bsessive-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. 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. 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. They vary in numerous ways beginning with the phenomenology of this inability to resist acting. Compulsive disorders include OCD, body dysmorphic disorder (BDD), hypochondria, 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-

Keywords: obsessive-compulsive disorder; body dysmorphic disorder; pathological gambling; sexual compulsivity; autism; Asperger's disorder; impulsivity

Author affiliations: Department of Psychiatry, Mount Sinai School of Medicine, New York, NY, USA

Address for correspondence: Andrea Allen, PhD, Department of Psychiatry, Mount Sinai School of Medicine, Box 1230, One Gustave L. Levy Place, New York, NY 10029-6574, USA
(e-mail: andrea.allen@mssm.edu)
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.

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. Structural imaging supports this hypothesis; studies have shown volumetric abnormalities in these structures in numerous OC spectrum disorders. 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. 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.

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. In adult clinical samples, OCD is equally common in females as in males, but, due to a higher incidence of childhood-onset OCD in males, younger samples have more males than females. Compared with clinical samples, epidemiological studies tend to show a later age of onset and a higher proportion of females than males. The lifetime prevalence of OCD is estimated to be between 1.9% and 3.3%. 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. 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, as well as from pharmacological challenge studies and cerebrospinal fluid neurotransmitter metabolite studies.

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. 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 cortico striatal dysfunction; 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 cortico striatal pathway involving the orbitofrontal cortex and the caudate nucleus. Importantly, successful treatment of OCD with either SRI or cognitive behavioral therapy (CBT) results in normalization of orbitofrontal activity.

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 noradrenergic reuptake inhibitor (SNRI). 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, clomipramine, fluoxetine, fluvoxamine, paroxetine, and sertraline. In addition, there is evidence for the efficacy of venlafaxine in treatment-resistant OCD. 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% and 42%. From 60% to 80% of patients with OCD respond to multiple trials of SRIs 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, 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. Unfortunately, even with a typical 30% to 60% decrease in their OCD symptom severity, 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. Additionally, several open-label trials have found olanzapine an effective augmentation of SRIs in OCD. 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, 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. 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. 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. There has been no controlled trial of trazodone augmentation. Small controlled trials have provided promising results for augmentation with clonazepam and clomipramine.

One other pharmacotherapy, pindolol, has been proven to be effective as an SRI augmentation agent in a small controlled study. 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. The first report of successful behavioral treatment of OCD was by Meyer in 1966; 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.

**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. 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 (e.g., a horribly ugly face, nose, or other body part), what it means for their life (e.g., rejection, humiliation, or social and occupational failure), and how they can solve the physical problem (e.g., cosmetic surgery, dermatological or other treatments, or camouflage). The compulsive behaviors include checking their appearance (e.g., looking in mirrors or asking others for reassurance), temporary solutions (e.g., 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. 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 non-clinical samples and 12% of psychiatric outpatients suffer from BDD. Like OCD, in clinical samples BDD appears to be equally prevalent among males and females. 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. 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. 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. 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, thus establishing the selective efficacy of an SRI, and the second comparing fluoxetine with placebo, 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. 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. 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 and individual 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.

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. 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%. 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%. 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%). 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. 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. Legalized gambling has led to more gambling opportunities and new forms; 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 or young adulthood, 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 history, with gradual development of PG. In some cases, PG suddenly occurs in male social gamblers following a significant loss, stressor, or increased exposure. 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. 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. 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, the results are generally consistent with symptom-provocation studies in OCD. A possible selective efficacy of SSRIs 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 and double-blind trials, we found that fluvoxamine reduced gambling urges and behavior. Other recently published studies further establish the efficacy of SSRIs in the treatment of PG. These include a small open-label citalopram trial and a larger double-blind, placebo-controlled paroxetine trial. Compared with OCD, the treatment response to SSRIs 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. PG seems to respond to a wider range of monotherapies than OCD; notably, there are case reports and a single-blind study 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 be beneficial in PG. 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. Inpatient treatment and rehabilitation programs for PG, also based on programs for substance abuse, emerged in the early 1970s. Outcome studies show that approximately 55% of patients report abstinence at 1-year follow-up, MI, which has been successful in treating alcohol use disorders, has recently been applied to PG with promising preliminary results. Behavioral and cognitive approaches have been used to treat PG. Aversive therapy was the most commonly employed early method with published studies primarily based on small sample, uncontrolled studies of in vivo aversive therapy technique (eg, electric shocks). Imaginal desensitization was found to be more effective than other behavioral techniques (aversion therapy, imaginal relaxation, and in vivo exposure) in a sample of 120 patients. CBT involving exposure and response pre-
vitation—the technique used effectively for OCD—was found to substantially decrease gambling urges as reported in two case studies of PG.\textsuperscript{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.\textsuperscript{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 compulsion**

