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Multiple pathways to functional impairment in obsessive–compulsive disorder

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A B S T R A C T

Obsessive–compulsive disorder (OCD) is a chronic and debilitating condition that is relatively common in both children and adults, and it is associated with a wide range of functional impairments. Mental health researchers and practitioners have placed considerable attention on OCD over the past two decades, with the goal of advancing treatment and understanding its etiology. Until recently, it was unknown to what extent this disorder was associated with functional impairment. However, recent research shows that the condition has significant social and occupational liabilities. This article discusses etiology, common symptom presentations (including comorbid and ancillary symptoms), basic OCD subtypes, neuropsychological functioning, and the relation these have with functional disability in OCD. Recommendations for future research are also considered.

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1. Introduction

Significant progress has occurred over the past two decades in the understanding of the phenomenology and pathophysiology of obsessive–compulsive disorder (OCD), a chronic and debilitating anxiety disorder found with relative frequency in both children and adults. Obsessive–compulsive disorder is more common than previously believed, affecting between 1% and 3% of the population (Rasmussen & Eisen, 1994; Torres et al., 2006). Epidemiological studies suggest that OCD represents the fourth most common mental disorder, following phobias, substance abuse, and major depression (Karno, Goldberg, Sorenson, & Burnam, 1988). Prevalence rates appear to be equal across genders, with a slight tendency toward a higher prevalence for women evident in some studies (Antony, Downie, & Swinson, 1998). Cross-culturally, the disorder has similar prevalence rates and a consistent symptom presentation (Weissman, Bland, Canino, & Greenwald, 1994). The World Health Organization (2001) ranked OCD as one of the most debilitating mental health disorders and estimated that, in 2000, OCD was among the top 20 causes of illness-related disability for people aged 15 to 44 years. In the great majority of cases, OCD symptoms start in childhood (Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995); for instance, in clinical samples, the mean age of onset was 10 years (Swedo, Rapoport, Leonard, Lenane, & Cheslow, 1989), with earlier age of onset in males (i.e., prepuberty) than in females (i.e., adolescence) (Geller, 2006; Tukel et al., 2005). Given these findings for age of onset and associated disability, it is clear that OCD contributes to significant functional impairment.

The course of OCD is typically chronic, with an increase of symptoms often associated with stressful life events (Cromer, Schmidt, & Murphy, 2007). In most cases, the course is continuous, although in some cases it may be episodic or even deteriorating. Initial onset is often marked by a gradual increase of symptoms (American Psychiatric Association [APA], 2000; Antony et al., 1998). Further, many writers emphasize chronicity of OCD, noting that sufferers often undergo setbacks in maintaining treatment gains and require ongoing attention to managing their symptoms (i.e., Jenike, Baer, & Minichiello, 1998; McKay, Todaro, Neziroglu, & Yaryura-Tobias, 1996). Finally, whereas effective treatments have been developed, many sufferers either fail to respond, respond partially, or relapse shortly after treatment (Abramowitz et al., 2003; Franklin et al., 2000). Such chronicity has led to the widespread view that OCD is associated with significant functional impairment and reduced quality of life. The present paper offers an integrative view of the facets of OCD that lead to functional impairment, including etiological and comorbid contributions, as well as the basic subtypes and dimensions of the disorder.

2. Obsessive–compulsive disorder and functional impairment

Obsessive–compulsive disorder is characterized by obsessions and/or compulsions that lead to significant distress and cause severe impairment in psychosocial functioning (APA, 2000; Leonard, Goldberg, Rapoport, Cheslow, & Swedo, 1990). Obsessions are repetitive, intrusive, and distressing thoughts, ideas, images, or urges that often are experienced as meaningless, inappropriate, and irrelevant, and persist despite efforts to suppress, resist, or ignore them (APA, 2000). Common obsessions include thoughts about contamination (55%), inappropriate aggressive (50%) or sexual (32%) thoughts or images, concern over symmetry or exactness (36%), and somatic fears (34%) (Abramowitz, Franklin, Schwartz, & Furr, 2003; Rasmussen & Tsuang, 1986). Compulsions are repetitive, stereotyped behaviors and/or mental acts that are used to diminish the anxiety and distress associated with the obsessions (APA, 2000). The most common compulsions include checking (80%), cleaning or decontamination rituals (e.g., excessive washing, bathing, or grooming; 46%), and counting (21%) (Abramowitz et al., 2003; Masi et al., 2005; Rasmussen & Tsuang, 1986).

Often, obsessions and compulsions are linked by content. For instance, individuals who have recurrent thoughts about making mistakes may check their work incessantly, while those who experience contamination concerns may engage in elaborate hand washing and bathing routines. Examination of symptoms among OCD patients along with factor analyses of OCD assessment measures provide support for dimensions and “subtypes” of OCD in which obsessions and compulsions load together on the same symptom-based factors and clusters (e.g., Leckman, Grice, Boardman, & Zang, 1997; Summerfeldt, Richter, Antony, & Swinson, 1999). Although the DSM-IV criteria imply that it is possible for a person to experience compulsions without obsessions (or vice versa), the vast majority of OCD patients have both obsessions and compulsions (Foa & Kozak, 1995). Indeed, only 2.1% of patients with OCD report predominant obsessions, 1.7% report predominant compulsions, and more than 95% reported both obsessions and compulsions on the Yale–Brown Obsessive Compulsive Symptom Checklist (Foa & Kozak, 1995; Goodman et al., 1989).

2.1. Etiology

Several lines of genetic, neuroanatomical, neurochemical, and psychological research point to OCD as a complex neurobehavioral illness that likely has multiple etiological determinants. What follows is a brief review of each domain.

