Leckman, Grice, & Barr, 1995; Leonard et al., 1992, 1999; Miguel et al., 1997; Zohar et al., 1997). Tic-like compulsions (touching, blinking, rubbing, tapping, staring) are more common in OCD patients with comorbid tics (Holzer et al., 1994; Leckman et al., 1995; Miguel et al., 1997).

Childhood OCD, compared to OCD arising later in life, also differs in particular clinical features (eg, Albert, Maina, Ravizza, & Bogetto, 2002; Alsobrook et al., 1999; Geller et al., 2001a, 2001b; Hanna et al., 2002; Rosario-Campos et al., 2001). Although there are some inconsistencies in the literature, it appears that childhood OCD is more likely to be associated with poor insight and comorbid attention-deficit hyperactivity disorder (ADHD). Some differences in the expression of OC symptoms over the lifespan may reflect developmental influences rather than being an indication of subtypes. Insight, for example, may simply reflect the person's level of cognitive development.

There is also evidence that adults with early- versus late-onset OCD differ in patterns of regional cerebral blood flow in the frontal-subcortical regions implicated in OCD (Busatto et al., 2001). These results offer preliminary evidence that brain mechanisms in OCD may differ depending on the age at which the disorder first arises.

People with childhood-onset OCD, compared to those with later-onset OCD, are more likely to have first-degree relatives with OCD or tics (Nestadt et al., 2001, 2002; Pauls et al., 1995; Rosario-Campos et al., 2001). Recent evidence suggests that familial OCD has a lower threshold for precipitating events. That is, life events prior to the onset of OCD appear to be more common and more severe in non-familial OCD (Albert et al., 2002).

In summary, three features, early age at onset, comorbid tics, and family history of OCD or tics, tend to co-occur and may define a particular subtype of OCD. However, the co-occurrence of these features is far from perfect. A number of people with childhood-onset OCD, for example, do not have tics or tic-like compulsions, and do not have a family history of OCD (Pauls et al., 1995). Therefore, the three features do not, strictly speaking, define a clear-cut OC subtype. Bimodality of age at onset may suggest a subtype, although under some circumstances a continuous (dimensional) variable can give rise to a bimodal phenotype (Waller & Meehl, 1998). It is possible that age at onset, occurrence of tics, and familiarity are markers of psychobiological dimension that determines the risk for OCD. At the present time, however, early-onset, tic-related, familial OCD seems to be a good candidate for an OC subtype.

Future research will be facilitated if investigators can identify an empirically defined demarcation point for distinguishing "early-onset" from "late-onset" OCD. Previous research has been inconsistent in this regard. Some investigators define early-onset as less than 10 years old, and late-onset as greater than 17 years old (eg, Rosario-Campos et al., 2001). Others use difference criteria; for example, early-onset as less than 10 years old and late-onset as greater than 12 years old (Busatto et al., 2001). Taxometric methods may prove helpful in identifying the optimal cut-off.

Infectious Diseases

It has long been observed that OC symptoms can arise from brain-injuring agents, such as particular infectious diseases. von Economo (1931), for example, described patients who developed OC as a result of encephalitis (ie, a postencephalitic syndrome). More recently, attention has been directed to the possibility that some forms of OCD,

particularly early onset OCD, may be a result of certain diseases that most commonly strike during childhood.

Swedo et al. (1998) observed that Sydenham's chorea, a well-recognized manifestation of rheumatic fever, is commonly associated with OC symptoms. These investigators also observed that some cases of childhood OCD are rapidly acquired after the child develops a Group A β-hemolytic streptococcal infection (GABHS), which is associated with illnesses such as scarlet fever or streptococcal pharyngitis. Streptococcus-related OC symptoms are thought to be commonly associated with tics, separation anxiety, motoric hyperactivity, and neurological symptoms such as clumsiness and choreiform movements. Swedo et al. (1998) referred to this syndrome as pediatric autoimmune disorder associated with streptococcal infection (PANDAS). This syndrome is defined when all of the following are met (Swedo et al., 1998):

- Presence of OCD or a tic disorder.
- Symptom onset between age of 3 and puberty.
- Episodic course, with abrupt and substantial symptom exacerbations
- Symptom onset and exacerbations are associated temporally with GABHS infection.
- Presence of neurologic abnormalities during symptom exacerbations.

The major distinguishing feature of the PANDAS subgroup is the temporal association between neuropsychiatric symptom exacerbations and GABHS infection, that is, positive (or rising) antistreptococcal antibody titers or a positive throat culture during neuropsychiatric symptom relapses and evidence of GABHS negativity during periods of remission (Swedo, 2002). Note that PANDAS is conceptualized as a poststreptococcal disorder; exacerbations usually occur long after the acute symptoms of the streptococcal infection have gone, not at the initial point of infection.

