Review Paper

Obsessive–Compulsive Spectrum Disorders: A Review of the Evidence-Based Treatments

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Objective: To provide a review of the evidence-based treatments for obsessive–compulsive spectrum disorders (OCSD), a group of conditions related to obsessive–compulsive disorder (OCD) by phenomenological and etiological similarities, the morbidity of which is increasingly recognized.

Method: Literature relating to the following disorders: body dysmorphic disorder, hypochondriasis, trichotillomania, onychophagia, psychogenic excoriation, compulsive buying, kleptomania, and pathological gambling, and published between January 1965 and October 2007, was found using PubMed. Included in this review were 107 treatment reports.

Results: Serotonin reuptake inhibitors (SRIs) have shown benefits as first-line, short-term treatments for body dysmorphic disorder, hypochondriasis, onychophagia, and psychogenic excoriation, with some benefits in trichotillomania, pathological gambling, and compulsive buying. There are also suggested benefits for several atypical antipsychotics in disorders with a high degree of impulsivity, including trichotillomania and pathological gambling, and to a lesser extent, kleptomania and psychogenic excoriation. Cognitive-behavioural interventions have generally shown evidence for use as first-line treatment across the spectrum, with some variability in degree of benefit.

Conclusions: As in OCD, several conditions in the proposed OCSD benefit from SRIs and (or) cognitive-behavioural interventions. However, the treatment literature is generally limited, and more randomized controlled trials (RCTs) are needed to evaluate individual and combination treatments, for short-term use and as maintenance.

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Clinical Implications

- The OCSD are a proposed cluster of clinical psychiatric conditions with overlapping phenomenology and course.
- As with OCD, the 2 main treatment forms are SRIs and behavioural or cognitive-behavioural approaches, but treatment response varies with individual conditions.
- There is preliminary evidence for the benefit of novel agents, for example, atypical antipsychotics and naltrexone, for certain conditions in the spectrum.

Limitations

- Evidence is mostly from open-case series, with a limited number of RCTs.
- Comorbidities are often not considered in evaluating treatment benefits.

Key Words: *obsessive–compulsive disorder, obsessive–compulsive spectrum disorders, pharmacotherapy, psychotherapy, cognitive-behavioural therapy, serotonin reuptake inhibitors, atypical antipsychotics*

A common anxiety disorder, OCD is characterized by recurrent obsessions and (or) compulsions that are time consuming and cause marked distress and (or) functional impairment.[1] In recent years, attention has been paid to clinical psychiatric syndromes with phenomenological similarities to OCD. Often referred to collectively as OCSD (Figure 1)[2], they include impulse control disorders (pathological gambling, trichotillomania, compulsive buying, kleptomania, sexual compulsions, onychophagia, and psychogenic excoriation), somatoform disorders (body dysmorphic disorder and hypochondriasis), eating disorders (anorexia nervosa and binge eating), and even neurological or developmental disorders (Tourette syndrome, Sydenham chorea, Huntington disease, epilepsy, and autism).[2–4]

The OCSD and OCD often occur comorbidly.[5–13] There are also high rates of OCD and OCD symptoms in family members of probands with OCSD,[7,14–17] suggesting a common genetic predisposition.[2,3,16] Further, dysregulation of neurotransmitter function has been implicated in OCD and has also been reported in some of the OCSD,[18–27] several of which also appear to overlap with OCD in demographics, clinical course, and treatment response, supporting the notion of shared pathophysiology and vulnerability.[2–4,16]

The breadth of the spectrum, and which conditions should be included in it, remain contentious. Some argue that all neuropsychiatric conditions with a core pathology of compulsiveness or impulsiveness should be part of the spectrum.[2,3,18] Others point out the lack of established operational criteria for inclusion in the spectrum and the limited empirical data on the disorders,[4] and prefer to emphasize the differences between disorders at the compulsive and impulsive ends of the proposed spectrum.[28] The phenomenological link between some of the OCSD and OCD has also been questioned. For instance, OCD-related rituals cause distress and may lead to attempts at resistance, but compulsive gambling, shopping, and sex are usually experienced as pleasurable, and are resisted only due to secondary consequences.[29] To accommodate such differences, a dimensional model has been proposed.[30]

Abbreviations used in this article

5-HT	serotonin
CBT	cognitive-behavioural therapy
ECT	electroconvulsive therapy
GA	Gamblers Anonymous
OCD	obsessive–compulsive disorder
OCSD	obsessive–compulsive spectrum disorders
RCT	randomized controlled trial
SRI	serotonin reuptake inhibitor
SSRI	selective serotonin reuptake inhibitor

While many of the neuropsychiatric disorders mentioned earlier share significant similarities with OCD, some fit this conceptualization more readily, based on their phenomenology and effective treatments. This review will focus specifically on: somatoform disorders (hypochondriasis and body dysmorphic disorder), and impulse control disorders (trichotillomania, onychophagia, psychogenic excoriation, pathological gambling, compulsive buying, and kleptomania). Paraphilias and eating disorders are excluded owing to their distinctly different phenomenology and treatment from OCD.

