Obsessive-compulsive spectrum disorders

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The obsessive-compulsive spectrum is an important concept referring to a number of disorders drawn from several diagnostic categories that share core obsessive-compulsive features. These disorders can be grouped by the focus of their symptoms: bodily preoccupation, impulse control, or neurological disorders. Although the disorders are clearly distinct from one another, they have intriguing similarities in phenomenology, etiology, pathophysiology, patient characteristics, and treatment response. In combination with the knowledge gained through many years of research on obsessive-compulsive disorder (OCD), the concept of a spectrum has generated much fruitful research on the spectrum disorders. It has become apparent that these disorders can also be viewed as being on a continuum of compulsivity to impulsivity, characterized by harm avoidance at the compulsive end and risk seeking at the impulsive end. The compulsive and impulsive disorders differ in systematic ways that are just beginning to be understood. Here, we review these concepts and several representative obsessive-compulsive spectrum disorders including both compulsive and impulsive disorders, as well as the three different symptom clusters: OCD, body dysmorphic disorder, pathological gambling, sexual compulsivity, and autism spectrum disorders.

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Obsessive-compulsive disorder (OCD) is characterized by obsessions and compulsions, but it has become clear that there are a significant number of other disorders that have core obsessive and compulsive features. Disorders that include such features cross several diagnostic categories and can be grouped according to the focus of the symptoms: bodily preoccupation, impulse control, or neurological disorders *(Table I)*. In addition to having obsessive and compulsive symptoms, all of these disorders also have some similarities in patient characteristics, course, comorbidities, neurobiology, or treatment response. Thus, an obsessive-compulsive (OC) spectrum has been proposed, for which all of these disorders are candidates.[1-4] Each of these disorders can often be chronic and devastating in terms of the suffering caused, the interference with functioning in important areas of life, and the economic toll to individuals and society.

Individuals with these disorders exhibit repetitive behaviors because they have a defect in the mechanism that enables them to inhibit acting.[2] The disorders vary in the extent to which they are characterized by compulsivity versus impulsivity, and this difference is often discussed in terms of a compulsive-impulsive spectrum.[2-4] They vary in numerous ways beginning with the phenomenology of this inability to resist acting. Compulsive disorders include OCD, body dysmorphic disorder (BDD), hypochondriasis, and anorexia nervosa. Individuals who act compulsively are avoiding risk and seeking safety; these individuals appear to have an exaggerated sense of harm and are driven to avoid harm or reduce anxiety and distress by performing the compulsive behaviors. The impulsive dis-

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Selected abbreviations and acronyms

ASD	*autism spectrum disorder*
BDD	*body dysmorphic disorder*
CBT	*cognitive behavioral therapy*
OCD	*obsessive-compulsive disorder*
OC	*obsessive-compulsive (spectrum)*
PG	*pathological gambling*
PRD	*paraphilia-related disorder*
SC	*sexual compulsivity*
SNRI	*serotonin and norepinephrine reuptake inhibitor*
SRI	*serotonin reuptake inhibitor*
SSRI	*selective serotonin reuptake inhibitor*

orders include, for example, pathological gambling (PG) and sexual compulsivity (SC). Those who act impulsively are risk takers, who underestimate the likelihood or severity of possible harm; they are seeking pleasure, arousal, or gratification; their actions may also be aggressive and are often accompanied by feelings of loss of control. The impulsive disorders are also often discussed as addictions, and treatment programs modeled after those used for substance abuse have arisen to treat them. These disorders have many similarities to addictions, but differ from traditional addictions in numerous ways, most notably in that they do not involve the intake of psychoactive substances. They are also sometimes considered as compulsive disorders, but are differentiated from compulsive disorders in our conceptualization for several reasons. For example, at least in the initial stages of the disorder, the repetitive behaviors are sought for pleasure and they involve risk taking rather than risk avoidance. The seemingly opposing drives of compulsivity and impulsivity can exist at the same time in one individual or appear at different times during the course of a disorder.

Category	Representative disorders
Bodily preoccupation	Body dysmorphic disorder (BDD)
	Hypochondriasis
	Eating disorders
	Depersonalization disorder
Impulse control	Pathological gambling (PG)
	Sexual compulsivity (SC)
	Kleptomania
	Trichotillomania
	Intermittent explosive disorder
	Borderline personality disorder
	Antisocial personality disorder
Neurological disorders	Autism
	Asperger disorder
	Tourette syndrome
	Sydenham chorea

Table I. Obsessive-compulsive disorders.