Many children develop streptococcal infections, yet few develop OCD. Swedo (eg, 2002) proposed that susceptibility to PANDAS is probably due to a combination of genetic, developmental, and immunologic factors. Developmental vulnerabilities are suggested by the high rates of streptococcal infection among grade-school age children. For the PANDAS subgroup, the peak age at onset of OC symptoms is 6–7 years (Swedo et al., 1998). The role of genetic factors is suggested by a family study finding increased rates (compared to controls) of rheumatic fever among the parents and grandparents of PANDAS children (Swedo, 2002). Such children also have increased rates of OCD and tics among family members (Lougee, Perlmutter, Nicolson, Garvey, & Swedo, 2000). Swedo (2002) speculates that "the combination of increased familial rates of OCD/tic disorders and increased rates of rheumatic fever suggests that children in the PANDAS subgroup may have a dual genetic vulnerability—with inherited susceptibility to both OCD/tic disorders and post-streptococcal sequelae" (p. S25).

The proposed pathophysiology of streptococcus-induced OCD is similar to that of Sydenham's chorea. Susceptible people respond to the infection by producing antibodies in a normal fashion. However, these antibodies are thought to cross-react with neuronal tissue and compromise their function, leading to the observed clinical symptoms (ie, an abnormal immune response) (Swedo, 2001). Thus, streptococcal infection is thought to produce OC symptoms by a process of inflammation of the basal ganglia (an autoimmune process), which occludes blood supply to these regions and eventually causes tissue necrosis. This suggests that treatments that reduce the inflammation-inducing antibodies (eg, plasma exchange) would reduce OC

symptoms for children with acute-onset OC symptoms, but probably not for more chronic OCD.

A handful of experimental treatment studies have investigated the role of streptococcal infection in causing some types of OCD. Garvey et al. (1999) attempted to treat PANDAS with prophylactic oral penicillin. Treatment had no impact on OC symptoms or tics. However, it also had no impact on the prevalence of streptococcal infections, and so their study failed to adequately test the hypothesis of streptococcal-induced OC symptoms. Perlmutter et al. (1999) conducted a randomized, placebo-controlled study of two treatments: intravenous immunoglobulin and plasma exchange. Such interventions should reduce or eliminate streptococcal antibodies and thereby eliminate the autoimmune-related inflammation of the basal ganglia. Consistent with this, both therapies produced significant improvements in OC and related symptoms; at one-year follow-up, 14 out of 17 children (82%) were rated as "much" or "very much" improved. Further consistent with the notion of PANDAS, Nicolson et al. (2000) found that plasma exchange was ineffective in reducing OC symptoms in children who did not have evidence of streptococcal infection.

Giedd, Rapoport, Garvey, Perlmutter, & Swedo (2000) performed an MRI study of PANDAS subjects versus controls. The PANDAS group had significantly larger basal ganglia (ie, caudate, putamen, and globus pallidus, but not total brain volume), which is consistent with the presence of localized inflammation. A case study suggested that this was normalized with plasma exchange treatment, which presumably reduced inflammation in these regions (Giedd, Rapoport, Leonard, Richter, & Swedo, 1996).

Immunological findings (eg, assays of antistreptococcal antibodies and autoantibodies) in OCD have yielded mixed results, with some but not all studies supporting a connection between streptococcus and OCD (Murphy, Petitto, Voeller, & Goodman, 2001). Peterson et al. (2000), for example, produced results that were interpreted as evidence that prior reports of an association between antistreptococcal antibodies and either tics or OCD may have been confounded by the presence of ADHD. The authors found that antibody titers were correlated with ADHD but not with tics or OCD. However, streptococcal infections are thought to account for a minority (no more than 10%) of childhood OCD (Trifiletti & Packard, 1999). Therefore, findings such as Peterson et al.'s results are not surprising; only a small proportion of their OCD patients would be expected to have antistreptococcal antibodies.

PANDAS-like syndromes associated with GABHS have been identified in adults (Bodner, Morshed, & Peterson, 2001; Greenberg, Murphy, & Swedo, 1998), although PANDAS is considered a childhood-onset disorder because GABHS infections are more common in childhood (Swedo, 2002).

It is noteworthy that PANDAS resembles the early-onset, tic-related, familial subtype of OCD described in the previous section of this chapter. It is possible that they refer to the same or perhaps highly overlapping subtypes. The abrupt onset and offset of PANDAS is consistent with a categorical rather than dimensional model of OCD; one either has infection-related OCD or one does not. In fact, unlike typical OCD, which presents during the teen or early adult years and is characterized by a gradual onset over months, patients with PANDAS tend to be younger and experience an explosive onset of symptoms, that sometimes could be pinpointed to a particular day (Stephenson, 2002).

Although some clinical investigators have expressed doubts about the value of the concept of PANDAS (Kurlan, 1998), others see it as an important advance

in understanding OCD. March and Vitiello (2001), for example, concluded that "PANDAS will likely yield the first empirical demonstration of an etiopathogenically defined subtype of OCD and tic disorders." (p. 142).