The 2 most effective forms of treatment for OCD are SRIs and cognitive and (or) behavioural interventions, but there is also significant literature on other treatments that may be beneficial, albeit to a lesser extent. This paper will review the evidence-based literature on treatments for OCSD, with recommendations to the clinician.

Method

A search of the literature using PubMed was conducted for all articles related to the above conditions, and dated from January 1965 to October 2007. All RCTs were included in the review. Open label studies, chart reviews, and retrospective analyses were also included if the sample size were 10 or more. This resulted in a total of 107 reports. Study results were evaluated using the standard methodology for considering the strength of evidence for efficacy and tolerability (Table 1).[31] The main treatment literature for each condition is described below, and where available, effect sizes were provided. The standard definition of response used was a 25% improvement in symptoms from baseline.

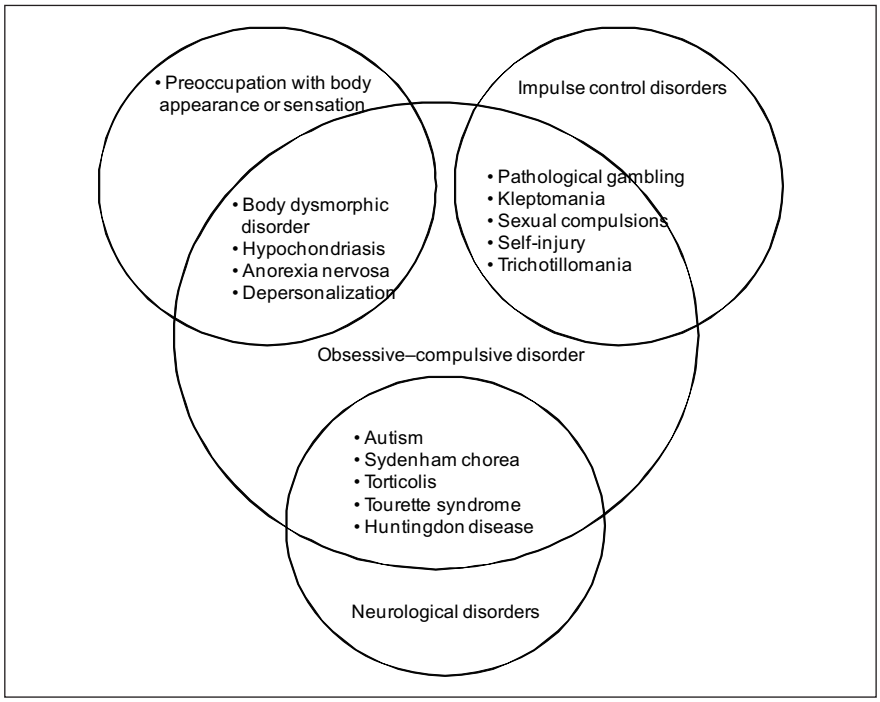
Results

Somatoform Disorders

Body Dysmorphic Disorder. Body dysmorphic disorder is characterized by a preoccupation with an imagined or overemphasized defect in appearance and an overestimation of the extent to which others notice the perceived defect.[1,32–34] Associated behaviours are repetitive and often ritualistic, such as mirror-checking and requests for reassurance. Marked distress and severe social and occupational impairment are common. The disorder affects up to 2% of nonclinical population samples,[35] and up to 12% of psychiatric outpatients.[8] Treatment is complicated by the fact that most patients seek treatment from plastic surgeons and dermatologists, with little positive effect, before seeing a psychiatrist.[36,37]

Serotonin Reuptake Inhibitors. Two case reviews and 2 retrospective analyses have noted that the SRIs fluoxetine and fluvoxamine produce greater improvement in body dysmorphic disorder patients than monoamine oxidase

Figure 1 The spectrum of obsessive–compulsive related disorders



Hollander E. Treatment of obsessive–compulsive spectrum disorders with SSRIs. Br J Psychiatry. 1998;173(Suppl 35):7–12.

Table I Levels of evidence[31]

Evidence criteria	
Level 1	Meta-analysis or replicated double-blind (DB), RCT that includes a placebo condition
Level 2	At least one DB-RCT with placebo or active comparison condition
Level 3	Prospective uncontrolled trial with 10 or more subjects
Level 4	Anecdotal reports or expert opinion
Treatment recommendations	
First line	Level 1 or Level 2 evidence plus clinical support for efficacy and safety
Second line	Level 3 evidence or higher plus clinical support for efficacy and safety
Third line	Level 4 evidence or higher plus clinical support for efficacy and safety
Not recommended	Level 1 or Level 2 evidence for lack of efficacy

inhibitors or tricyclic antidepressants (excluding clomipramine).[7,38,39,42] The efficacy of fluoxetine is also supported by a placebo-controlled RCT (Cohen's $d = 0.70$) and its follow-up study,[41,42] and further evidence to support fluvoxamine from 2 open trials.[43,44] As with OCD, an RCT found clomipramine significantly superior to desipramine in body dysmorphic disorder.[45] Open trials of escitalopram and citalopram also noted benefit.[46,47] Of note, response to SRIs is often partial, rather than complete, in body dysmorphic disorder, and 40% to 50% of patients may not respond adequately to SRIs alone.[35,45] Many patients often do not receive adequate trials of SRIs, contributing to suboptimal response.[48]