Baxter and his colleagues have suggested that OC spectrum disorders as a whole may involve corticostriatal dysfunction with the specific disorders having different areas of dysfunction within this system.[5,6] Structural imaging supports this hypothesis; studies have shown volumetric abnormalities in these structures in numerous OC spectrum disorders.[6] In addition, the different ends of the compulsive-impulsive spectrum seem to differ systematically in their pathophysiology and thus differ somewhat in their treatment response.[2,7] Indications are that compulsive disorders are characterized by increased frontal lobe activity and increased sensitivity of specific serotonin receptor subsystems, while impulsive disorders are characterized by decreased frontal lobe activity and decreased presynaptic serotonergic function.[2]

We will first outline the characteristics of OCD, the prototypical OC spectrum disorder, and then compare it with several OC spectrum disorders drawn from different symptom categories and from different ends of the compulsive-impulsive spectrum.

Obsessive-compulsive disorder

OCD is characterized by obsessions and compulsions. The obsessions are recurrent thoughts, impulses, or images, which are intrusive and ego dystonic; they are related to basic fears or urges that are distressing to the individual, such as contamination, aggression, sex, religion/scrupulosity, order/symmetry, hoarding, or pathological doubt. The compulsions are repetitive behaviors, including mental acts that the individual feels compelled to perform to reduce the anxiety created by the obsessions. The compulsions are often performed in specific ways, and can result in elaborate rituals.

With the exception of children, individuals with OCD recognize at some point in time that their obsessions are excessive or unreasonable. This insight can vary over time and from situation to situation. It is not unusual for an individual to have insight when not in an OCD-provoking situation, but to have insight disappear when faced with an OCD fear and thus feel compelled to perform a ritual.

The obsessions and compulsions are intrusive, preoccupying, and distressing. The obsessions interfere with

attention and concentration, thus interfering with cognitive tasks and often social interactions. The obsessions and compulsions can be very time-consuming: they interfere with functioning because of the time they occupy, and because patients with OCD often develop patterns of avoidance of situations or things that provoke their obsessions or compulsions.

OCD typically begins in late adolescence or early adulthood with an earlier age of onset for males than females.[8-10] In adult clinical samples, OCD is equally common in females as in males,[11] but, due to a higher incidence of childhood-onset OCD in males, younger samples have more males than females.[12] Compared with clinical samples, epidemiological studies tend to show a later age of onset and a higher proportion of females than males.[12]

The lifetime prevalence of OCD is estimated to be between 1.9% and 3.3%.[12] Most studies show a chronic course that extends across the lifetime with waxing and waning of symptoms, although in about 10% of cases there is a malignant deteriorating course.[9,13]

Neurobiological evidence shows clearly that the serotonin system is important in OCD. This evidence has come from treatment response to serotonin reuptake inhibitors (SRIs), including studies of SRIs versus desipramine, which demonstrated the selective efficacy of SRIs,[14,15] as well as from pharmacological challenge studies and cerebrospinal fluid neurotransmitter metabolite studies.[16] There is also evidence, however, of a role for the dopamine system in OCD on the basis of both theory (derived from basic human and animal research) and the efficacy of dopaminergic augmentation in refractory OCD.[16,17]

Neuroimaging in OCD has revealed much about the disorder and about the effects of treatment. Structural imaging supports the hypothesis that the OC spectrum disorders involve corticostriatal dysfunction[6]; specifically, magnetic resonance imaging (MRI) studies have shown volumetric abnormalities in the caudate and a rightward shift in caudate volume. Functional imaging in OCD has shown increased activity in the corticostriatal pathway involving the orbitofrontal cortex and the caudate nucleus.[6,18] Importantly, successful treatment of OCD with either SRI or cognitive behavioral therapy (CBT) results in normalization of orbitofrontal activity.[6,19,20]

There are now a number of pharmacotherapies available for treating OCD. The first medication discovered to be effective in OCD was clomipramine, a serotonin and norepinephrine reuptake inhibitor (SNRI).[21] The development of selective serotonin reuptake inhibitors (SSRIs) greatly expanded the options for treatment of OCD. The SSRIs have more favorable side-effect profiles than clomipramine, and have become the first-line treatments for OCD. They include citalopram, escitalopram, fluoxetine, fluvoxamine, paroxetine, and sertraline. Venlafaxine, a newer SNRI, is also used to treat OCD. Most have been established as effective in OCD through large controlled trials: citalopram,[22] clomipramine,[23] fluoxetine,[24,25] fluvoxamine,[26,27] paroxetine,[28] and sertraline.[29,30] In addition, there is evidence for the efficacy of venlafaxine in treatment-resistant OCD.[31] The SRIs (including all the SSRIs and the SNRIs clomipramine and venlafaxine) are generally used in higher doses for OCD than for depression and may require an extended period of time, 8 to 12 weeks or longer, before they ameliorate symptoms to a clinically significant degree. Two reasonably large studies report rates of response to SRI treatment for SRI-naive patients; these rates were about 53%[32] and 42%.[33] From 60% to 80% of patients with OCD respond to multiple trials of SRIs,[34-38] with most studies reporting nonresponder rates closer to 60%. No SRI has been proven more effective than others in head-to-head comparisons, so the selection of an SRI for individual patients with OCD can be made on the basis of side effects and half-life. The efficacy seems to be maintained over time with continuing SRI treatment,[28,39,40] but since OCD symptoms generally worsen during stress, some fluctuations in symptom severity while taking medication are not unusual; symptoms recur when treatment is ended.[28,39-41]

