CHAPTER 45

Obsessive–Compulsive Disorder

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WHAT IS OBSESSIVE–COMPULSIVE DISORDER (OCD)?

OCD is defined by recurrent obsessions and/or compulsions that significantly impair functioning (American Psychiatric Association, 1994). Obsessions involve intrusive thoughts, images, or impulses that cause significant distress. Common obsessions include preoccupation with contamination, concerns about harming oneself or others, intrusive sexual thoughts, fear of throwing possessions away, and preoccupation with things not being "just right." Compulsions can be either mental or physical behaviors that people have a difficult time resisting; functionally, over 90% of patients with OCD report that they perform compulsions to reduce the distress associated with obsessions (Foa et al., 1995). Common compulsions include washing, checking, reviewing, hoarding, reassurance seeking, and repeating acts until they feel as if they have been performed correctly. Although DSM-IV criteria do not require the presence of both obsessions and compulsions, only 2% of patients in the DSM-IV field study reported obsessions alone (Foa et al., 1995). Covert mental actions, such as repeating a phrase mentally, or replacing an anxiety-provoking thought or image with a neutralizing thought or image, are important to identify as they can be overlooked in favor of more overt compulsive behaviors. When patients do not recognize that their obsessions and compulsions are excessive or unreasonable, they are given the diagnosis of OCD "with poor insight." From the standpoint of cognitive behavioral therapy, it is crucial to identify the functional relationship between obsessive thoughts and ritualistic behaviors, and to note the presence of poor insight.

BASIC FACTS ABOUT OCD

OCD is a relatively common disorder, estimated to occur in approximately. 5–3% of the population (Andrews, Henderson, & Hall, 2001; Rasmussen & Eisen, 1992). OCD can be observed in childhood, and has been reported as young as age 2 (Rapoport, Swedo, & Leonard, 1992), although it more commonly begins in early adolescence and young adulthood (Rasmussen & Eisen, 1992). In adult samples, approximately 50% of patients with OCD are female (Rasmussen & Tsuang, 1986). Consistent with the finding that OCD has an earlier age of onset in males (Lochner et al., 2004), reports indicate OCD in twice as many males as females in some pediatric samples (Swedo, Rapoport, Leonard, Lenane, & Cheslow, 1989).

Most patients with OCD experience a chronic course with waning and waxing symptoms (Rasmussen & Eisen, 1992). OCD has been associated with significant comorbid psychiatric illnesses, especially depression and other anxiety disorders (Denys Tenny, van Megen, de Geus, & Westenberg, 2004, LaSalle et al., 2004). In fact, these studies indicate that between one-third and two-thirds of patients with OCD meet criteria for major depressive disorder. There also appear to be high rates of comorbidity between OCD and tic disorders (Eichstedt & Arnold, 2001), as well

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as potentially higher rates of comorbidity with other impulse-control disorders (cf, Foa & Franklin, 2001). In terms of impact on quality of life and functioning, OCD has been found to adversely affect employment, social functioning, physical functioning, and general quality of life (Quilty, Van Ameringen, Mancini, Oakman, & Farvolden, 2003; Bijl & Rivelli, 2000).

WHAT CAUSES OCD?

There is no single cause that currently explains why some people develop OCD. There are, however, a number of theoretical accounts regarding the development and maintenance of OCD. Dollard & Miller (1950) suggest that fear associated with obsessions begins via classical conditioning (e.g., the experience of fear is paired with a cue such as a dirty bathroom or an intrusive thought), and rituals are maintained through operant conditioning (e.g., avoiding dirty places or performing rituals reduces anxiety associated with the obsession). More cognitive theories focus on the false assumptions commonly found in patients with OCD, especially exaggerated personal responsibility (Salkovskis, 1985). Foa and Kozak (1985) proposed that patients with OCD overestimate threat because they fail to take the absence of danger as a signal of safety.

Although data indicates that there is some type of familial transmission of OCD (Nestadt et al., 2000), this also appears to be the case for most anxiety disorders (Nestadt et al., 2001). A number of genes believed to be involved in OCD have been identified; however, progress in this area seems to be hampered because of the clinical heterogeneity of OCD (Pato, Pato, & Pauls, 2002). For instance, recent studies have suggested that gender, neurobiological differences, comorbidity, and symptom types may all contribute to etiological heterogeneity in OCD. Specifically, one OCD phenotype appears to involve earlier onset, being male, a more chronic course, higher incidence of tic-related disorders, higher familial incidence of OC symptoms, and may be associated with distinct genetic susceptibility (Eichstedt & Arnold, 2001; Lochner et al., 2004).