2.1.1. Genetics

Twin and family study findings suggest that genetic factors are involved in the transmission and expression of OCD (Hanna, Himle, Curtis, & Gillespie, 2005; Jonnal, Gardner, Prescott, & Kendler, 2000; Karno et al., 1988; Nestadt et al., 2000; Pauls et al., 1995). Twin studies report higher concordance rates for OCD in monozygotic (80%–87%) than dizygotic twins (Carey & Gottesman, 1981) and variable heritability rates ranging from 26% to 65% (Eley et al., 2003; Hudziak et al., 2004). Family studies reveal that, generally, risk of developing OCD is greater in first-degree relatives of individuals with OCD than among the general population (Nestadt et al., 2000; Pauls et al., 1995). For example, a meta-analysis of controlled family studies based on adults with OCD showed an aggregate risk of 8.3% in first-degree relatives of participants with OCD and 2% in first-degree relatives of control participants (Hettema, Neale, & Kendler, 2001). In other words, a person with OCD is about 4 times more likely to have another family member with OCD than is a person without OCD. Moreover, it appears that childhood onset OCD has a stronger genetic contribution than does later onset OCD, with studies showing that OCD is more common in relatives of those with child onset (11%–23%) compared with adult onset (8.3%) (do Rosario-Campos et al., 2005; Hanna et al., 2005; Nestadt et al., 2000; Pauls et al., 1995).

Although twin and family studies point to a genetic component of OCD, the specific genes and inheritance mechanisms involved are less clear. Theories of the genetic factors involved in the pathogenesis of the disorder have varied from a multifactorial conceptualization (Lipsman,
Neimat, & Lozano, 2007), to specific genes, such as the chromosome 9p24 (Willour et al., 2004). To date, the most consistent findings have centered on the sapap3 and serotonin transporter genes (reviewed in Abramowitz, Taylor, & McKay, 2009). Still other studies have considered candidate genes affecting serotonin, dopamine, and glutamate neuro-transmissions (Hemmings & Stein, 2006; for a review of genetic studies of OCD, see Arnold & Richter, 2007). Given this, it appears that there are genetic features of the disorder, but that no one set of genes has been isolated that explain the disorder. This may be due, in part, to the considerable heterogeneity of the disorder noted earlier. It is important to note that most of the studies reviewed above do not control for shared environment and common learning histories that might confer greater familial transmission of the disorder (Jang & Shikishima, 2009, for a methodological discussion of identifying unique variance associated with environmental and genetic factors).

2.1.2. Neuroanatomy

There is increasing evidence for involvement of neurobiological factors in the etiology of OCD. The precise neurobiological mechanisms underlying OCD symptoms remain poorly understood (Maia, Cooney, & Peterson, 2008); however, relatively recent neurobiological studies on OCD patients compared with controls show hyperactivity during rest in a cortico-basal-thalamo-cortical loop that includes the orbitofrontal cortex (OFC), the anterior cingulate cortex (ACC), and the head of the caudate nucleus (Maia et al., 2008; Maltby, Tolin, Worhunsky, O’Keefe, & Kiehl, 2005). As compared with those who do not have OCD, individuals with the disorder evidence comparatively more activity in the orbitofrontal cortex (Saxena, Bota, & Brody, 2001). Thus, frontal–striatal circuitry that is ‘wired’ for atypical conscious awareness may explain the repetitive nature of OCD-related thoughts and rituals. Further, a meta-analysis of OCD functional neuroimaging studies found consistent differences between patients with OCD and healthy controls — specifically, in the orbito–frontal gyrus (within the frontal lobe) and the head of the caudate nucleus (part of the striatum; Whiteside, Port, & Abramowitz, 2004). There is some evidence to suggest differential pathology dependent upon either age of onset or current age, as the thalamus is commonly implicated in children (Busatto et al., 2001; Friedlander & Desrocher, 2006).

2.1.3. Neurochemistry

The serotonergic and dopaminergic neurotransmitter systems are among several that have been associated with the pathogenesis of OCD. Support for the serotonin hypothesis (i.e., implication of the serotonergic system in OCD presentation) has been drawn mainly from clinical trials demonstrating the efficacy of selective serotonin reuptake inhibitors (SSRIs) in the treatment of both adult and pediatric OCD (March et al., 1998; Insel, 1990; Pediatric OCD Treatment Study [POTS], 2004; Rapoport, Leonard, Swedo, & Lenane, 1993).

The mechanism proposed is that individuals with OCD have excessive baseline activity of excitatory glutamatergic neurons in the orbitofrontal cortex; serotonin inhibits these neurons, resulting in an increased release of serotonin in the orbitofrontal cortex, thus leading to a decline in OCD symptoms (Husted, Shapira, & Goodman, 2005). Further support for the serotonin hypothesis was derived from studies investigating the function of specific serotonin receptors (Adams et al., 2005; Hollander et al., 1992; Zohar, Mueller, Insel, Zohar-Kadouch, & Murphy, 1987). For example, the administration of certain serotonin agonists (e.g., meta-chlorophenylpiperazine) led to an exacerbation of OCD symptoms (Stein, Van Heerden, Wessels et al., 1999).

A recent review of pharmacological treatment of OCD indicates that SSRIs are associated with a meaningful clinical response in approximately 40–60% of patients and generally produce only mild side effects (Math & Janardhan-Reddy, 2007). Patients whose symptoms do not reduce after an initial SSRI trial may show improvement on a second SSRI trial (Math & Janardhan-Reddy, 2007). Individuals with OCD who experience high levels of side effects from SSRIs or who are refractory to SSRI treatment may require additional pharmacotherapeutic or behavioral interventions (Simpsen et al., 2008). Tricyclic antidepressants (TCAs), such as clomipramine and imipramine, are effective in the treatment of OCD symptoms (Denys, 2006), with clomipramine having good efficacy relative to placebo across a number of studies (Geller et al., 2003; Math & Janardhan-Reddy, 2007); however, the frequency of adverse side effects is higher for TCAs than for SSRIs. Thus, SSRIs remain the first-line pharmacotherapeutic treatment for OCD symptoms.