Unfortunately, even with a typical 30% to 60% decrease in their OCD symptom severity,[42] many patients are left with significant symptoms. Because of this, other pharmacological strategies have been used. Most commonly, neuroleptics or agents with serotonergic properties are used to augment SRI treatment. Of the neuroleptics, only risperidone has been established in controlled trials as an effective augmentation of SRIs in treatment-resistant OCD.[43,44] Additionally, several open-label trials have found olanzapine an effective augmentation of SRIs in OCD.[45-49] Subgroups of OCD patients may be particularly helped by neuroleptic augmentation; most definitively, patients with OCD and comorbid tics have responded well to this strategy,[17] which supports the position that the dopaminergic system plays a role in some subtypes of OCD. Haloperidol was found to be an effective augmentation to fluvoxamine in a placebo-controlled trial in patients with comorbid OCD and tics, but not in those with OCD

alone.[50] Similarly, there is some evidence that patients with schizotypal personality disorder may do better with neuroleptic augmentation. An open-label trial of pimozide was effective in treating the OCD symptoms in patients with either comorbid tics or schizotypal personality.[51]

Among the serotonergic agents reported in the literature as useful augmentations to SRIs in OCD are buspirone, lithium, trazodone, clonazepam, and clomipramine (augmenting an SSRI). Buspirone and lithium were reported to be helpful in OCD on the basis of open-label trials and case series, but controlled trials have produced disappointing results[21,52] There has been no controlled trial of trazodone augmentation. Small controlled trials have provided promising results for augmentation with clonazepam[42] and clomipramine.[53]

One other pharmacotherapy, pindolol, has been proven to be effective as an SRI augmentation agent in a small controlled study.[54]

The only proven psychological treatment for OCD is CBT; exposure and response prevention is the most established specific therapeutic technique and has been endorsed as the treatment of choice by the Expert Consensus Panel for Obsessive-Compulsive Disorder.[55] The first report of successful behavioral treatment of OCD was by Meyer in 1966[56]; since then numerous trials have been conducted to support its efficacy. Several meta-analyses of CBT trials have concluded that OCD symptoms improved significantly with CBT treatment.[57-61]

Body dysmorphic disorder

BDD or "imagined ugliness" is a disorder of body image in which a person is preoccupied and distressed by an appearance defect that is either imagined or, if there is a slight anomaly, their distress is markedly excessive compared with the anomaly itself.[62] The symptom dynamics are similar to OCD in that individuals suffering from BDD have obsessive thoughts or images that create distress, and they perform compulsive behaviors in an attempt to reduce the distress. In BDD, the obsessive thoughts focus on their imagined defect (eg, a horribly ugly face, nose, or other body part), what it means for their life (eg, rejection, humiliation, or social and occupational failure), and how they can solve the physical problem (eg, cosmetic surgery, dermatological or other treatments, or camouflage). The compulsive behaviors include checking their appearance (eg, looking in mirrors or asking others for reassurance), temporary solutions (eg, camouflaging with makeup, clothing, or accessories), or the search for permanent solutions (searching the Internet for new procedures, shopping for new creams or appliances, or consulting experts). They also compulsively scrutinize the appearance of others, particularly focusing on the feature(s) they dislike in themselves; this comparison, usually increases their distress at how badly they look, leading one patient to refer to it as "compare and despair." As with OCD, avoidance is prominent; BDD patients typically avoid social situations and situations in which they believe their disliked feature is particularly noticeable.

Like OCD, BDD is on the compulsive, harm-avoidant end of the compulsive-impulsive spectrum; patients are driven to prevent the social rejection and humiliation that they feel is inevitable due to their flawed appearance. Aside from the different obsessional focus, BDD differs from OCD in several other significant ways. BDD rituals tend to be less effective at reducing distress than OCD rituals. BDD is also characterized by poorer insight than OCD.[63] As noted earlier, most OCD patients realize, at least when not in an OCD moment, that their rituals make no sense; in contrast, many BDD patients believe that they really are ugly, abnormal, or even monstrous, and that their ritual behaviors not only make sense, but are also essential. Poor insight in BDD is a major deterrent to psychiatric and psychological treatment; most BDD patients present to cosmetic surgeons, dermatologists, dentists, or others who they think can resolve the appearance problem. They can be frustrated and angered by referral for mental health treatment because they see their appearance as the problem and fixing their appearance as the only solution.