Cognitive-Behavioural Therapy. Behavioural interventions such as exposure and response prevention (that is, exposure to social situations, avoiding camouflage, and resisting compulsive behaviours such as mirror-checking and reassurance-seeking), and cognitive interventions such as cognitive restructuring (that is, focusing on their misperceptions of their appearance, rather than on the belief that others judge them solely on their physical appearance) have shown benefits in body dysmorphic disorder.[33,49] An RCT found individual exposure and response prevention significantly effective as both open treatment and in the randomized maintenance phase (compared with control subjects).[50] However, results

Table 2 RCTs: pharmacotherapy of body dysmorphic disorder, hypochondriasis, and trichotillomania

Study	Agents	Sample size, *n*	Duration, weeks	Dose range, mg daily	Results
Body dysmorphic disorder					
Phillips et al[41]	Fluoxetine, compared with placebo	74	13	20–80	Fluoxetine superior[a]
Hollander et al[45]	Clomipramine, compared with desipramine	29	16	Both 25–250	Clomipramine superior[a]
Hypochondriasis					
Greeven et al[54]	Paroxetine, compared with CBT, compared with placebo	112	16	10–60	CBT and paroxetine comparably effective and superior to placebo[a]
Trichotillomania					
van Minnen et al[69]	Fluoxetine, compared with behaviour therapy, compared with wait-list	43	12	20–60	BT superior to Fluoxetine and placebo[a]; placebo superior to fluoxetine[a]
Christenson et al[70]	Fluoxetine, compared with placebo	16	18	20–80	Fluoxetine and placebo equally ineffective
Streichenwein and Thornby[71]	Fluoxetine, compared with placebo	16	31	20–80	Fluoxetine and placebo equally ineffective
Dougherty et al[73]	Sertraline, compared with HRT, compared with sertraline and HRT	26	22	25–200	Combination treatment superior overall[a]
Swedo et al[75]	Clomipramine, compared with desipramine	13	12	n/a	Clomipramine superior[a]
Ninan et al[76]	Clomipramine, compared with CBT, compared with placebo	23	9	50–250	CBT superior to clomipramine and placebo[a]; clomipramine and placebo similar
Christenson et al[79]	Naltrexone, compared with placebo	17	6	50	No significant difference

[a] Statistically significant $P < 0.05$

BT = behavioural therapy; HRT = habit reversal training; n/a = not available

for CBT have been mixed. One RCT found individual CBT no different from wait-list,[37] but another found group CBT superior to no treatment.[51] It should be noted that 20% to 30% of body dysmorphic disorder patients do not respond to cognitive and (or) behavioural strategies alone,[37,51] and a retrospective chart review suggests that CBT–SRI combination treatment may elicit greater response than either treatment alone.[52]

Clinical Implications. SRIs have Level 2 evidence in body dysmorphic disorder, with adequate medication trials recommended to optimize benefit (Table 2). Level 2 evidence for individual behavioural therapy is also notable, though preliminary, but the mixed results thus far with CBT suggest that behavioural modification may be more pertinent than cognitive change. Large-sample RCTs would allow for more definitive conclusions.

Hypochondriasis. Hypochondriasis is a persistent fear or belief that one has a serious illness based on one's misinterpretation of bodily signs or symptoms.[1] This leads to hypervigilance to physical sensation, which helps to maintain the disorder. Even after a thorough medical evaluation that determines there is no illness, there is little sustained reduction in anxiety. Estimates of the prevalence of hypochondriasis in general medical practice range from 2% to 9%.[53]

Serotonin Reuptake Inhibitors. The only published RCT found paroxetine and individual CBT comparably effective and significantly superior to placebo ($d = 0.58$ CBT; $d = 0.53$ paroxetine).[54] Open trials have found fluoxetine and fluvoxamine effective,[53,55] though a longer time to response (at least 6 weeks) was also noted.[53]

Other Pharmacological Agents and Physical Methods of Treatment. While there are no RCTs of non-SRI agents, a retrospective study suggests that non-SSRI antidepressants may be useful in this disorder, and that ECT may also be beneficial, as 50% of the ECT sample showed good maintenance of gains.[56]