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.\textsuperscript{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),\textsuperscript{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%.\textsuperscript{117} Paraphiliac sexual behaviors are thought to begin in childhood, adolescence, or early adulthood,\textsuperscript{118} and PRDs are thought to begin around age 18 on average.\textsuperscript{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.\textsuperscript{121} SC is three times more common in males than females.\textsuperscript{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\textsuperscript{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\textsuperscript{125} and sertraline\textsuperscript{126} found behavioral improvement in men with paraphilias and in men with sexual addiction.
PRDs. Sertraline was also found helpful in reducing pedophilic fantasy in an open-label trial. A retrospective study of SSRIs found them useful in reducing fantasies in men with paraphilias. 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. In addition, compared with OCD and BDD, case reports indicate that SC may have a less preferential response to SSRIs, also responding to monotherapy with mood stabilizers and non-SRI antidepressants. 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 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. 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.

**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, 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. 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.

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. The familial aggregation of psychiatric disorders in the relatives of autistic probands has also been studied. Bolton et al 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. These researchers have also included OCDS as an indicator of ASD. Piven et al 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. 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, which may be related to some of the early repetitive behaviors. There is also bilateral temporal hypoperfusion. Overall, there seems to be a widespread disorganized establishment of
neural circuits. Abnormalities in the cerebellum with a wide range of consequences has also been established. As in OCD, hypotheses of the etiology of ASD suggest dysregulation of the serotonin system. 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, 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.

We would like to acknowledge grants from the National Institutes of Health (1 U54 MH66673), the National Institute of Mental Health (5 RO1 MH58935), the National Institute of Drug Abuse (DA 10234), the Food and Drug Administration (FD R 002026; FD R 001520), the National Institute of Neurological Diseases and Stroke (1 R21 NS543979), and an unrestricted grant from the Paula and Bill Oppenheim (PBO) Foundation.

REFERENCES

1. Hollander E. Obsessive-Compulsive-Related Disorder. Washington, DC: American Psychiatric Press; 1993.
2. Hollander E, Wong CM. Obsessive-compulsive spectrum disorders. J Clin Psychiatry. 1995;56(suppl 4):7-12.
3. Hollander E, Rosen J. Obsessive-compulsive spectrum: a review. In: Maj M, Santorius N, Okasha A, Zohar J, eds. Obsessive-Compulsive Disorder. New York, NY: John Wiley & Sons; 2000.
4. Phillips KA. The obsessive-compulsive spectrums. Psychiatr Clin N Am. 2002;25:791-809.
5. Baxter LR, Schwartz JM, Guze BH, Bergman K, Szuba MP. Neuroimaging in obsessive-compulsive disorder: seeking the mediating neuroanatomy. In: Jenike MA, Baer L, Minichiello W, eds. Obsessive-Compulsive Disorders: Practical Management. 2nd ed. Chicago, Ill: Year Book Medical Publishers; 1990:167-188.
6. Rauch SL, Baxter LR. Neuroimaging of OCD and related disorders. In: Jenike MA, Baer L, Minichiello WE, eds. Obsessive-Compulsive Disorders: Practical Management. Boston, Mass: Mosby; 1998:289-317.
7. Hollander E. Obsessive-compulsive disorder-related disorders: the role of selective serotonergic reuptake inhibitors. Int Clin Psychopharmacol. 1996;11(suppl 5):75-87.
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 sistémicamente. 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.