There have been no epidemiological studies of BDD and so clear prevalence rates are not available; however, it has been estimated that as many as approximately 2% of nonclinical samples[64,65] and 12% of psychiatric outpatients[66] suffer from BDD. Like OCD, in clinical samples BDD appears to be equally prevalent among males and females.[67] It also has a chronic lifelong course with some waxing and waning of symptoms, including worsening under stress, but the majority of patients with BDD report a generally deteriorating course, rather than a steady or improving one.[68] BDD has a somewhat earlier age of onset than OCD with the average age of onset being in adolescence at 16 to 17 years of age.[67,68] In BDD, the focus of concern can change from one body part to another over time.

Work on the pathophysiology of BDD is just beginning. Recently, the first imaging study in BDD reported a shift

in caudate nucleus asymmetry and increased total white-matter volume.[69] These findings are consistent with the hypothesis that BDD is an OC spectrum disorder.

Like OCD, BDD has been shown to respond to SRIs and rarely to other pharmacological monotherapy. Two controlled SRI trials have been performed, one comparing clomipramine with desipramine,[70] thus establishing the selective efficacy of an SRI, and the second comparing fluoxetine with placebo,[71] further supporting the efficacy of SRIs. In practice, pharmacotherapy for BDD generally follows the same guidelines as for OCD, in terms of the agents used, dosages, and latency and maintenance of response. This similarity to OCD is supported by the two controlled trials, open-label trials, case series, and retrospective studies.[72-75] Since there are more cases with poor insight and perhaps more refractory cases, use of augmentation strategies may be more frequent. One difference from OCD is that pimozide seems to be ineffective in BDD on the basis of a double-blind, placebo-controlled trial of pimozide as an augmentation of fluoxetine (K. A. Phillips, personal communication). This is somewhat surprising since it is not only effective in some cases of OCD (albeit those with comorbid tics or schizotypal personality disorder), but also because it is effective in parasitosis, which was included along with BDD in the earlier diagnostic category monosymptomatic hypochondriasis.[76,77] A common difficulty in pharmacotherapy for BDD is that appearance-altering side effects must be kept in mind; patients with hair or skin concerns, for example, will be unlikely to accept a medication or be compliant with it if hair loss or skin problems are possible side effects.

Like OCD, BDD also seems to respond to CBT, particularly exposure and response prevention, rather than other psychotherapeutic interventions. A number of studies and case reports of group[78,79] and individual[80-82] treatment with CBT have shown promising results. Poor insight presents a challenge in terms of engaging patients in therapy, but does not preclude successful treatment. Indeed, correction of the misperception about their appearance does not seem to be necessary for successful treatment or to add to treatment success.[83]

Pathological gambling

PG is a disorder of impulse control characterized by recurrent gambling behavior that is maladaptive (ie, loss of judgment or excessive gambling) and in which personal, family, and/or vocational endeavors are disrupted.[62] PG shares many characteristics with other impulse control disorders such as kleptomania and pyromania in that individuals with these disorders have the irresistible impulse to perform harmful acts, have loss of control, may harm self or others, and engage in risky behavior. They share a pre-act arousal and/or tension, and the performance of the act results in relief or gratification, sometimes followed by guilt. PG is characterized by an inability to resist the urge to gamble and is often progressive. Patients may show "tolerance" and thus need to gamble with increasing amounts of money.

The course of PG tends to be chronic, although the pattern of gambling may be regular or episodic. During periods of gambling, the individuals often have hours of daily preoccupation with gambling, including planning future gambling, reliving past gambling experiences, and figuring out how to obtain money for gambling. They may lie and defraud people to finance their gambling. Individuals with PG commonly experience tormenting and devastating distress over their gambling behavior. Chronicity is often associated with increases in frequency and amount gambled; additionally, gambling may increase during periods of heightened stress. Gambling thus leads to more and more severe consequences, and more gambling, and may spiral out of control. Thus, the combination of illness chronicity, severe interference with normal life activities, and unavailability of treatment frequently leads to severe personal, familial, financial, social, and occupational impairment.

In 1998, 86% of adults in the USA were estimated to have participated in some type of gambling over their lifetime, up from 63% in 1975; the past-year figures increased only slightly, from 61% to 68%.[84] The past-year prevalence of PG among adults has been estimated to be between 0.9% and 2.0%, while the lifetime adult prevalence has been estimated to be between 1.5% and 2.3%.[85] The prevalence rates are higher among adolescents and college students.