Cognitive-Behavioural Therapy. CBT for hypochondriasis aims to challenge and restructure faulty assumptions about physical symptoms, and to modify maladaptive patterns of behaviour that help to maintain those symptoms (for example, bodily checking or repeatedly seeking reassurance from a physician or a friend). A recent meta-analysis found cognitive or behavioural therapies significantly superior to wait-list and psychoeducation in improving hypochondriacal symptoms; CBT was equivalent to wait-list at posttreatment, but significantly superior at 1-year follow-up ($d = –1.26$ cognitive therapy; $d = –0.60$ behavioural therapy; $d = –2.40$ behavioural stress management; $d = –0.43$ CBT at follow-up).[57] In addition, 2 RCTs found individual CBT significantly superior to wait-list[58] and to medical care as usual.[59] A third RCT also found individual behavioural stress management or individual CBT significantly superior to wait-list; CBT was superior to behavioural stress management during active treatment but not at 12-month follow-up.[60] Yet another RCT found individual cognitive therapy or individual exposure and response prevention equally effective and significantly superior to wait-list.[61]

No controlled trials of group CBT have been reported. The only controlled trial to compare CBT with medication, cited previously, found individual CBT and paroxetine equally effective and significantly superior to placebo.[54]

Other Psychological Treatments. A chart review noted significant improvement in 64% of patients who received individual psychotherapy focusing on illness education and symptom perception.[62] An RCT found individual explanatory therapy significantly superior to wait-list, though residual symptoms were numerous and persistent.[63] Support for psychoeducational group therapy was offered by an open trial,[64] but a recent meta-analysis found no difference between wait-list and psychoeducation.[57]

Clinical Implications. Level 1 evidence for individual CBT and behavioural therapy in hypochondriasis makes these first-line treatment recommendations. Level 2 evidence for SRIs is preliminary but sound, and their benefit in comorbid conditions (for example, depression) and easy availability would support their use (Table 2). The delay in response to SRIs seen in one open study suggests longer administration.[53] The suggested benefits of non-SRIs, ECT, and other psychotherapies require confirmatory data from RCTs.

Impulse Control Disorders

Trichotillomania. Trichotillomania is the recurrent pulling of one's own hair that results in noticeable hair loss. Typically, there is increased stress immediately prior to hair pulling, or when attempting to resist the behaviour, and experience of pleasure, gratification, or relief when pulling the hair.[1] Depending on criteria used, prevalence rates for trichotillomania in the general population range from 0.6% to 3.4%.[65] However, among college-aged populations, other surveys have found hair-pulling rates of 10% to 13%.[66] Substantial subjective distress or impairment is often reported, and the condition is frequently chronic and unremitting.[67]

Serotonin Reuptake Inhibitors. An open trial noted the efficacy of fluoxetine,[68] but 3 RCTs have had negative results. One RCT found behavioural therapy and wait-list significantly superior to fluoxetine, with behavioural therapy the most effective treatment overall ($d = 3.80$ behavioural therapy; $d = 1.09$ wait-list; $d = 0.42$ fluoxetine),[69] and 2 crossover RCTs found no difference between fluoxetine and placebo, despite up to 3 months of treatment.[70,71] Among other SRIs, an open trial of fluvoxamine reported only partial efficacy, with significant improvement in some symptoms, but only limited reduction of actual hair pulling.[72] An RCT of sertraline, compared with placebo, or sertraline augmented with individual habit reversal therapy, found sertraline plus habit reversal therapy significantly more effective than either treatment alone.[73] In an open-label trial, escitalopram benefited 50% of patients.[74] A crossover RCT found clomipramine significantly superior to desipramine,[75] while another RCT found individual CBT significantly superior to clomipramine or placebo, with a nonsignificant trend favouring clomipramine over placebo.[76] Interestingly, a meta-analysis found clomipramine significantly more effective than SSRIs or placebo in treating trichotillomania, and no difference between SSRIs and placebo ($d = 0.68$ clomipramine; $d = 0.02$ SSRIs).[77]

Other Pharmacological Treatments. An open trial found olanzapine beneficial.[78] An RCT noted no difference between the opioid receptor antagonist, naltrexone, and placebo.[79] The anticonvulsant, topiramate, showed efficacy in an open trial, but side effects were significant.[80]

Cognitive-Behavioural Therapy. Controlled trials support the efficacy of behavioural therapy in treating trichotillomania, with the core techniques of habit reversal therapy (that is, self-monitoring, competing response, and thought-stopping) as the primary focus of treatment. An RCT of individual behavioural therapy found habit reversal therapy significantly superior to negative practice.[81] Another RCT found individual combination therapy (habit reversal therapy plus acceptance and commitment therapy) significantly superior to wait-list.[82] Further, a recent meta-analysis

found habit reversal therapy significantly superior to SRIs (d = 1.14 habit reversal therapy; d = 0.68 clomipramine; d = 0.02 SSRIs).[77] In the only RCT of group therapy, group behavioural therapy was significantly superior to group supportive therapy, but residual symptoms and relapse at follow-up were common.[83] A 2-year follow-up study of individual behavioural therapy also noted deterioration of treatment gains over time.[84]