8. Myers JK, Weissman MM, Tischler GL, et al. Six-month prevalence of psychiatric disorders in three communities 1980 to 1982. Arch Gen Psychiatry. 1984;41:959-967.
9. Rasmussen SA, Tsuang MT. DSM-III obsessive-compulsive disorder: clinical characteristics and family history. Am J Psychiatry. 1986;143:317-322.
10. Weissman MM, Bland RC, Canino GJ, et al. The cross-national epidemiology of obsessive-compulsive disorder. J Clin Psychiatry. 1994;55(suppl 3):5-10.
11. Foa EB, Kozak MJ, Goodman WK, Hollander E, Jenike MA, Rasmussen SA. DSM-IV field trial: obsessive-compulsive disorder. Am J Psychiatry. 1995;152:90-96.
12. Horwath E, Weissman MM. The epidemiology and cross-national presentation of obsessive-compulsive disorder. Psychiatr Clin N Am. 2000;23:493-507.
13. Goodwin DW, Guze SB, Robbins E. Follow-up studies in obsessive neurosis. Arch Gen Psychiatry. 1969;20:182-187.
14. Goodman WK, Price LH, Delgado PL, et al. Specificity of serotonin reuptake inhibitors in the treatment of obsessive-compulsive disorder. Arch Gen Psychiatry. 1990;47:577-585.
15. Leonard H, Swedo SE, Rapoport J, et al. Treatment of childhood obsessive-compulsive disorder with clomipramine and desipramine: a double-blind crossover comparison. Arch Gen Psychiatry. 1989;46:1088-1092.
16. Stein DJ. Neurobiology of the obsessive-compulsive spectrum disorders. Biol Psychiatry. 2000;47:296-304.
17. Goodman WK, McDougle CJ, Price LH, Riddle MA, Pauls DL, Leckman JF. Beyond the serotonin hypothesis: a role for dopamine in some forms of obsessive-compulsive disorder. J Clin Psychiatry. 1990;51(suppl):36-43.
18. Saxena S, Rauch SL. Functional neuroimaging and the neuroanatomy of obsessive-compulsive disorder. Psychiatr Clin North Am. 2000;23:563-586.
19. Baxter LR, Schwartz JM, Bergman KS, et al. Caudate glucose metabolic rate changes with both drug and behavior therapy for obsessive-compulsive disorder. Arch Gen Psychiatry. 1992;49:681-689.
20. Saxena S, Brody AL, Ho ML, et al. Differential cerebral metabolic changes with paroxetine treatment of obsessive-compulsive disorder versus major depression. Arch Gen Psychiatry. 2002;59:250-261.
21. Hollander E, Kaplan A, Allen A, Cartwright C. Pharmacotherapy for obsessive-compulsive disorder. Psychiatr Clin North Am. 2000;23:643-656.
22. Montgomery SA, Kasper S, Stein DJ, Bang Hedgaard K, Lemming OM. Citalopram 20 mg, 40 mg and 60 mg are all effective and well tolerated compared with placebo in obsessive-compulsive disorder. Int Clin Psychopharmacol. 2001;16:75-86.
23. Clomipramine Collaborative Study Group. Clomipramine in the treatment of patients with obsessive-compulsive disorder. Arch Gen Psychiatry. 1991;48:730-738.
24. Montgomery SA, McIntyre A, Osterheider M, et al. A double-blind, placebo-controlled study of fluoxetine in patients with DSM-III-R obsessive-compulsive disorder. Eur Neuropsychopharmacol. 1993;3:143-152.
25. Tollefson GD, Rampey AH, Potvin JH, et al. A multicenter investigation of fixed-dose fluoxetine in the treatment of obsessive-compulsive disorder. Arch Gen Psychiatry. 1994;51:559-567.
26. Goodman WK, Price LH, Rasmussen SA, Delgado PL, Heninger GR, Charney DS. Efficacy of fluoxetine in obsessive-compulsive disorder. A double-blind comparison with placebo. Arch Gen Psychiatry. 1989;46:36-44.
27. Jenike MA, Hyman S, Baer L, Holland A, et al. A controlled trial of fluoxetine in obsessive-compulsive disorder: implications for a serotonergic theory. Am J Psychiatry. 1990;147:1209-1215.