As with OCD, demographic factors in treatment-seeking populations differ from those in epidemiological/general population surveys. Treatment-seeking PG patients are more likely than those identified by survey to be male (93% versus 64%), to be over 30 years old (82% versus 62%), and to be Caucasian (91% versus 57%).[86] Reported lifetime gambling increased for both males and females from 1975 to 1998; however, the increase was much larger for women, from 61% to 83%, than for men, from 75% to 88%, resulting in a decrease in the sex difference in gambling.[84] Yet, past-year gambling remains unchanged for

men, 68% versus 67%, while it increased slightly for women from 55% to 60%, resulting in only a slight decrease in the sex difference.[84] Legalized gambling has led to more gambling opportunities and new forms[87]; the explosion seems likely to account for the decrease in the sex difference in social gambling, yet the sex difference in PG has remained. This sex difference in PG, with males predominating in both clinical and population samples, is in contrast to the sex parity often found in OCD and BDD.

Gender differences have also been reported in the onset and course of PG. In males, PG usually begins in adolescence[88-90] or young adulthood,[90] and may remain undiagnosed for years. When male PG patients are first diagnosed, they often present with a 20- to 30-year gambling history, with gradual development of PG. In some cases, PG suddenly occurs in male social gamblers following a significant loss, stressor, or increased exposure.[91] In contrast, PG in females is more likely to occur later in life and delay in seeking treatment is approximately 3 years. Thus, as a result of the differences in onset and duration, female PG patients generally have a better prognosis than male PG patients.[91] Male and female gamblers differ in the types of gambling they prefer, with men more likely to bet on sporting events, cards, and at the track, while women prefer slot machines and bingo.[90] It is unknown whether males and females with PG represent truly different subgroups with differences in pathophysiology and treatment response.

We recently completed an FDG (fluorodeoxyglucose) positron emission tomography (PET) study in PG. The scans were acquired while the patients were engaged in a computerized gambling task either for a monetary reward or for computer points only. Gambling for monetary reward blackjack was associated with significantly higher relative metabolic rate in the primary visual cortex, the cingulate gyrus, the putamen, and prefrontal areas. We would expect normal subjects to show activation in both monetary and pure gambling conditions, but a study including both PG and social gamblers has not yet been done. In addition to demonstrating that the unique aspects of monetary reward compared with pure gambling are reflected in the activation patterns similarly to past imaging studies of reward strategy planning,[92] the results are generally consistent with symptom-provocation studies in OCD.

A possible selective efficacy of SRIs has been demonstrated in PG. Our studies have assessed the efficacy and tolerability of the SSRI fluvoxamine in PG without comorbidities. In small single-blind[93] and double-blind[94] trials, we found that fluvoxamine reduced gambling urges and behavior. Other recently published studies further establish the efficacy of SRIs in the treatment of PG. These include a small open-label citalopram trial[95] and a larger double-blind, placebo-controlled paroxetine trial.[96] Compared with OCD, the treatment response to SRIs is evident earlier and at lower doses. In addition, in an open-label trial, the serotonin antagonist nefazodone has been found to be effective in PG.[97]

PG seems to respond to a wider range of monotherapies than OCD; notably, there are case reports[98,99] and a single-blind study[100] suggesting that mood stabilizers are effective in PG. We recently completed a double-blind, placebo-controlled study of sustained-release lithium in PG patients with comorbid bipolar disorder. Lithium significantly improved both impulsive gambling and affective instability compared with placebo in this population. In addition, the opiate antagonist naltrexone may beneficial in PG.[101]

Just as PG responds to a wider range of pharmacological agents than does OCD, PG also responds to more psychotherapeutic modalities. Many treatment interventions for PG are similar to those for substance abuse disorders rather than OCD, and were created on the basis of the addiction model. The interventions reported in the literature for PG are self-help groups, inpatient treatment programs, and motivational interviewing (MI) approaches, as well as CBT.

Self-help groups such as Gamblers Anonymous (GA), which is structurally similar to Alcoholics Anonymous (AA), are widely available, but their efficacy is limited; only 8% of GA members reported total abstinence at a 1-year follow-up and 7% at a 2-year follow-up.[102] Inpatient treatment and rehabilitation programs for PG, also based on programs for substance abuse, emerged in the early 1970s.[103,104] Outcome studies show that approximately 55% of patients report abstinence at 1-year follow-up.[105,106] MI, which has been successful in treating alcohol use disorders, has recently been applied to PG with promising preliminary results.[107]