Cognitive treatment strategies for trichotillomania have been described, but it is suggested that behavioural therapy combined with cognitive therapy (that is, CBT) may be more effective.[85,86] However, there is limited literature on CBT for trichotillomania. An open trial of individual CBT noted significant improvement and sustained gains.[87] An RCT, previously described in this section, has offered support for individual acceptance and commitment therapy, which involves mindfulness strategies and behavioural change, but it was applied concurrent with habit reversal therapy.[82]

The 3 reported comparative RCTs of cognitive and (or) behavioural interventions and medication in the treatment of trichotillomania were noted previously. One found individual behavioural therapy significantly superior to fluoxetine,[69] another found individual habit reversal therapy plus sertraline significantly superior to either treatment alone,[73] and a third found individual CBT significantly superior to clomipramine.[76]

Clinical Implications. Generally, SRIs have shown limited efficacy in trichotillomania, with the exception of clomipramine, which has Level 1 evidence of benefit (Table 2). Trials with other agents with multiple neurotransmitter targets may be useful. Preliminary Level 2 evidence for the benefit of atypical agents also needs further investigation. There is also Level 1 evidence for the first-line use of individual behavioural therapy (particularly habit reversal therapy), though maintenance of gains achieved during behavioural therapy appear to attenuate over time; thus behavioural therapy may also be useful as a maintenance treatment. Level 2 evidence for individual CBT is promising and it has good patient acceptance, supporting its first-line use. The contributions of cognitive techniques, compared with behavioural components, have not been fully evaluated.

Pathological Gambling. Pathological gambling is characterized by an uncontrollable urge or impulse to gamble that progressively increases in intensity.[1] It is associated with severe personal, social, and occupational problems, as well as a high rate of suicide attempts.[88–90] The prevalence of pathological gambling is 1% to 3% in the general population, with reports as high as 5.7% in adolescent populations.[91,92]

Serotonin Reuptake Inhibitors. Citalopram has shown efficacy in an open trial.[93] Escitalopram is supported by a discontinuation RCT, compared with placebo,[94] and by an open trial.[95] An RCT found paroxetine significantly superior to placebo,[96] but another RCT found a trend favouring CBT (alone or combined with paroxetine) over paroxetine alone, though the results did not reach significance.[97] An RCT of sertraline noted a high placebo response and no difference between sertraline and placebo.[98] Results for fluvoxamine are mixed. A single-blind crossover trial found fluvoxamine significantly superior to placebo,[99] as did a double-blind crossover RCT; a high placebo effect was noted in the first phase of the latter study but dissipated by end of treatment.[100] However, another RCT noted a persistent placebo response and no difference between fluvoxamine and placebo.[101]

Other Pharmacological Treatments. Lithium has shown efficacy in a placebo-controlled RCT,[102] and in a single-blind controlled trial in which lithium and valproate were found equally effective.[103] Evidence for naltrexone is more extensive. A retrospective chart review found naltrexone significantly superior to SSRIs,[104] and an open trial also found it effective.[105] RCTs found naltrexone significantly superior to placebo,[106] and as effective as bupropion.[107] An open trial also found bupropion beneficial,[108] but a recent RCT noted a high placebo response and no difference between bupropion and placebo.[109] An RCT found topiramate and fluvoxamine comparably effective, though topiramate was better tolerated.[110] An open trial noted the efficacy of nefazodone,[111] and a dose-ranging RCT found the opioid antagonist, nalmefene, significantly superior to placebo, with the lowest dose best tolerated.[112] A discontinuation RCT, compared with placebo, found N-acetyl cysteine significantly effective.[113]

Of note, a recent meta-analysis found antidepressants, opiate antagonists, and mood stabilizers equally effective and superior to placebo in improving pathological gambling symptoms (overall d = 0.78).[114]

Cognitive-Behavioural Therapy. Behavioural interventions have shown clear benefits in treating pathological gambling. An early RCT found imaginal desensitization more effective than aversion-relief therapy,[115] and the follow-up study noted significantly greater maintenance of gains with imaginal desensitization than with other behavioural treatments.[116] A comparative RCT of individual exposure and response prevention, group cognitive restructuring therapy, the combination, or wait-list, found individual exposure and response prevention significantly superior to the other treatments, with combination treatment no different from wait-list.[117] In a 2-phase study, individual exposure and response prevention was found significantly effective as open treatment, and in the randomized maintenance phase, individual or group relapse prevention therapy were comparably and significantly superior to control group at follow-up.[118]

Recent focus of psychotherapy research has been on CBT. Three RCTs found individual CBT significantly superior to wait-list.[119–121] An RCT also found group CBT significantly superior to wait-list.[122] However, comparisons of individual therapy and group therapy (CBT or forms of behavioural therapy), have tended to favour individual therapy. Though one RCT found group behavioural therapy as effective as individual behavioural therapy,[118] another RCT found individual behavioural therapy superior,[117] as did a recent RCT of individual CBT, compared with group CBT, that found both effective in reducing gambling behaviour, but individual CBT significantly superior in general psychological improvement and sustained gains at follow-up.[123] However, pathological gambling is a highly treatment-refractory condition and it should be noted that CBT for pathological gambling, though offering much potential, has also been associated with high attrition and relapse rates.[124,125]