28. Hollander E, Allen A, Steiner M, Wheadon DE, Oakes R, Burnham D. Acute and long-term treatment and prevention of relapse of obsessive-compulsive disorder with paroxetine. J Clin Psychiatry. 2003. In press.
29. Chouinard G, Goodman W, Greist JH, et al. Results of a double-blind placebo-controlled trial of a new serotonin uptake inhibitor, sertraline, in the treatment of obsessive-compulsive disorder. Psychopharmacol Bull. 1990;26:279-284.
30. Greist J, Chouinard G, Duboff E, et al. Double-blind parallel comparison of three dosages of sertraline and placebo in outpatients with obsessive-compulsive disorder. Arch Gen Psychiatry. 1995;52:289-295.
31. Hollander E, Friedberg J, Wasserman S, Allen A, Birnbaum M, Koran LM. Venlafaxine in treatment-resistant obsessive-compulsive disorder. J Clin Psychiatry. 2003;64:546-550.
32. Rasmussen SA, Baer L, Eisen J, Shera D. Previous SRI treatment and efficacy of sertraline for OCD: combined analysis of 4 multicenter trials. Poster presented at the 150th Annual Meeting of the American Psychiatric Association, San Diego, Calif, May 17-22, 1997.
33. Ackerman DL, Greenland S, Bystritsky A. Clinical characteristics of response to fluoxetine treatment of obsessive-compulsive disorder. J Clin Psychopharmacol. 1998;18:185-192.
34. Goodman WK, McDougle CJ, Price LH. Pharmacotherapy of obsessive compulsive disorder. J Clin Psychiatry. 1992;53(suppl 4):29-37.
35. Rasmussen SA, Eisen JL, Pato MT. Current issues in the pharmacologic management of obsessive compulsive disorder. J Clin Psychiatry. 1993;54(suppl 6):4-9.
36. Jenike MA, Rauch SL. Managing the patient with treatment-resistant obsessive-compulsive disorder: current strategies. J Clin Psychiatry. 1994;55(suppl 3):11-17.
37. Piccinni M, Pini S, Bellantuono C, Wilkinson G. Efficacy of drug treatment in obsessive-compulsive disorder: a meta-analytic review. Br J Psychiatry. 1995;166:424-443.
38. Hollander E, Pallanti S. Current and experimental therapeuticse of obsesso-compulsive disorders. In: Davis K, Charney D, Coyle J, Nemeroff C, eds. Neuropsychopharmacology: The Fifth Generation of Progress. Philadelphia, Pa: Lippincott Williams & Wilkins; 2002:1647-1664.
39. Leonard HL, Swedo SE, Lenane MC, et al. A double-blind desipramine substitution during long-term clomipramine treatment in children and adolescents with obsessive-compulsive disorder. Arch Gen Psychiatry. 1991;48:922-927.
40. Koran LM, Hackett E, Rubin A, Wolkow R, Robinson D. Efficacy of sertraline in the long-term treatment of obsessive-compulsive disorder. Am J Psychiatry. 2002;159:88-95.
41. Pato MT, Zohar-Kadouch R, Zohar J, Murphy DL. Return of symptoms after discontinuation of clomipramine in patients with obsessive-compulsive disorder. Am J Psychiatry. 1988;145:1521-1525.
42. Jenike MA. Drug treatment of obsessive-compulsive disorders. In: Jenike MA, Baer L, Minichiello W, eds. Obsessive-Compulsive Disorders: Practical Management. New York, NY: Mosby; 1998.
43. McDougle CJ, Epperson CN, Pelton GH, Wasylink S, Price LH. A double-blind, placebo-controlled study of risperidone addition in serotonin reuptake inhibitor-refractory obsessive-compulsive disorder. Arch Gen Psychiatry. 2000;57:794-801.
44. Hollander E, Baldini Ross N, Sood, E, Pallanti S. Risperidone augmentation in SRI-resistant OCD: a preliminary double-blind, placebo controlled study. Int J Neuropsychopharmacol. 2003. In press.