Behavioral and cognitive approaches have been used to treat PG. Aversive therapy was the most commonly employed early method[108] with published studies primarily based on small sample, uncontrolled studies of in vivo aversive therapy technique (eg, electric shocks).[109-112] Imaginal desensitization was found to be more effective than three other behavioral techniques (aversion therapy, imaginal relaxation, and in vivo exposure) in a sample of 120 patients.[113] CBT involving exposure and response pre-

vention—the technique used effectively for OCD—was found to substantially decrease gambling urges as reported in two case studies of PG.[114] It is known that gamblers make a number of cognitive errors that play a role in maintaining their disorder, and cognitive therapy aimed at correcting these errors and misperceptions has shown promise.[115,116] Thus, cognitive therapy may have potential for the treatment of PG either alone or, more likely, as part of a comprehensive treatment program; however, further structured and controlled investigations and long-term outcome studies are needed.

Sexual compulsivity

There are two general categories of SC. One category consists of paraphilias, which are recurrent sexual fantasies, urges, or behavior that involves nonhuman objects, the suffering or humiliation of oneself or one's partner, or children or other nonconsenting persons. They cause clinically significant distress or interfere with functioning in interpersonal and other areas.[62] Paraphilias include exhibitionism, fetishism, frotteurism, pedophilia, sexual masochism and sadism, and voyeurism, some of which have serious legal consequences. The second category of SC, referred to as paraphilia-related disorders (PRDs) and sometimes as sexual addiction, consists of individuals who engage in normative sexual arousal and behaviors, that is, masturbation and/or sexual behaviors that are typical in heterosexual or homosexual relationships, but carry out these behaviors at a frequency or intensity that creates problems in relationships or other areas of functioning. PRDs are not specified as disorders in the *Diagnostic and Statistical Manual of Mental Health, Fourth Edition, Text Revision (DSM-IV)*,[62] but can be diagnosed as a paraphilia not otherwise specified. Initially, the sexual behaviors of both the paraphilias and the PRD are usually pleasure producing; however, at least when the sexual compulsion is severe, it is clear that they are compulsive-repetitive behaviors. Individuals who have sexual compulsions often feel their behavior is out of control and the sexual activities themselves and the amount of time spent searching out or planning them can become extremely distressing and disruptive.

Sexual compulsions are distinct from the sexual obsessions commonly found in OCD. Sexual obsessions in OCD consist of sexual thoughts and images that are experienced as intrusive, ego dystonic, and morally repugnant. Ordinarily, these obsessions do not lead to carrying out the sexual acts and the individuals engage in ritual behaviors to prevent themselves from actually carrying out the sexual behavior or to "undo" their thoughts or potential behaviors. Although individuals with PRD may feel guilt or disgust at their behavior, they do carry out these behaviors and, at least initially, find them pleasurable.

Like PG, SC is on the impulsive side of the compulsive-impulsive spectrum; the behaviors can be considered risk seeking and, at least at the time of the activity, can be characterized by an underestimation of the negative consequences and an inability to control the behavior. This is the key to the increased risk of human immunodeficiency virus (HIV) among this population. Phenomenologically, as is characteristic of impulsive disorders, the repetitive sexual behavior has pleasurable aspects; however, over time, pleasure seems to be outweighed by distress.

Good epidemiological data are not available since sexual disorders have not been included in the major population studies conducted; indeed, it is difficult to see how these disorders could have been covered adequately since the likelihood of obtaining honest replies seems minimal. Estimates of the prevalence of SC range from 3% to 6%.[117] Paraphiliac sexual behaviors are thought to begin in childhood, adolescence, or early adulthood,[118] and PRDs are thought to begin around age 18 on average.[119,120] SC tends to be cyclical, but there is generally a worsening trend with the sexual activities becoming more extreme and functioning becoming more disrupted over time.[121] SC is three times more common in males than females.[122,123] This is a more extreme preponderance of males than found in PG, which is in contrast to the gender neutrality often found in clinical OCD and BDD samples.

Research on the pharmacotherapy of SC is limited, to date only case reports and small, non–placebo-controlled studies have been published, although we are currently conducting a placebo-controlled trial of citalopram in narcissistic personality disorder. As was found for OCD, there is evidence that SRIs are beneficial in SC. This is probably due in part to the side effect of decreasing libido, but it seems that SRIs also work by reducing obsessive thoughts and behaviors more directly. Their efficacy, however, seems to be more complex than that found for OCD. Stein[124] found that while patients with sexual obsessions had a strong response to SRIs, those with paraphilias had a more moderate response and those with PRD had a positive response on low doses, but a worsening of symptoms on high doses. Open-label trials of fluoxetine[125] and sertraline[126] found behavioral improvement in men with paraphilias and in men with