Of note, in the only report comparing CBT with medication (previously described), there was a nonsignificant trend favouring CBT (alone or combined with paroxetine) over paroxetine alone.[97]

Other Psychological Treatments. Self-help programs, such as GA, appear to be the most popular intervention for pathological gambling. However, retrospective studies on GA report low success rates, drop-out rates as high as 70% to 90%, and minimal maintenance of gains.[126–128] Outcome studies indicate that combining individual and group psychotherapy and GA may improve outcomes,[129,130] and in this line, 2 RCTs found GA plus individual CBT significantly superior to GA alone.[131,132] However, the treatment resistance of pathological gambling and the high drop-out and relapse rates associated with this condition, even with CBT, are cautionary notes.[124,125]

Motivational therapy, a form of CBT which seeks to change patients' perspectives and behaviour by enhancing incentive for change, has been investigated as another treatment for pathological gambling and as a means of reducing treatment attrition. An RCT found individual motivational therapy plus self-help significantly more effective than self-help alone or wait-list.[133,134] A pilot nonrandomized study found individual combination therapy (CBT plus motivational therapy) produced significantly greater improvement and sustained gains than a naturalistic control group receiving treatment as usual.[135]

Clinical Implications. Among medications, the Level 2 benefits of naltrexone stand out, but there is mixed support for SRIs (Table 3). While addiction physicians are at ease with the use of naltrexone, others tend to prefer SSRIs, alone or in combination with atypical antipsychotics. The mixed findings for SSRIs have been attributed to subtypes of pathological gambling with specific treatment responses. Level 2 evidence for behavioural therapy or CBT is also strong, with individual therapy recommended over groups. The preliminary Level 2 results for motivational therapy are exciting as it may be more cost-effective and likely has better client preference over CBT or medications, but it needs further evaluation.

Compulsive Buying. Although there are no widely accepted operational diagnostic criteria for compulsive buying, one definition focuses on 2 components, shopping preoccupations and (or) behaviours.[136,137] Its prevalence is estimated to be between 2% and 8% in the general population.[138]

Serotonin Reuptake Inhibitors. The efficacy of citalopram is supported by an open trial[139] and by a discontinuation RCT, compared with placebo.[140] However, in a similar discontinuation RCT with escitalopram, efficacy during open treatment was followed by high relapse in both the placebo and escitalopram groups in the randomized discontinuation phase.[141] Results for fluvoxamine have also been mixed. While an open trial found fluvoxamine significantly effective,[142] 2 RCTs noted a high placebo response and no difference between fluvoxamine and placebo.[143,144]

Cognitive-Behavioural Therapy. The possible benefits of cognitive restructuring techniques to enable patients to develop more appropriate responses to their impulses has been suggested.[145] The only available report on CBT is of a nonrandomized study that noted the significant superiority of group CBT to wait-list control subjects.[146]

Clinical Implications. Lack of good RCTs preclude any definitive conclusions on treatment recommendations. The evidence for the benefit of SRIs is tenuous (Table 4) but anecdotal evidence suggests their advantage when compulsive buying is comorbid with depression or occurs as a result of it. The benefit of naltrexone and atypical agents in conditions such as pathological gambling would suggest benefits in compulsive buying, but this needs evaluation. Preliminary results for CBT also suggest potential benefits, but need confirmation.

Kleptomania. Kleptomania is characterized by the recurrent failure to resist impulses to steal items that are not needed for personal use or for their monetary value. Patients experience an increased sense of tension prior to the act and a sense of pleasure, relief, or gratification when committing theft.[1] Although the prevalence of kleptomania has been estimated at 6 per 1000, this may be an underestimation, as it is likely underreported.[17]

Serotonin Reuptake Inhibitors. The only report, a discontinuation RCT of escitalopram, had mixed results.[147] Efficacy during open treatment was followed by high relapse in both