Although PANDAS is a promising OC-related subtype, its importance should not be overestimated. It is thought that poststreptococcal autoimmunity might be responsible for up to 10% of cases of childhood OCD (Trifiletti & Packard, 1999). This leaves the other 90% unaccounted for. Other sorts of infectious diseases might account for another proportion of OCD, although many patients are seemingly in good physical health when the disorder arises.

Comment

There are two broad approaches to categorizing psychopathology. Following Robins and Guze (1970), some psychopathologists—called *splitters*—have sought to define smaller and smaller diagnostic categories. The concept of *neurosis*, for example, has been split into distinct disorders (eg, the *DSM-IV* anxiety disorders), and, in turn, these disorders have been split into smaller units (eg, the various sub-forms of specific phobia are listed in *DSM-IV*). Researchers proposing OCD subtypes have continued this tradition.

A contrasting approach is taken by *lumpers*, who argue for broad diagnostic categories. Tyrer (1985) is perhaps the best known advocate of this approach. Lumpers begin with the observation that disorders such as OCD are commonly comorbid with many other disorders, such as other anxiety disorders and mood disorders (APA, 2000). Comorbidity may be concurrent (disorders present at the same time) or lifetime (disorders may or may not co-occur at a given time). A common diathesis may account for much of the comorbidity among the disorders. Tyrer (1985) and others have argued that the frequent comorbidity among anxiety and mood disorders indicates the presence of a unitary, general neurotic syndrome.

> "Acceptance of the existence of a broad neurotic syndrome does not necessarily deny the existence of separate neurotic disorders. . . . However, such diagnoses can only be retained for those patients who have pure syndromes, maintain their diagnostic appearance, and who do not pass, chameleon-like, through different diagnostic hues depending on the nature of the stresses they encounter." (Tyrer, 1985, p. 687).

A challenge for proponents of OCD subtyping schemes is to demonstrate that splitting OCD into subtypes had advantages over other, broader classifications, such as "unsplit" OCD or the general neurotic syndrome. Researchers and clinicians would be more likely to adopt a given subtyping scheme if it can be shown to have clear advantages over other schemes. The work on PANDAS seems most promising in this regard. Preliminary work suggests that splitting OCD into PANDAS and non-PANDAS types may have important implications for treatment (eg, whether or not to use plasma exchange).

An important research direction is to compare the various subtyping schemes with one another to discern their relative merits. Some schemes may be compatible with one another. Research may eventually show, for example, that an infection-based scheme can be integrated with a scheme in which OCD is subtyped according to age at onset, presence of tics, and familiarity. Obsessive-compulsive disorder arising from various childhood diseases might largely correspond to early-onset, tic-related

OCD. If Swedo and colleagues are correct in assuming a genetic predisposition for developing infection-related OCD, then an early-onset, tic-related subtype would also tend to have a family history of OCD or tic disorders.

CONCLUSIONS AND FUTURE DIRECTIONS

The various dimensional and subtype approaches to understanding OC symptoms have largely developed in isolation of one another. The time is ripe for a new generation of studies to compare these models with one another. The models can be compared on the extent that they help us understand, predict, and treat OC symptoms. The available evidence, although limited in all sorts of ways, suggests that age at onset, tics, familiarity, and presence of particular infections (eg, streptococcal infections) are the most promising variables for subtyping OCD. The least promising approach, in my view, is to subtype OCD according to symptom clusters. This approach has two disadvantages compared to dimensional models. First, subtypes (categories) defined by OC symptoms are unable to account for the fact that, phenotypically, OC symptoms vary along a continuum of severity. A subtype model leads one to expect that a person either falls into a subtype category or does not. The range of symptom severity, including so-called "normal" obsessions and compulsions is inconsistent with this notion. A further problem is that discrete, non-overlapping subtypes of OC symptoms are the exception rather than the rule.

Dimensional models, particularly models consisting of correlated dimensions, are more consistent with the patterns of covariance of OC symptoms; people vary in their severity along each dimension, and since the dimensions are correlated, the person can have more than one type of OC symptom (eg, washing compulsions plus checking rituals).

It remains to be established whether taxometric methods support the dimensional and subtyping models. Some putative subtypes might turn out to be better regarded as dimensions. A subtyping scheme based on infectious diseases is most likely to be truly categorical; barring the possibility of subclinical syndromes, one either has an infection or does not have it. The rapid onset and offset of PANDAS-related symptoms is consistent with this conjecture.

The approaches considered in this chapter have consisted of either subtypes or dimensions. It is possible that other, more complex models may be needed to account for OC phenomena. Hybrid models combining dimensions and categories await investigation. Such models could posit that some forms of OCD are categorical (eg, a PANDAS subtype), while others are dimensional. With greater understanding of the brain circuits, genes, and environmental factors involved in OCD and related phenomena, classification will eventually move from phenotype-based classification (eg, based on symptoms, age at onset, etc) to one based on mechanisms.

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