PRDs. Sertraline was also found helpful in reducing pedophiliac fantasy in an open-label trial.[127] A retrospective study of SSRIs found them useful in reducing fantasies in men with paraphilias.[128] Overall, these studies suggest that, in contrast to OCD, symptom improvement in SC can be seen in the first few weeks of treatment and at relatively low doses. Most of these trials were of short duration, and so response maintenance is unclear; however, there is indication that response may not be maintained in some patients.[126] In addition, compared with OCD and BDD, case reports indicate that SC may have a less preferential response to SRIs, also responding to monotherapy with mood stabilizers[129-131] and non-SRI antidepressants.[131-133] In terms of SRI dosage, time to response, response maintenance, and response to other pharmacotherapies, SC is more like PG, another disorder characterized by impulsivity, rather than OCD or BDD, which are more compulsive. One successful augmentation strategy has been reported. In a case series, Kafka and Hennen[134] found that men with SC and comorbid attention deficit-hyperactivity disorder (ADHD) (assessed retrospectively), who had residual SC symptoms despite adequate SSRI treatment, responded to psychostimulant augmentation.

There has been extensive research into psychological treatment for several of the paraphilias, such as pedophilia, due to the severity of the consequences and the involvement of the justice system. These generally indicate that CBT programs are relatively effective treatment, though, since they are not 100% effective, there is a problem with recidivism.[135-137] Few reports of psychological treatments for SC are available. Following the addiction model, self-help groups similar to AA are available, however, their efficacy has not been studied. Case reports suggest CBT may be effective.[138]

Autism spectrum disorders

Individuals with autism spectrum disorders (ASDs), including autistic disorder, pervasive developmental disorder, and Asperger's disorder, have significant deficits and/or delays in language and communication, and in social functioning, and they exhibit significant repetitive behaviors and restricted interests.

The diagnostic criteria for repetitive behaviors and restricted interests include ritualistic behaviors, such as counting, tapping, flicking, or repeatedly restating information, and compulsive behaviors, such as lining up objects, requiring a rigid adherence to routine, a marked resistance to change, and needing things to be "just so." These features are described as obsessive and compulsive features of the disorder,[138] marking its similarity to OCD and the OC spectrum disorders. The ASDs appear to be on the compulsive, harm-avoidant end of the compulsive-impulsive spectrum.

The lifetime prevalence for all pervasive developmental disorders, excluding Asperger's disorder, is 18.7/10 000 in studies done since 1989; the figure for the full syndrome of classical autistic disorder is 7.2/10 000.[139] There is a large sex difference in these disorders with males being much more likely to be affected than are females. The sex ratio is estimated at 3.1:1 overall for classical autism.[139]

Anxiety disorders have been studied in children with high functioning autism, such as Asperger's disorder, and results have shown that anxiety disorders, particularly OCD, are more prevalent in populations of these children compared with controls.[140,141] The familial aggregation of psychiatric disorders in the relatives of autistic probands has also been studied. Bolton et al[142] found the occurrence of OCD was significantly more common in first-degree relatives of autistic probands (3%) compared with relatives of Down syndrome probands (0%). In addition, the authors found that family members with OCD were also more likely to exhibit autistic-like social and communication impairments.[142] These researchers have also included OCDs as an indicator of ASD.[142] Piven et al[143] reported a significant rate of lifetime anxiety disorders in the parents of autistic children compared with controls (23.5% versus 2.9%).

Onset of these disorders is believed to be prior to or at birth, while symptoms are usually not evident until age 2 years or later; generally Asperger's disorder is not recognized until later. ASDs are chronic, devastating neuropsychological disorders and are four times more common in males than females.

While many hypotheses have been explored to explain the etiology of this cluster of disorders, no single cause has been agreed upon, though the research exploring genetic factors is one of the most promising.[144,145] Recent advances in imaging have been fruitful in research on understanding ASDs. These disorders have very complex and vast symptoms, but their neural substrates are beginning to be untangled. At this time, it seems clear that delayed frontal lobe metabolic maturation occurs in autism,[146] which may be related to some of the early repetitive behaviors. There is also bilateral temporal hypoperfusion.[147,148] Overall, there seems to be a widespread disorganized establishment of

neural circuits.[149] Abnormalities in the cerebellum with a wide range of consequences has also been established.[150]
As in OCD, hypotheses of the etiology of ASD suggest dysregulation of the serotonin system.[151] SRIs, the treatments of choice for OCD, have been used clinically in the treatment of repetitive behaviors in autism. Promising results have been found in small controlled trials of the efficacy of clomipramine and fluvoxamine,[152] and we are currently conducting controlled studies of the efficacy of fluoxetine versus placebo in both childhood and adult autism.
Given the complex, multifaceted symptomatology found in ASDs, we do not expect one class of agents to be uniquely effective in treating their global severity. Rather, it is likely that treatments will be most effective against targeted symptoms. Since these disorders also have an impulsive element, with sometimes prominent aggression, self-injury, and mood instability, we are conducting a double-blind, placebo-controlled study of the efficacy of the mood-stabilizer divalproex sodium in children and adolescents with autism.
Other successful treatments of ASDs include intensive behavioral therapies are the most widely recognized modalities of treatment for ASDs. Home- and school-based behavioral therapies aim toward reducing repetitive and self-/other injurious behaviors and increasing communication and social skills.