Table 3 RCTs: pharmacotherapy of pathological gambling

Study	Agents	Sample size, *n*	Duration, weeks	Dose range, mg daily	Results
Grant and Potenza[94]	Escitalopram, compared with placebo	Open label:13 Randomized: 4	20	10–30	Escitalopram superior[a]
Kim et al[96]	Paroxetine, compared with placebo	45	8	20–60	Paroxetine superior[a]
Ravindran et al[97]	Paroxetine, compared with CBT, compared with CBT and paroxetine	34	16	10–40	No significant difference overall
Saiz-Ruiz et al[98]	Sertraline, compared with placebo	60	24	50–150	No significant difference
Hollander et al[99]	Fluvoxamine, compared with placebo	10	16	100–250	No significant difference
Hollander et al[100]	Fluvoxamine, compared with placebo	15	16	50–250	Fluvoxamine superior overall[a]
Blanco et al[101]	Fluvoxamine, compared with placebo	32	24	100–0	No significant difference
Hollander et al[102]	Lithium, compared with placebo	40	10	300–900	Lithium superior[a]
Pallanti et al[103]	Lithium, compared with valproate	42	14	600–1200; 600–1500	Both comparably effective
Kim et al[106]	Naltrexone, compared with placebo	45	12	25–250	Naltrexone superior[a]
Dannon et al[107]	Bupropion, compared with naltrexone	36	12	150–450; 25–150	Both comparably effective
Black et al[109]	Bupropion, compared with placebo	39	12	75–375	No significant difference
Dannon et al[110]	Topiramate, compared with fluvoxamine	31	12	Both 25–200	Both comparably effective overall
Grant et al[112]	Nalmefene, compared with placebo	207	16	25–100	Nalmefene superior[a]
Grant et al[113]	NAC, compared with placebo	Open label: 27 Randomized: 13	14	600–1800	Only open label NAC superior[a]

[a] Statistically significant $P < 0.05$

NAC = N-acetyl cysteine

the escitalopram and placebo groups in the randomized discontinuation phase.

Other Pharmacological Treatments. The only non-SRI of note is naltrexone. An open trial found naltrexone significantly effective,[148] and a chart review noted significant improvement and remission in three-quarters of naltrexone patients.[149]

CBT and Other Psychological Treatments. There are no published reports of controlled studies of cognitive or behavioural treatments, or other psychotherapies, in kleptomania.

Clinical Implications. There is preliminary Level 3 support for the second-line benefits of non-SRIs (Table 4), but the need for RCTs both SRIs and of non-SRIs, as well as of psychotherapeutic treatments, is strongly indicated. Anecdotally, some patients with depression (for example, the elderly) may present with shoplifting. Many of these patients are treated effectively with antidepressants. Thus a trial of SSRIs may be warranted in a subgroup of patients with kleptomania. For patients without depression, a trial of naltrexone may also be considered.

Onychophagia and Psychogenic Excoriation. Although not formally classified in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, onychophagia (chronic nail biting) and psychogenic excoriation (compulsive skin picking) are considered impulse control disorders, given their phenomenological similarities and significant comorbidity with OCD and OCSD.[12,150,151] The self-injurious behaviours are habitual, ritualistic, tension-reducing, and ego-dystonic.[152]

Table 4 RCTs: pharmacotherapy of compulsive buying, kleptomania, onychophagia, and psychogenic excoriation

Study	Agents	Sample size, *n*	Duration, weeks	Dose range, mg daily	Results
Compulsive Buying					
Koran et al[140]	Citalopram, compared with placebo	Open label: 24 Randomized: 15	16	10–60	Citalopram superior[a]
Koran et al[141]	Escitalopram, compared with placebo	Open label: 26 Randomized: 17	16	10–20	Escitalopram superior only as open label[a]
Black et al[143]	Fluvoxamine, compared with placebo	18	9	50–300	No significant difference
Ninan et al[144]	Fluvoxamine, compared with placebo	42	13	50–300	No significant difference
Kleptomania					
Koran et al[147]	Escitalopram, compared with placebo	Open label: 24 Randomized: 15	24	10–20	Escitalopram superior only in open label[a]
Onychophagia					
Leonard et al[153]	Clomipramine, compared with desipramine	14	12	Mean 120; mean 135	Clomipramine superior[a]
Psychogenic excoriation					
Simeon et al[154]	Fluoxetine, compared with placebo	21	10	20–80	Fluoxetine superior overall[a]
Bloch et al[155]	Fluoxetine vs placebo	Open label: 15 Randomized: 8	12	20–60	Fluoxetine superior[a]

[a] Statistically significant $P < 0.05$

Serotonin Reuptake Inhibitors. The only published report of SRIs in onychophagia is of a comparative RCT that found clomipramine significantly superior to desipramine.[153]

Most SRI evidence in psychogenic excoriation is for fluoxetine. Two RCTs found fluoxetine significantly superior to placebo.[154,155] Fluvoxamine was found to be effective in an open trial, but there were significant side effects.[156] Open trials have also noted the efficacy of sertraline[157] and escitalopram.[158]

Other Pharmacological Treatments. The literature is lacking on the use of non-SRI agents for onychophagia.

In psychogenic excoriation, an open trial found lamotrigine effective.[159]

Cognitive-Behavioural Therapy. In onychophagia, several forms of behavioural therapy have been investigated. An RCT found individual competing response therapy significantly superior to aversion therapy plus self-monitoring or self-monitoring alone; aversion therapy was also superior to self-monitoring alone.[160] However, a replicating RCT had reverse results, with aversion stimulus showing significant efficacy over competing response and self-monitoring alone; results for competing response did not reach significance.[161] Another RCT found individual habit reversal therapy significantly superior to the control group.[162] There are no reports on primarily cognitive strategies or CBT.