45. Weiss EL, Potenza MN, McDougle CJ, Epperson CN. Olanzapine addition in obsessive-compulsive disorder refractory to selective serotonin reuptake inhibitors: an open-label case series. J Clin Psychiatry. 1999;60:524-527.
46. Rogetto E, Bellino S, Vasettro P, Ziero S. Olanzapine augmentation of fluvoxamine-refractory obsessive-compulsive disorder (OCD): a 12-week open trial. Psychiatry Res. 2000;96:91-98.
47. Koran LM, Ringold AL, Elliott MA. Olanzapine augmentation for treatment-resistant obsessive-compulsive disorder. J Clin Psychiatry. 2000;61:514-517.
48. Francobandiera G. Olanzapine augmentation of serotonin uptake inhibitors in obsessive-compulsive disorder: an open study. Can J Psychiatry. 2001;46:356-358.
49. Crocq MA, Leclercq P, Guillou MS, Bailey PE. Open-label olanzapine in obsessive-compulsive disorder refractory to antidepressant treatment. Eur Psychiatry. 2002;17:296-297.
50. McDougle CJ, Goodman WK, Leckman JF, Lee NC, Heninger GR, Price LH. Haloperidol addition in fluvoxamine-refractory obsessive-compulsive disorder. Arch Gen Psychiatry. 1994;51:302-308.
51. McDougle CJ, Goodman WK, Price LH, et al. Neuroleptic addition in fluvoxamine-refractory obsessive-compulsive disorder. Am J Psychiatry. 1999;147:652-654.
52. Jenike MA. An update on obsessive-compulsive disorder. Bull Menninger Clin. 2001;65:4-25.
53. Pallanti S, Quercioli L, Paiva RS, Koran LM. Citalopram for treatment-resistant obsessive-compulsive disorder. Eur Psychiatry. 1999;14:101-106.
54. Dannon PN, Sasso Y, Hirschman S, Ianu I, Grunhaus L, Zohar J. Pindolol augmentation in treatment-resistant obsessive compulsive disorder: a double-blind placebo-controlled trial. Eur Neuropsychopharmacol. 2000;10:165-169.
55. Expert Consensus Panel for Obsessive-Compulsive Disorder. Treatment of obsessive-compulsive disorder. J Clin Psychiatry. 1997;58(suppl 4):3-28.
56. Meyer V. Modification of expectations in cases with obsessional rituals. Behav Res Ther. 1966;4:273-280.
57. Christensen H, Hadzi-Pavlovic D, Andrews G, Mattick R. Behavior therapy and tricyclic medication in the treatment of obsessive-compulsive disorder: a quantitative review. J Consult Clin Psychol. 1987;55:701-711.
58. Cox BJ, Swinson RP, Morrison BL, Paul S. Clomipramine, fluoxetine, and behavior therapy in the treatment of obsessive-compulsive disorder: a meta-analysis. J Behav Ther Exp Psychiatry. 1993;24:149-153.
59. van Blakom AJLM, van Oppen P, Vermeulen AWA, et al. A meta-analysis on the treatment of obsessive-compulsive disorder: a comparison of antidepressants, behavior, and cognitive therapy. Clin Psychol Rev. 1994;14:359-381.
60. Abramowitz JS. Does cognitive-behavioral therapy cure obsessive-compulsive disorder? A meta-analytic evaluation of clinical significance. Behav Ther. 1998;29:339-355.
61. Kobak KA, Greist JH, Jefferson JW, Katselniuk DJ, Henk HJ. Behavioral versus pharmacological treatments of obsessive-compulsive disorder: a meta-analysis. Psychopharmacology, 1998;136:205-216.
62. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 4th ed, Text Revision. Washington, DC: American Psychiatric Association; 2000.
66. Zimmerman M, Mattia JI, Phillips KA. Screening for body dysmorphic disorder in an outpatient clinic. Syllabus and Proceedings Summary, American Psychiatric Association 149th Annual Meeting, New York, NY: American Psychiatric Association; 1996.