Although not entirely consistent, studies of peripheral markers, pharmacologic challenge, and treatment response imply that dopamine excess may also be involved in the etiology of OCD (Creese & Iversen, 1974; Denys, Zohar, & Westenberg, 2004; Eilam, Golani, & Szechtman, 1989; van der Wei et al., 2004). For instance, Neale and Olmmanns (1988) found that, following levodopa treatment (i.e., which increases dopamine reserves) of tardive dyskinesia, patients exhibited OCD symptoms. Moreover, higher levels of dopamine transporter density were found in the left caudate and left putamen of patients with OCD versus control patients. Additionally, studies have supported antipsychotic augmentation in SSRI non-responders and incomplete responders (Bloch et al., 2006). For example, McDougle et al. (1994) found that the addition of haloperidol (an antipsychotic medication that blocks dopamine D2 receptors) led to augmentation of SSRI response, particularly in patients with comorbid tics. Other controlled adult studies (McDougle, Epperson, Pelton, Wasylink, & Price, 2000; Skapinakis, Papaiothodorou, & Mavreas, 2007) and several adolescent case series (e.g., Fitzgerald, Stewart, Tawile, & Rosenberg, 1999; Storch, Lehmkuhl, Geffken, Touchton, & Murphy, 2008; Thomsen, 2004) have supported atypical antipsychotic augmentation. In treatment refractory cases, benzodiazepines may also be used; however, individuals should be aware of the increased possibility of dependence, side effects, and interactions with other medications and substances (Bandelow, 2008; Denys, 2006).

There are also limitations of pharmacotherapy as a therapeutic strategy for OCD. First, as many as 40% to 50% of OCD patients treated with SSRIs fail to respond (Bandelow, 2008; Denys, 2006). In addition, several pharmacotherapy interventions have side effect profiles and several require extensive monitoring by medical professionals (e.g., TCAs and benzodiazepines). Further, SSRIs can require 6 to 8 weeks before reaching optimal efficacy. This information is particularly important for patients since side effects such as restlessness, jitteriness, an increase in anxiety symptoms, or insomnia which lasts through the first days or weeks of treatment may hamper treatment compliance (Murphy, Segarra, Storch, & Goodman, 2008). Finally, discontinuation of pharmacological treatment is clearly associated with symptomatic relapse, irrespective of treatment duration, and a substantial proportion of OCD patients require lifelong medication use (Math & Janardhan-Reddy, 2007). This is likely because patients have not learned how to directly cope with their obsessive thoughts and compulsive avoidance and rituals. While there are a number of efficacious pharmacologic strategies, most of these approaches involve potentially serious and difficult to tolerate adverse side effects (i.e., TCAs, typical neuroleptic augmentation) or provide relief while leaving sufferers with residual clinically-significant symptoms (i.e., SSRIs).

2.1.4. Neuropsychology

Neuropsychological studies of individuals with OCD show a consistent pattern of deficits in fronto-striatal executive functions, particularly in utilization of organization strategies, as well as some limited evidence for memory impairments (Greisberg & McKay, 2003). The pattern of deficits in executive skills is demonstrated in multiple studies using a variety of executive-functioning tasks that
show impairments in the ability to shift set, organize, plan, and quickly solve problems (Gambini, Abbruzzese, & Scarone, 1993; Mataix-Cols, Jungque, & Sanchez-Turet, 1999; Veale, Sahakian, Owen, & Marks, 1996).

Memory functions in individuals with OCD, however, are more variable. Some have suggested that, given OCD-related reports of extensive doubt for actions, or sense of incompleteness in ritual behavior (such as doubting for actions, as in checking the stove when turning it off), OCD is associated with memory dysfunction. While this is a plausible consideration, accumulated data on memory functioning, separate from other neuropsychological areas of assessment, do not suggest poorer memory than that for other anxiety disorders (Savage, 1998). In some instances, memory performance is even superior in individuals with OCD relative to controls, and that the distinguishing factor was memory confidence (i.e., Radomsky, Gilchrist, & Dussault, 2006). These memory findings produce a complex clinical picture whereby individuals with OCD likely have at least normal memory functioning but poorer trust in their memory for specific threat-relevant events. The marked deficits in executive functioning that occur in the frontal–striatal circuit loop as well as lack of trust in memory abilities may help to explain global cognitive processing impairment in OCD. This, in turn, can lead to issues with decision-making, thus impacting both social and occupational functioning.

However, an additional question regarding neuropsychological functioning in OCD involves the manner in which memories are organized. For all the neuropsychological investigations conducted for OCD, the greatest consistency in research findings has come from the interplay between organization strategy and memory encoding (Greisberg & McKay, 2003). Specifically, using a scoring system that ranks efficiency of organizational approach in completing the designs on the Rey Complex Figure Test (RCFT; Rey, 1941), individuals with OCD consistently score lower than other patient groups (Savage et al., 1999). Organizational strategy on the RCFT is critical for better performance, and thus highly salient when considering working memory functioning in individuals with OCD (Greisberg & McKay, 2003). Thus, while more information is clearly needed in this area, OCD-related memory impairments are likely related to executive planning and organization difficulties, rather than direct impairments to learning and memory systems.