A subset of pediatric patients develop OCD more following streptococcal infection, and these cases are referred to as Pediatric Autoimmune Neuropsychiatric Disorders Associated with Strep (PANDAS; Swedo et al., 1998). Hallmark features of PANDAS include a relatively sudden onset and symptom exacerbations and reductions that follow the course of step infections. Few data are available to date to establish the prevalence of PANDAS within patients with OCD, but our clinical experience suggests a small percentage of patients follow the hallmark features.

In terms of the neural substrates of OCD, recent neuroimaging studies have implicated hyperactivity in frontostriatal circuits, including the orbitofrontal cortex, anterior cingulate cortex, and structures of the basal ganglia (Saxena, Brody, Schwartz, & Baxter, 1998). Evidence suggests that symptom reduction following either psychotherapy or psychopharmacology is reliably related to reductions in activity in these areas of the brain. Functionally, the frontostriatal circuit has been found to be involved in action monitoring. Consistent with both OCD symptoms and OCD-related abnormalities of these areas, recent studies have reported hyperactive brain activity related to response monitoring in Obsessive–Compulsive subjects (Gehring, Himk, & Nisenson, 2000; Hajcak & Simons, 2002; Ursu, Stenger, Shear, Jones, & Carter, 2003). Although neuropsychological findings vary somewhat from study to study, patients with OCD may show deficits in some memory tasks and tasks that assess executive functions such as organization (cf, Kuelz, Hohagen, & Volderholzer, 2004). [Au1]

WHAT IS INVOLVED IN EFFECTIVE ASSESSMENT?

OCD symptom severity can be assessed with either clinical interviews or self-report measures. Although structured clinical interviews can be used to determine whether or not patients meet DSM-IV criteria for OCD, the semistructured Yale-Brown Obsessive–Compulsive Scale (Y-BOCS; Goodman et al., 1989a, 1989b) is considered the gold standard in OCD assessment. The Y-BOCS is a semistructured clinician-administered interview that involves both a symptom checklist that assesses the presence of 40 obsessions and 29 compulsions, and a measure of symptom severity. Severity of obsessions and compulsions are calculated separately, where each are rated for time occupied, interference, distress, resistance, and control. The Y-BOCS total score is the sum of both the obsession and compulsion severity scales. The total scores run from 0 to 40, with the average patient in most studies ranging between a 24 and 28, and a clinical cut-score of 14.

There are several self-report instruments including the Maudsley Obsessive–Compulsive Inventory (Hodgson & Rachman, 1977), the Padua Inventory—Washington State University Revision (Burns, Keortge, Formea, & Sternberger, 1996), and the Vancouver Obsessive–Compulsive Inventory (in press). One of the most recent, and easy to administer, is the Obsessive–Compulsive Inventory—Revised (OCI-R; Foa, Huppert, et al., 2002), an 18-item self-report measure that assesses the distress associated with obsessions and compulsions. In addition to the total score, separate subscale scores can be calculated for Washing, Checking, Ordering, Obsessing, Hoarding, and Neutralizing. Foa, Huppert, et al. (2002) report excellent psychometric properties for the OCI-R in clinical patients with a range of anxiety disorders and nonanxious controls; excellent psychometric properties have also been reported in a nonclinical sample (Hajcak, Huppert, Simons, & Foa, 2004).

WHAT SHOULD BE RULED OUT?

Because of the high rates of psychiatric comorbidity in patients with OCD, it can be difficult to differentiate OCD from other disorders with related symptoms. Specifically, obsessions should be differentiated from depressive rumination and pathological worry characteristic of MDD and generalized anxiety disorder (GAD), respectively. These related symptoms can often be differentiated in the following way: the content of worry is usually verbally based, typically involves real-world concerns (e.g., the health of older parents, money in difficult financial times, etc.) and is experienced as appropriate or ego-syntonic. Ruminations generally surround negativistic thoughts about the past and the self and/or world, and depressed patients rarely struggle to suppress ruminations. On the other hand, obsessions generally involve magical or unrealistic thinking and images that are experienced as ego-dystonic; furthermore, patients with OCD continually attempt to suppress obsessions (cf, Foa & Franklin, 2001).

WHAT TREATMENTS ARE EFFECTIVE?

Behavior therapy that involves both exposure and response prevention (EX/RP) are considered the first-line treatment for OCD by experts (Greist et al., 2003). EX/RP entails exposing patients to feared stimuli in a hierarchical fashion, and having patients completely refrain from ritualizing. Exposures can be in vivo (e.g., touching contaminated objects) and/or *imaginal* (e.g., thinking about

spreading contamination). Imaginal exposures should be utilized, in particular, to confront patients with their unrealistic feared catastrophes that cannot and/or should not be produced in reality (e.g., finding out that one has a brain tumor). Importantly, both imaginal and in vivo exposures must be long enough and repeated frequently enough to allow for anxiety habituation (cf, Foa & Franklin, 2001). During the course of EX/RP, patients learn that they do not need to ritualize to reduce their anxiety—that anxiety habituates on its own. Importantly, they learn that the feared disasters they anticipate do not materialize and therefore they do not need to protect themselves by ritualizing or avoiding feared situations.