Conclusions

The concept of an OC spectrum of related disorders is a powerful one that has helped generate theoretical discussion and research questions in broad areas of their etiology, neurobiology, and treatment. Though coming from a wide range of diagnostic categories and differing in significant ways, research to date suggests that, in addition to sharing some symptom patterns, these disorders have many other similarities. OCD has the most developed knowledge base of these disorders and serves as a guide for future research in the others. The significant amount of empirically based knowledge available in OCD has been valuable in providing direction for both pharmacological and psychological treatment research, and is proving important in areas where research is just beginning, such as neuroimaging.
It is clear that the OC spectrum disorders differ in systematic ways and that looking at them in terms of compulsivity and impulsivity is adding focus to research on their etiology, neurobiology, and treatment. Most notably, research available to date indicates that, while many of these disorders seem to respond meaningfully to SRI treatment, the compulsive disorders seem to require higher dosages, have a substantial latency to response, and that response is maintained throughout treatment; in contrast, impulsive disorders may require lower doses and have a relatively quick response. As research into the etiology and neurobiology continues, both the concept of the OC spectrum and the significance of compulsivity and impulsivity will be tested further. ❑

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Trastornos del espectro obsesivo compulsivo

El espectro obsesivo compulsivo constituye un importante concepto que se refiere a un número de trastornos descritos a partir de algunas categorías diagnósticas que comparten características comunes obsesivo compulsivas. Estos trastornos se pueden agrupar según los síntomas más relevantes: preocupaciones corporales, control de impulsos o trastornos neurológicos. Aunque los trastornos son claramente distintos unos de otros, son muchas sus semejanzas en la fenomenología, la etiología, la fisiopatología, las características de los pacientes y la respuesta terapéutica. En combinación con el conocimiento obtenido a través de muchos años de investigación en el trastorno obsesivo compulsivo (TOC), el concepto de espectro ha generado una investigación muy fructífera acerca de los trastornos del espectro. En apariencia, estos trastornos también pueden ser considerados como un continuo desde la compulsión hasta la impulsividad, caracterizados por la evitación de daño en el extremo compulsivo y la búsqueda de riesgo en el extremo impulsivo. Recientemente se está empezando a comprender que los trastornos compulsivos e impulsivos se pueden diferenciar sistemáticamente. En este artículo se revisan estos conceptos y algunos trastornos representativos del espectro obsesivo compulsivo, incluyendo tanto los trastornos compulsivos e impulsivos como las tres diferentes agrupaciones sintomáticas: TOC, trastorno corporal dismórfico, juego patológico, compulsión sexual y trastornos del espectro autista.

Troubles du spectre des obsessions-compulsions

Le spectre des obsessions-compulsions est un concept important qui se réfère à un certain nombre de troubles issus de catégories diagnostiques différentes ayant comme point commun des caractéristiques intrinsèques d'obsessions-compulsions. Ces troubles peuvent être regroupés autour d'une symptomatologie centrale : préoccupations corporelles, contrôle d'impulsions, troubles neurologiques. Bien que les troubles soient vraiment distincts les uns des autres, ils présentent des ressemblances remarquables que ce soit dans la phénoménologie, l'étiologie, la physiopathologie, les caractéristiques des patients et la réponse au traitement. L'association du concept d'un spectre des obsessions-compulsions avec les connaissances acquises au cours des nombreuses années d'études des troubles obsessionnels compulsifs (TOC), a été plus fructueuse sur le plan de la recherche. Il est apparu que ces troubles pouvaient être envisagés comme un continuum allant de la compulsion à l'impulsion, caractérisé par l'évitement de la souffrance à l'extrémité compulsive et par la recherche du risque à l'extrémité impulsive. Les troubles impulsifs et compulsifs diffèrent par des aspects systématiques que l'on commence seulement à comprendre. Nous passons ici en revue ces concepts et plusieurs troubles représentatifs du spectre des obsessions-compulsions y compris les troubles compulsifs et impulsifs ainsi que différents groupes de symptômes : TOC, peur d'une dysmorphie corporelle, jeu pathologique, compulsion sexuelle et troubles du spectre autistique.

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