2.1.5. Implications of neurobiological findings for functional impairment

Each of the above reviewed areas point to (a) complex systems of impairment at the genetic, neurochemical, neurobiological, and neuropsychological levels; and (b) challenges in remedying these impairments. To take one example, psychopharmacological approaches are designed to target a subset of neurotransmitters and their associated brain areas. However, the degree that each neurotransmitter must be targeted, and in what sequence, in order to properly alleviate symptoms, is complicated by the heterogeneity of symptom expression. Without a clear unifying underlying mechanism for the condition (Taylor, McKay, & Abramowitz, 2005), the search for a more uniformly effective neurobiological treatment strategy will remain limited.

2.2. Psychological models

2.2.1. Learning principles

Development of OCD appears to be influenced by learning principles. Mowrer’s (1960) two-factor learning model has been used as an explanation for the acquisition and maintenance of OCD symptoms. According to this model, OCD 1) develops through classical conditioning, such that a previously neutral stimulus, through pairing with an aversive stimulus, comes to elicit a conditioned fear response; and 2) is maintained through operant conditioning, as the initiation of compulsions or rituals serves to neutralize obsessions and the subsequent reduction in anxiety serves as a negative reinforcer that maintains the compulsive behaviors. The operant conditioning component of this model has found strong empirical support (Rachman & Hodgson, 1980). Involvement of classical conditioning, on the other hand, remains questionable: namely, (a) individuals with OCD do not generally report a specific traumatic experience linked with the development of their fears; (b) the onset of the disorder tends to be rather pervasive and gradual (Abramowitz, Taylor, & McKay, 2007); and (c) individuals’ symptoms often change over time, a pattern that cannot widely be explained by new traumatic events or generalization of the fear from a particular initial traumatic event. Whereas the underlying classical conditioning basis of this model may not adequately explain symptoms of the disorder (Taylor, Abramowitz, & McKay, 2007), it has served as the basis for exposure with response prevention, which remains the dominant approach to treatment (Abramowitz, Taylor, & McKay, 2005).

Moreover, recent research indicates that symptoms of OCD may be motivated by factors other than fear, such as feelings of incompleteness or “not just right experiences” (NJREs) (e.g., Leckman et al., 1997; Coles, Heimberg, Frost, & Steketee, 2005), which are frequently reported among clients with OCD. In fact, it has been reported that as symptom severity increases, so too do the presence and severity of NJREs. Not just right experiences are phenomenological in nature, whereby the sufferer complains that something is not right, or that actions must be repeated until they feel ‘right.’ In some individuals with OCD, these symptoms can be severely disabling. For example, one of the authors (DM) worked with a client for whom the NJREs were sufficiently severe that she required up to an extra three hours per day in repeating and completing actions until they ‘felt right.’ These symptoms generally disappeared when another person was present, making exposure exercises difficult to develop and execute since these procedures could not be completed in the presence of a therapist.

The experimental literature suggests that NJREs are common among individuals without OCD as well, and that these sensations can be provoked in the lab (Coles et al., 2005). When induced experimentally, such experiences can cause distress and repeated urges to correct the phenomena. Correspondingly, given the highly aversive nature of NJREs, individuals generally feel a strong urge to complete the action until it feels ‘right,’ even if it means neglecting other pressing activities.

2.2.2. Cognitive model

The cognitive model builds on the premise that OCD is the result of a set of reactions that stem from, and intensify, otherwise normal intrusive thoughts (Rachman, 1997; Salkovskis, 1985, 1989). The theory proposes that mental intrusions are normal experiences that individuals have periodically and that obsessions experienced by people with OCD are not qualitatively different from the intrusive thoughts that occur in people who do not suffer from the disorder. However, unlike those without the disorder, people with OCD appraise such thoughts as highly significant and threatening and view themselves as personally responsible for whatever harm may result. Common dysfunctional beliefs or thinking errors include blame or inflated responsibility for outcome (i.e., the belief that one has special powers to cause, and/or the duty to prevent, negative outcomes), thought–action fusion (i.e., the belief that thoughts can influence events in the world), need to control thoughts (i.e., the belief that complete control over one’s own thoughts is necessary and possible), overestimation of threat (i.e., the belief that negative and catastrophic events are extremely likely), and intolerance for uncertainty (i.e., the belief that it is possible to completely prevent negative outcomes from occurring) (Obsessive Compulsive Cognitions Working Group [OCCWG], 1997).

Whereas the cognitive model has grown to dominate the contemporary conceptualization of OCD, there are several important caveats to its adequacy in describing the disorder. First, recent research suggests that the emphasis on inflated responsibility may not describe significant numbers of OCD sufferers. For example, using data from the OCCWG
scale development trial for the development of a major scale for assessing cognitive distortions associated with OCD, approximately half of those with OCD did not endorse theoretically associated distortions (Taylor et al., 2006). Further research with the same dataset showed that while there is one cognitive subtype associated with responsibility, there are separate subtypes of sufferers primarily characterized by perfectionism and by excessive concerns regarding the importance of thoughts (Haslam, Williams, Kyrios, McKay, & Taylor, 2005).

Finally, there also exists substantial numbers of sufferers whose symptoms are characterized by overvalued ideation (Kozak & Foa, 1994). Overvalued ideas in this context involve an experience whereby the individual with OCD feels not only compelled to complete the compulsions that arise from the obsession, but also frequently views the obsession as reasonable. While not necessarily delusional, this problem creates difficulties in conceptualizing the disorder as based on primary cognitive errors that can be challenged, and may serve as a barrier to behavioral interventions such as exposure with response prevention. Overvalued ideas are a poor prognostic indicator in the treatment of OCD. Central to contemporary treatment is that individuals have frank recognition that the link between obsessions and compulsions is illusory (McKay & McKiernan, 2005), a process that breaks down when overvalued ideas are present. The presence of overvalued ideas represents a significant barrier to treatment, since the client does not readily recognize the illusory link between obsessions and compulsions. A fundamental feature of CBT for OCD involves exposure with response prevention (ERP) (Abramowitz et al., 2005). However, individuals with overvalued ideas express great reluctance to engage these exercises, or refuse altogether. As yet, there are no accepted modifications to treatment to address this problem in OCD, although motivational interviewing (Miller & Rollnick, 2002) has been suggested a potentially promising approach (Merlo et al., in press; Tolin & Maltby, 2007).