67. Phillips KA, Diaz SF. Gender differences in body dysmorphic disorder. J Nerv Ment Dis. 1997;185:570-577.

68. Phillips KA, McElroy SL, Keck PE, Hudson J, Pope HG. A comparison of delusional and non-delusional body dysmorphic disorder in 100 cases. Psychopharmacol Bull. 1994;30:179-186.

69. Rauch SL, Phillips KA, Segal E, et al. A preliminary morphometric magnetic resonance imaging study of regional brain volumes in body dysmorphic disorder. Psychiatry Res. 2003;122:13-19.

70. Hollander E, Allen A, Kwan J, et al. Clomipramine vs desipramine crossover trial in body dysmorphic disorder. Arch Gen Psychiatry. 1999;56:1033-1039.

71. Phillips KA, Albertini RS, Rasmussen SA. A randomized placebo-controlled trial of fluoxetine in body dysmorphic disorder. Arch Gen Psychiatry. 2002;59:381-388.

72. Phillips KA, McElroy SL, Hudson JI, Pope HG Jr. Body dysmorphic disorder: an obsessive-compulsive spectrum disorder, a form of affective spectrum disorder, or both? J Clin Psychiatry. 1995;56(suppl 4):41-51.

73. Hollander E. Treatment of obsessive-compulsive spectrum disorders with SSRIs. Br J Psychiatry. 1998;35(suppl):7-12.

74. Phillips KA, Albertini RS, Siniscalchi JM, Khan A, Robinson M. Effectiveness of pharmacotherapy for body dysmorphic disorder: a chart-review study. J Clin Psychiatry. 2001;62:721-727.

75. Saxena S, Winograd A, Dunkin JJ, et al. A retrospective review of clinical characteristics and treatment response in body dysmorphic disorder versus obsessive-compulsive disorder. J Clin Psychiatry. 2001;62:67-72.

76. Munro A. Monosymptomatic hypochondriacal psychosis manifesting as delusions of parasitosis: a description of four cases successfully treated with pimozide. Arch Dermatol. 1978;140:940-943.

77. Zomer SF, DeWit RF, Van Bronswijk JE, Nabarro G, Van Vloten WA. Delusions of parasitosis. A psychiatric disorder to be treated by dermatologists? An analysis of 33 patients. Br J Dermatol. 1998;138:1030-1032.

78. Rosen JC, Reiter J, Orosan P. Cognitive-behavioral body image therapy for body dysmorphic disorder. J Consult Clin Psychol. 1995;63:263-269.

79. Wilhelm S, Otto MW, Lohr B, Deckersbach T. Cognitive behavior group therapy for body dysmorphic disorder: a case series. Behav Res Ther. 1999;37:71-75.

80. Veale D, Gournay K, Dryden W, et al. Body dysmorphic disorder: a cognitive-behavioural model and pilot randomised controlled trial. Behav Res Ther. 1996;34:717-729.

81. McKay D, Todaro J, Neziroglu F, Campisi T, Moritz EK, Yaryura-Tobias JA. Body dysmorphic disorder: a preliminary evaluation of treatment and maintenance using exposure with response prevention. Behav Res Ther. 1997;35:67-70.

82. Neziroglu F, Yaryura-Tobias JA. Exposure, response prevention, and cognitive therapy in the treatment of body dysmorphic disorder. J Behav Ther Exp Psychi. 1993;24:431-438.

83. Rosen JC, Cado S, Silberg NT, et al. Cognitive behavior therapy with and without size perception training for women with body image disturbance. Behav Ther. 1990;21:481-498.

84. National Gambling Impact and Behavior Study Commission. National Opinion Research Center, University of Chicago, Chicago, Ill. Available at: http://www.norc.uchicago.edu/new/gamb-fin.htm. Accessed 23 June 2003.

85. Shaffer HJ, Korn DA. Gambling and related mental disorders: a public health analysis. Annu Rev Public Health. 2002;23:171-212.

86. Volberg RA, Steadman HJ. Refining prevalence estimates of pathological gambling. Am J Psychiatry. 1988;145:502-505.

87. Welte JW, Barnes GM, Wieczorek WF, Midwell MC, Parker J. Gambling participation in the US—results from a national survey. J Gambl Stud. 2002;18:313-337.

88. Lesieur HR, Rosenhal RJ. Pathological gambling: a review of the literature. J Gambl Stud. 1991;7:5-39.

89. Custer RL, Milt H. When Luck Runs Out. New York, NY: Facts on File Publications; 1985.

90. Grant JS, Kim SW. Gender differences in pathological gamblers seeking medication treatment. Compr Psychiatry. 2002;43:56-62.

91. Rosenthal RJ. Pathological gambling. Psychiatr Ann. 1992;22:77-78.

92. Rogers RD, Owen AM, Middleton HC, et al. Choosing between small, likely rewards and large, unlikely rewards activates inferior and orbitofrontal prefrontal cortex. J Neurosci. 1999;19:9029-9038.

93. Hollander E, DeCaria CM, Mari E, et al. Short-term single-blind fluvoxamine treatment of pathological gambling. Am J Psychiatry. 1998;155:1781-1783.