A number of studies have shown EX/RP to be superior to a number of control treatments (Abramowitz, 1997). Furthermore, many studies have demonstrated that treatment gains following EX/RP are maintained over long periods of time—up to 5 years in one study (see Marks, 1997). Studies examining whether the beneficial effects of EX/RP generalize beyond therapy delivered by experts in research settings have found support for the general effectiveness of EX/RP in both nonresearch and private practice settings (Franklin, Abromawitz, Kozak, Levitt, & Foa, 2000; Warren & Thomas, 2001), as well as in ethnically diverse populations (Friedman et al., 2003). Importantly, EX/RP also appears generally effective for patients with comorbid depression (Abramowitz, Franklin, Street, Kozak, & Foa, 2000; Overbeek, Schruers, Vermetten, Griez, 2002) and personality disorders (Franklin, Harap, & Herbert, 2004), further suggesting the generalizability of EX/RP as an effective treatment.

Many studies have also found that psychopharmacological treatment with selective serotonin reuptake inhibitors (SSRIs) results in significantly greater OCD symptom reduction relative to placebo (for a review, see Dougherty, Rauch, & Jenike, 2002). However, when the medicine is discontinued, some studies have reported high rates of relapse (Koran et al., 2002), although longer-term treatment may reduce rates of relapse somewhat (Hollander et al., 2003). Relatively few studies have directly compared therapy with SSRI with EX/RP; however, the available evidence suggests that EX/RP is at least as effective if not superior to existing SSRIs (Dougherty et al., 2002). In a recent study, EX/RP was superior to clomipramine, and the combined treatment outcome was superior to medication only, but equivalent to EX/RP only (Foa et al, in press). Thus, although there is some evidence that the combination of SSRI and EX/RP produces slightly better outcome than EX/RP alone (Hohagen et al., 1998), their combination does not appear to have reliable synergistic effects (cf, Foa, Franklin, & Moser, 2002). Alternative treatments for intractable cases of OCD have also been evaluated. Notably, deep-brain stimulation (cf, Kopell, Greenberg, & Rezai, 2004) and the surgical removal of the cingulate have shown to improve some treatment-resistant cases (Dougherty et al., 2002).

WHAT PREDICTS TREATMENT OUTCOME?

Most patients that get adequate treatment with either EX/RP or pharmacotherapy will experience significant reductions in symptom severity. However, there are a number of factors that appear related to poor outcome following therapy. Some studies have found that specific patterns of pretreatment brain activity differentially predict positive treatment response to both pharmacotherapy and behavior therapy (cf, Hurley, Saxena, Rauch, Hoehn-Saric, & Taber, 2002). An early study suggested that OCD patients that have obsessions in the absence of overt rituals may fare

more poorly than patients with overt rituals (Rachman & Hodgson, 1980); however, this difference may have resulted from early failures to identify and target covert mental rituals (cf, Abramowitz, Franklin, Schwartz, & Furr 2003). Subsequent studies suggest that poor insight is associated with poorer outcome with EX/RP (Abramowitz et al., 2003). Additionally, a number of studies have reported that hoarding is related to poorer outcome following both cognitive-behavioral therapy (Abramowitz et al., 2000; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002), and pharmacotherapy (Mataix-Cols, Rauch, Manzo, Jenike, & Baer, 1999). Severe depression may also be associated with a somewhat attenuated treatment response (Abramowitiz et al., 2000; Overbeek, Schruers, Vermetten, & Griez, 2002).

WHAT DO WE STILL NEED TO KNOW?

OCD is a heterogeneous disorder, and further progress may depend on research that focuses on particular symptom subtypes (cf, Calamari et al., 2003). For instance, whether hoarding represents a distinct subtype of OCD or should be considered a separate but related disorder should be a topic of future studies (see Steketee & Frost, 2003). Similarly, the OCD with childhood-onset should be investigated with respect to OCD that begins later in life. Within the latter subtype, its relationship to tic-related disorders is another area that requires further study. Identifying distinct phenotypes is a necessary step toward better understanding OCD at the level of molecular mechanisms, including the involvement of both specific genes and neurotransmitters.

In addition to furthering our understanding of basic psychopathology and mechanisms underlying the subtypes of OCD, more research will be needed to guide practitioners in terms of how to treat treatment-resistant patients (i.e., those who do not respond to SSRIs or EX/RP), how to maintain treatment gains, and how to treat patients to full remission. Finally, how to best export the most effective treatments to the community and ensure that more patients are receiving adequate treatment also requires further examination.

WHAT ARE SOME HELPFUL RESOURCES?

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Author Queries:

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