Whereas overvalued ideas involve a pathologically extreme belief in the realistic aspect of thoughts, and the necessity of corresponding compulsions, scrupulosity is rooted more closely in cultural or religious beliefs that are held in an extreme manner. This connection is most apparent in individuals who present for treatment due to religious or cultural practice obsessions and compulsions. There is a wide range of ways that these concerns may be manifested including precision of religious practices (i.e., perfect ritualized prayer), excessive concerns over blasphemous thoughts, doubts that a sin has been committed, or concerns over offending tribal elders even when no opportunity for such offenses have occurred. Symptoms of scrupulosity are particularly troubling and the barrier to treatment is much the same as for overvalued ideas, in that most clients express extreme reluctance in practicing exercises that may violate religious or cultural beliefs as the beliefs are viewed as rational. That is, scrupulosity may be viewed as an amplification of an existing religious or cultural belief, and not necessarily excessive. For example, hand washing is prominent in many religious practices. If someone has contamination fears related to religious practices, such as concerns over contact with non-kosher meats, the associated hand washing could become a problem of scrupulosity. This would stand in contrast to the contamination fear sufferer who washes after contact with doorknobs not out of religious concerns but because of fears that others left behind germs. Abramowitz (2008) details modifications to CBT to engage individuals with scrupulosity in practicing ERP-based treatment, which is heavily reliant on a combination of motivational interviewing and illustrations of ways the rules that have developed around the OCD symptoms have been violated routinely by the client. Problems associated with scrupulosity represent another important barrier to treatment implementation and outcome (McKay, Taylor, & Abramowitz, 2010) and in turn, a significant contributor to difficulties in everyday functioning.

Subsequent to dysfunctional thinking, people with OCD experience an increase in distress and negative affect that leads to attempts to control or suppress the disturbing thoughts and efforts to neutralize any possible harmful consequences. Thus, compulsions and avoidance behaviors are maintained because they: (a) are followed by a short-term reduction in anxiety (negative reinforcement); (b) prevent a natural decrease in anxiety; (c) are reminders of obsessional thoughts thus further triggering such cognitions; and (d) are further reinforced by being attributed the ability to prevent feared consequences from occurring. In short, compulsions momentarily reduce the distress and discomfort associated with the misinterpretation of normal mental intrusions, but paradoxically increase the subsequent occurrence of intrusions and further strengthen negative interpretations (Rachman, 1997; Salkovskis, 1999). The intrusions escalate into distressing and persistent clinical obsessions maintained by compulsive rituals that prevent the rectification of mistaken appraisal (Abramowitz et al., 2007).

Different aspects of cognitive-behavioral theory for OCD have been empirically investigated. For instance, studies found that intrusive thoughts are indistinguishable in terms of form and content from clinical obsessions and occur in about 80% of the general population, but patients with OCD experience more intense, longer and more frequent mental intrusions than patients without OCD and they are less able to ignore or disregard them (Rachman & de Silva, 1978; Salkovskis & Harrison, 1984). Several studies have demonstrated that catastrophic appraisal of intrusive thoughts and perceptions of personal responsibility were correlated with measures of OCD symptom severity and that patients with OCD reported higher responsibility for outcomes related to their thoughts than normal controls (e.g., Clark & Purdon, 1993; OCCWG, 2003; Småri & Hölmstein, 2001). Moreover, experimental studies of the effects of responsibility appraisal showed that experimentally induced thought-action fusion beliefs in naïve participants led to an exacerbation of distressing thoughts and ritualistic behavior similar to OCD symptoms (Rassin, Merckelbach, Muris, & Spaan, 1999). On the same lines, other experimental studies demonstrated that efforts to suppress unwanted thoughts led to an increase in the frequency of such thoughts (Abramowitz, Tolin, & Street, 2001) and that repetitive checking led to an increase in doubt and uncertainty (van den Hout & Kindt, 2003). These studies provide support for the hypotheses that OCD is maintained by rituals and that attempts to suppress obsessive thoughts further trigger these cognitions and subsequent associated compulsions.

2.3. Subtypes of OCD

Although the DSM-IV-TR specifies symptom parameters (i.e., obsessions, compulsions) there is less specificity regarding the content of symptoms that are associated with OCD. Recently, there has been considerable focus on distinctions among the heterogeneous symptom presentation that characterizes OCD. OCD is a symptomatically diverse condition, in which there are a wide range of differing types of obsessions and compulsions, with varying degrees of functional impairment and responsiveness to treatment. For these reasons, many researchers now agree that particular kinds of obsessions and compulsions tend to co-occur to form five main empirically-derived subtypes of OCD: (1) Contamination (contamination obsessions with cleaning/washing compulsions); (2) Harming (doubting obsessions with checking compulsions); (3) Symmetry/ordering (symmetry obsessions with ordering, arranging, and counting compulsions); (4) Pure Obsessions (often of a sexual, religious, aggressive or somatic nature, without overt compulsions); and (5) Hoarding (hoarding obsessions and collecting compulsions). Among these, individuals with either cleaning or contamination rituals are among the most common within OCD patient samples, accounting for 75% of OCD patients (Ball, Baer, & Otto, 1996).