94. Hollander E, DeCaria CM, Finkel J, Begaz T, Wong CM, Cartwright C. A randomized double-blind fluvoxamine/placebo crossover trial in pathological gambling. Biol Psychiatry. 2000;47:813-817.

95. Zimmerman M, Breen RB, Posternak MA. An open-label study of citalopram in the treatment of pathological gambling. J Clin Psychiatry. 2002;63:44-48.

96. Kim SW, Grant JE, Adson DE, Shin YC, Zaninelli E. A double-blind placebo-controlled study of the efficacy and safety of paroxetine in the treatment of pathological gambling. J Clin Psychiatry. 2002;63:501-507.

97. Pallanti S, Baldini Rossi N, Sood E, Hollander E. Nefazodone treatment of pathological gambling: a prospective open-label controlled trial. J Clin Psychiatry. 2002;63:1034-1039.

98. Moskowitz JA. Lithium and lady luck; use of lithium carbonate in compulsive gambling. N Y State J Med. 1980;80:785-788.

99. Haller R, Hinterhuber H. Treatment of pathological gambling with carbamazepine. Pharmacopsychiatry. 1994;27:129.

100. Pallanti S, Quercioli L, Sood E, Hollander E. Lithium and valproate treatment of pathological gambling: a randomized single-blind study. J Clin Psychiatry. 2002;63:559-564.

101. Kim SW, Grant JE, Adson DE, Shin YC. Double-blind naltrexone and placebo comparison study in the treatment of pathological gambling. Biol Psychiatry. 2001;49:914-921.

102. Brown RIF. The effectiveness of Gamblers Anonymous. In: Eadington WR, ed. The Gambling Studies Proceedings of the Sixth National Conference on Gambling and Risk Taking. Reno, Nev: Bureau of Business and Economic Research, University of Nevada; 1985.

103. Glen AM. The treatment of compulsive gamblers at the Cleveland VA Hospital, Brecksville Division. Paper presented at the 84th Annual convention of the American Psychological Association, Washington, DC, September 1976.

104. Taber JI. Group psychotherapy with pathological gamblers. Paper presented at the 5th National Conference on Gambling and Risk Taking, South Lake Tahoe, Nev, 1981.

105. Russo AM, Taber JI, McCormick RA, Ramirez LF. An outcome study of an in-patient treatment program for pathological gambling. Hosp Commun Psychiatry. 1984;35:823-827.

106. Taber JI, McCormick RA, Russo AM, Adkins BJ, Ramirez LF. Follow-up of pathological gamblers after treatment. Am J Psychiatry. 1987;144:757-761.

107. Hodgins DC, Currie SR, el-Guebay N. Motivational enhancement and self-help treatments for problem gambling. J Consult Clin Psychol. 2001;69:50-57.

108. Victor R, Cruz C. Paradoxical intention in the treatment of compulsive gambling. Am J Psychother. 1967;21:808-814.

109. Barker J, Miller M. Aversion therapy for compulsive gamblers. J Nerv Ment Dis. 1968;146:285-302.

110. Goorney AB. Treatment of a compulsive horse race gambler by aversion therapy. Br J Psychiatry. 1968;114:329-333.

111. Seager CP. Treatment of compulsive gamblers using electrical aversion. Med J Aust. 1972;1:742-745.

112. McCnaughey N, Blaszczynski A, Frankova A. Comparison of imaginal desensitisation with other behavioural treatments of pathological gambling. A 2- to 9-year follow-up. Br J Psychiatry. 1991;159:390-393.

113. Symes Bain, Nicki RM. A preliminary consideration of cue-exposure, response-prevention treatment for pathological gambling behavior: two case studies. J Gambl Stud. 1997;13:145-157.

114. Symes BA, Nicki RM. A preliminary consideration of cue-exposure, response-prevention treatment for pathological gambling behavior: two case studies. J Gambl Stud. 1997;13:145-157.

115. Sylver C, Ladouceur R, Boisvert JM. Cognitive and behavioral treatment of pathological gambling: a controlled study. J Consult Clin Psychol. 1997;65:727-732.

116. Ladouceur R, Sylver C, Letarte H, Giroux I, Jacques C. Cognitive treatment of pathological gamblers. Behav Res Ther. 1998;36:1111-1119.