2.3.1. Contamination

The contamination subtype of OCD is characterized by overwhelming fear and intrusive thoughts of either becoming contaminated or accidentally spreading contamination in some way. There is evidence from the cognitive literature that individuals who suffer
from the Contamination subtype may possess excessive memory capabilities for threat-associated cues and enhanced attention toward contamination-associated stimuli (Foa, Ilii, McCarthy, Shoyer, & Murdock, 1993; Tata, Leibowitz, Prunya, Cameron, & Pickering, 1996). The object of contamination can vary, and involves common sources (e.g., germs, poisons, etc.) or unlikely sources (e.g., feeling ‘dirty’ in response to particular thoughts). Fears of contamination are often rooted in implausible beliefs and generalize over time, sometimes leading benign substances to be viewed as being contaminated due to similarity in appearance to actual contagions (e.g., chocolate fudge in the shape of dog feces), or to magical beliefs that a contagion will last forever (e.g., refusal to touch an object belonging to a person with hepatitis) (Rozin, Millman, & Nemeroff, 1986). This connection to implausible beliefs has led to research suggesting that disgust reactions may underlay the anxiety connected with contamination concerns leading to avoidance of potentially contaminating/contaminated stimuli (McKay & Moretz, 2009; Moretz & McKay, 2008; Olatunji & McKay, 2007). Problematic in treatment for many individuals with contamination fears is that disgust appears to habituate more slowly than anxiety when treatment involving exposure is applied (McKay, 2006). Given the dominant role of disgust in contamination fear, and comparatively limited research on this emotional state, problems associated with contamination fear may be more difficult to treat than previously considered.

Individuals with contamination obsessions will typically engage in excessive washing or cleaning to either reduce the feeling of contamination, or to avoid potential danger of contamination (McKay & Robbins, 2008). Such cleaning/washing compulsions are repetitive, ritualistic, time-consuming behaviors that the person feels compelled to perform to reduce distress caused by the obsessive thoughts of contamination, or mental ‘ dirtiness,’ disgust, and shame (Sookman, Abramowitz, Calamari, Wilhem, & McKay, 2005).

2.3.2. Harming

Typically, the harming subtype is characterized by distressing doubting obsessions with checking compulsions. While prevalence rate of these obsessions is not clearly described in the research literature, it is a commonly reported primary symptom, and constitutes a subtype based on prior cluster and factor analytic research (McKay et al., 2004).

Salkovskis describes maladaptive assumptions in OCD which are triggered by enduring beliefs related to the theme of responsibility. Such beliefs are rooted in critical events early in childhood and include a general understanding that one carries an excessive burden of responsibility for preventing threat. Further, by not taking direct action to prevent harm, one becomes essentially responsible for any harm that is produced (Salkovskis, 1985). This overwhelming sense of responsibility is viewed by Salkovskis as the core of all obsessional thinking in OCD, and specifically the increased sense of responsibility towards protecting from harm. Indeed, responsibility beliefs significantly predict harm obsessions but no other symptoms of OCD (Clark, 2004). Additionally, recent refinements to the inflated responsibility perspective include over-importance and control over thoughts (OCCWG, 2003) and the belief that individuals possess the power to cause, or prevent negative, even catastrophic events and considerable harm to others through a cognitive process referred to as ‘thought–action fusion’ (e.g., having the thought is like performing the action) (Shafarian, Thordardson, & Rachman, 1996). This inflated sense of responsibility leads to compulsive checking of those items for which the individual feels personally responsible for (e.g., sharp objects they own), as opposed to undifferentiated harmful objects (e.g., knives belonging to others). Further, individuals with harming obsessions become concerned about the content of their thoughts, assigning significance to thoughts that might be deemed unacceptable or potentially harmful to others. Individuals may spend hours each day physically checking and re-checking not only the security of objects in their environments (e.g., a locked door, an unplugged appliance), but may also engage in countless ‘mental’ checks to review whether their enacted checks were performed correctly, which may interfere with their ability to concentrate and focus on other tasks.

The harming subtype of OCD is perhaps the most heterogeneous (McKay et al., 2004) and has shown considerable symptom overlap in factor analytic studies with other symptom clusters of OCD. Typically, doubting obsessions will lead individuals to engage in checking compulsions to confirm a lack of harm. Due to symptom overlap with other obsessive symptoms (e.g., contamination or aggression), some individuals will compulsively check not only to reduce harm, but to neutralize contamination or aggressive obsessions (e.g., constantly checking the rear-view mirror for injured persons). Alternatively, one may wash repeatedly not out of concern of becoming contaminated but out of concern that they will carry infection to others. These manifestations are but a few ways that harming obsessions may differentiate, and yet appear topographically similar to other major symptom subtypes thus challenging the current models of OCD in their ability to adequately account for the full range of potential symptoms (McKay et al., 2004; Taylor, McKay, & Abramowitz, 2005). Finally, as harming is primarily associated with obsessions, with less prominent overt compulsions, individuals with this subtype are more likely to experience depression (Ricciardi & McNally, 1995), which is a significant liability for behavioral treatment of OCD (Steketee & Shapiro, 1995).

2.3.3. Symmetry/ordering

Symmetry obsessions have been found in 10–43% of OCD samples, and compulsions concerning symmetry/precision have been reported in 6–52% of individuals with OCD (Rasmussen & Eisen, 1992; Foa et al., 1995; Okasha, Saad, Khail, Dawla, & Yehia, 1994), making the symmetry/ordering subtype the second most commonly reported. This subtype of OCD is characterized by compulsions involving extensive and ritualistic repeating, counting, symmetry, ordering and arranging, as well as obsessions related to exactness/perfectionism and ‘obsessional slowness.’

A differentiating feature of this subtype is that, in contrast to the overwhelming fear and distress associated with obsessions of producing/preventing danger in contamination and harming subtypes, individuals with symmetry/ordering obsessions perform compulsive behaviors in order to reduce feelings of dissatisfaction, discomfort, or inadequacy, rather than anxiety or distress (McKay et al., 2004). Further, the underlying motivational factor for compulsions of symmetry and ordering seems to be markedly different from that of other forms of OCD in its attempt to resolve a sense of incompleteness rather than a fear of harm or shame (McKay et al., 2004).

2.3.4. Pure obsessions

Approximately 20–25% of OCD patients report distressing obsessions without overt compulsive behavioral rituals (Abramowitz, McKay, & Taylor, 2008; McKay et al., 2004). The pure obsessions subtype of OCD is characterized by the presence of obsessions in the absence of traditional anxiety-reducing compulsive behavior. Such obsessions may be of a religious/blasphemous, violent/aggressive, or sexual nature, and although these obsessions may co-occur with compulsions in other cases, for individuals with the pure obsessions subtype, they are not linked with any overt behavioral compulsions (Abramowitz et al., 2008). However, recent conceptualizations suggest that pure obsessions have distinct and unique compulsions such as mental rituals that resemble overt ones (Clark & Guyitt, 2008). Individuals with pure obsessions may experience their obsessions as either involuntary intrusive thoughts, impulses, or images (e.g., spontaneous and unacceptable aggressive thoughts) or reactive thoughts (e.g., cognitive rituals, repeating prayers) which are in response to external stimuli and are connected with a fear concerning harm, contamination, or perfection (Sookman et al., 2005). In either case, the cognitive model suggests that these intrusive thoughts are appraised as important by their mere occurrence, and
result in negative affect by their aversive quality (Taylor, Abramowitz, McKay, & Cuttler, in press).

2.3.5. Hoarding

One of the most disabling forms of OCD is hoarding, which is defined as the compulsive acquisition of items that often appear worthless to others (e.g., rotten food, trash, nail clippings), and associated difficulty discarding such items. Prevalence of hoarding symptoms among OCD patients has been estimated to range from 18–42% (Sookman et al., 2005). Individuals with hoarding symptoms report greater anxiety and depression, poorer insight, and more overvalued ideas (e.g., overemphasized and idealized beliefs which become congruent with self-identify to the point of creating rigidity and resistance to treatment) than individuals with other subtypes of OCD (McKay et al., 2004). Cognitively, these patients possess dysfunctional beliefs about the value of possessions and develop excessive emotional attachment to the hoarded items. In addition, these individuals develop associated symptoms such as obsessional fear of losing possessions they believe may be eventually necessary; perfectionism; intolerance of uncertainty; doubt (and overimportance) about memory; difficulty making decisions; procrastination; and behavioral avoidance (McKay et al., 2004; Taylor et al., 2007). Hoarding is so substantially unique that recent literature has called into question the appropriateness of its inclusion as a part of OCD (Petrusa et al., 2008).

2.4. Stability and change in OCD symptoms

Although OCD symptom presentation is generally similar across ages, there are some differences in pediatric and adult expression. For instance, some authors suggest that harm obsessions and hoarding compulsions are less frequent in adults than among children and adolescents, while sexual obsessions are thought to be more common in adults (e.g., Geller et al., 2001). At the individual level, symptoms often change over time and many individuals with childhood onset experience a wide range of symptoms by the time they reach adulthood (Rettew, Swedo, Leonard, Lenane, & Rapoport, 1992). As opposed to adults, children with OCD often have limited insight into their condition and may be less likely to recognize that their obsessions and compulsions are excessive or bizarre (Geffken et al., 2006). Storch, Milsom et al. (2008) investigated the relation between level of insight and clinical characteristics among 78 children and adolescents with OCD. They found that 45% of their sample had limited insight into their symptoms and experienced higher levels of OCD-related impairment. Nevertheless, independent of the level of insight, both children and adults may feel ashamed and embarrassed by their fears and ritualistic behaviors, so they often hide their symptoms, making early detection and treatment difficult.

2.5. Scope of functional impairment

In light of the review thus far, and the extent of potential complicating factors that may interfere with treatment, as well as the wide variety of symptoms and commonly associated comorbid presentations, there are many ways in which individuals with OCD can suffer significant functional impairments from the condition. At this point, it appears that OCD is (a) heterogeneous in symptom presentation; (b) commonly comorbid with other psychiatric disorders; and (c) has several potentially complicating additional presenting characteristics (i.e., overvalued ideas; scrupulosity) that limit direct therapeutic intervention. Collectively, these features lead to impairment in a number of specific areas as follows.

2.5.1. Sleep disturbance

Insomnia is a problem evident in a wide range of psychiatric problems. Recent reviews suggest that treating insomnia alleviates primary psychiatric disturbance (Smith, Huang, & Manber, 2005). In a review by Smith, Huang, & Manber (2005), there were few studies examining sleep in individuals with OCD. However, the authors reported that individuals with subjective complaints about their symptoms had poorer sleep. While the literature is sparse on the role of sleep disturbance in OCD, it is evident that insomnia is associated with greater psychopathology in a wide range of psychiatric conditions (Manber & Harvey, 2005). Among children, Storch, Murphy et al. (2008) found that 92% (N = 66) of children with OCD experienced at least one sleep problem, with 27.3% reporting five or more. Sleep problems were positively associated with OCD symptom severity, child-rated anxiety, and parent-proxy ratings of internalizing problems; following CBT, total and several specific SRPs (sleep related problems) were reduced.

2.5.2. Occupational and educational functioning

The research on occupational functioning is limited in OCD. On the one hand, some have found that the incidence of subclinical obsessive–compulsive attributes serves an adaptive function and may be particularly associated with some professional fields (Akiskal, Savino, & Akiskal, 2005). In a large (n = 409) study of outpatients, Yaryura-Tobias et al. (2000) found that symptom severity in OCD was negatively associated with occupational functioning. Common problems included loss of work, reduction to part-time status, or work in occupations unrelated to professional training (i.e., non-professional work when trained for professional career). Whereas the data are limited on abridged education in adults with OCD, the emerging literature on childhood OCD suggests that academic functioning is negatively associated with symptoms as well (Ledley & Pasupuleti, 2007).

2.6. Quality of life

Recently, in one of the most comprehensive assessments of the relation between symptoms and quality of life, Huppert et al. (2009) showed that individuals with OCD had significantly lower quality of life compared to individuals whose symptoms had remitted. Further, there was pervasive functional impairment, including work, social and family life. Finally, in additional support for the chronic impact of the disorder, individuals with a prior history of OCD, but few acute symptoms at the time of the study, had poorer quality of life and overall functioning than healthy controls. These findings are consistent with other research showing a direct negative relationship between symptom severity and quality of life in individuals with OCD (i.e., Eisen et al., 2006; Lack et al., 2009). One study found that the only group with a reported lower level of quality of life than individuals with OCD was those with schizophrenia (Bobes et al., 2001).

2.6.1. Healthcare utilization

Individuals with anxiety disorders in general have significantly higher rates of healthcare utilization than other psychiatric groups and non-psychiatric patients (Kessler et al., 1994; Levy, Maselko, Bauer, Richman, & Kubzansky, 2007). One possibility identified is the higher level of additional medical illness that aggregate in individuals with anxiety disorders (Levy et al., 2007) suggesting that the stress of having an anxiety disorder has physical health consequences. Interestingly, the physical health picture for individuals with OCD may be better than for other psychiatric disorders. While the study by Bobes et al. (2001) cited above noted very severe subjective quality of life, the group with OCD reported fewer physical health concerns than other patient groups, which included individuals with schizophrenia, substance abuse, and major depression. However, in light of the heterogeneity of OCD, it is not clear that this applies to the full range of individuals with the disorder, or how individuals with OCD compare to those with other anxiety disorders.
2.7. Comorbidity

Epidemiologic surveys suggest that, among individuals with OCD, 50% or more have at least one other psychological disorder, most commonly a comorbid anxiety disorder (e.g., social phobia), or a unipolar mood disorder (e.g., major depressive disorder) (Steketee & Barlow, 2002; Torres et al., 2006). Over a quarter of individuals with OCD present with comorbid alcohol abuse/dependence (Mancebo, Grant, Pinto, Eisen, & Rasmussen, 2009). OCD is also frequently comorbid with personality disorders. For example, Denys, Tenney, van Megen, de Geus, & Westenberg (2004) found that 20.7% of an OCD sample of 420 individuals had a personality disorder in the anxious (cluster C) cluster, 9.7% had a personality disorder in the emotionally expressive (cluster B) cluster, and 1.4% had a personality disorder in the bizarre/eccentric (cluster A) cluster (Denys, Tenney, van Megen, de Geus, & Westenberg, 2004). Comorbid obsessive–compulsive personality and dependent personality disorders were especially prevalent (Denys, Tenney, van Megen, de Geus, & Westenberg, 2004). Additionally, obsessive–compulsive symptoms have been reported in up to 59.2% of patients with schizophrenia, and up to one-third have met criteria for clinical OCD (Poyurovsky, Weizman, & Weizman, 2004).

3. Conclusions

The clinical picture for individuals with OCD is complex. The disorder is marked by a wide heterogeneity of presenting symptoms arranged into distinct domains requiring specific therapeutic interventions. Further complicating the implementation of treatment is the wide array of additional complexities described here, such as comorbid symptoms, deficits in neuropsychological functioning, not just right symptoms, and overvalued ideas or scrupulosity. These complex presentations all have important implications for functioning and quality of life in individuals with the disorder. However, research on the effects of treatment on quality of life and functional improvements is limited. The little empirical research accumulated to date suggests that some quality of life and functional impairments may persist, albeit to a lesser degree, following successful treatment of the condition (Huppert et al., 2009). While it appears that the adverse functional consequences of the disorder are evident at an early age (Ledley & Pasupuleti, 2007), it is unclear whether this may be reversed with successful treatment earlier in the developmental cycle such as through early intervention or preventative measures with at risk groups (i.e., in relation to psychiatric disorders generally; Taylor, Abramowitz, & McKay, 2010). These early findings are encouraging and merit replication and further investigation in the treatment of functional impairments and quality of life among individuals with various forms of OCD. In addition, presumed prevalence rates have been on the rise and are still probably underestimated as a result of embarrassment (e.g., of symptoms such as sexual images or elaborate blinking rituals) that may keep individuals from seeking treatment.

Although our understanding of OCD has increased significantly in the past two decades, and many theories have been proposed to explain the nature and etiology of OCD, no single model has fully accounted for the disorder’s complexity (Taylor, McKay, & Abramowitz, 2005). Accordingly, in future work, researchers and clinicians are encouraged to view OCD in a multi-dimensional manner, actively collaborating across such fields of study as genetics, neuroanatomy, and psychology. Future research may test multifaceted models examining, for example, cognitive, behavioral, and family factors that may serve as mediating or moderating variables between genetic and neurological risk factors and disorder onset and course. Given the wide heterogeneity of OCD, both in symptom presentation and complicating factors, additional research is warranted on which combination of factors are associated with greater functional impairment. This could shed light on treatment approaches which are most appropriate for a given subtype of OCD, and lead to meaningful treatment decision frameworks. Decision trees for treatment implementation have begun to emerge in other areas within cognitive behavior therapy (Marder & Chorpita, 2009). Such decision trees, taking into consideration symptom subtypes, complicating factors (such as overvalued ideation), comorbidity, and current functional impairment, would enhance treatment outcome for clients with obsessive–compulsive disorder.

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References