In psychogenic excoriation, the only reported controlled study is an RCT that found individual habit reversal therapy significantly superior to wait-list.[163]

Clinical Implications. In onychophagia, there is preliminary Level 2 support for first-line use of SRIs (Table 4) and strong Level 2 evidence for first-line use of behavioural therapy. Further RCTs with SRIs, as well as non-SRIs and CBT, would aid in uncovering other treatment strategies.

In psychogenic excoriation, there is good Level 2 evidence for SRIs, but Level 3 evidence for second-line use of non-SRIs is still tentative (Table 4). Preliminary Level 2 data on the first-line benefits of behavioural therapy suggest its potential value in clinical practice. More RCTs in all of these areas, as well as in CBT, would be highly useful.

Conclusions

In general, the treatment literature is limited in OCSD, especially with RCTs. Of specific treatments, CBT and (or) behavioural therapy appears to be beneficial across the

spectrum. As in OCD,[164] improved efficacy of CBT and (or) behavioural therapy in OCSD may be contingent on advances in the cognitive and behavioural modelling of the disorders. However, it is notable that in the few available efficacy studies comparing medication and psychotherapy, CBT and (or) behavioural therapy has shown comparable or superior efficacy to medication.

Response to specific classes of medication provides some support for the impulsivity–compulsivity dimensional model.[108] Thus disorders at the compulsive end respond better to SRIs and those at the impulsive end, including body dysmorphic disorder, hypochondriasis, onychophagia, and psychogenic excoriation, appear to benefit from a wider range of thymoleptics. For example, the opioid, naltrexone, and atypical agents have shown evidence as first-line agents in trichotillomania and pathological gambling, and to a lesser degree in kleptomania and psychogenic excoriation. However, the relatively few reported RCTs may limit the validity of the treatment data.

Augmentation with atypical antipsychotics is now the most frequently used therapeutic strategy for treatment of refractory OCD.[165,166] Patients with comorbid tic disorders seem to respond particularly well to this augmentation. Early evidence for the efficacy of these agents in trichotillomania, pathological gambling, kleptomania, and psychogenic excoriation further supports their therapeutic use both in OCD and in OCSD. Atypical antipsychotics appear to have multiple effects on several neurotransmitter systems. In addition to the prominent dopamine D_2 antagonism, they have been shown to upregulate postsynaptic 5-HT_{1A} receptors, downregulate 5-HT_{2A} receptors, and as well, have 5-HT transporter blockade mechanisms, which are all proposed mechanisms of action of antidepressant and antiobsessive agents. Preclinical studies also report effects on several other neurotransmitter targets, including neurotensin, glutamate receptors, and brain-derived neurotrophic factor action similar to SSRIs. Elevation of c-Fos in limbic areas is another effect common to both classes of agents. Atypical agents probably offer a therapeutic strategy likely to benefit several of the OCSD conditions.

Superior efficacy of clomipramine (compared with SSRIs), especially in trichotillomania, has been attributed to its reuptake inhibition of several neurotransmitters, including dopamine. Unfortunately, there are few published RCTs of clomipramine in other OCSD conditions (other than body dysmorphic disorder and onychophagia). Similarly, the benefit of novel agents including selective norepinephrine reuptake inhibitors, such as duloxetine or venlafaxine, or the noradrenergic and specific serotonergic antidepressant, mirtazapine, has not been explored. With broader spectrum of effect and superior tolerability, these agents have been shown to be effective in a spectrum of anxiety disorders, and may benefit at least a subgroup of patients with OCSD.

Overall, OCSD remains a significant treatment challenge for practising clinicians, as many patients with severe forms of these disorders do not respond well to currently available treatments, with the illness often following a chronic, recurrent course. While well-designed RCTs of available treatment forms are a priority, further exploration of etiological factors would be equally relevant to develop novel therapeutic agents. Basic research employing genetic and neuroimaging strategies may be particularly useful in this respect.

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Résumé : Les troubles du spectre obsessionnel-compulsif : une revue des traitements fondés sur des données probantes

Objectif : Présenter une revue des traitements fondés sur des données probantes des troubles du spectre obsessionnel-compulsif (TSOC), un groupe d'affections liées au trouble obsessionnel-compulsif (TOC) par des similitudes phénoménologiques et étiologiques, dont la morbidité est de plus en plus reconnue.

Méthode : La documentation sur les troubles suivants : peur d'une dysmorphie corporelle, hypocondrie, trichotillomanie, onychophagie, excoriation psychogène, achat compulsif, kleptomanie, et jeu pathologique, publiée entre janvier 1965 et octobre 2007, a été trouvée à l'aide de PubMed. Cent sept rapports de traitements sont inclus dans la revue.

Résultats : Les inhibiteurs du recaptage de la sérotonine (IRS) ont démontré des avantages comme traitement de base à court terme pour la peur d'une dysmorphie corporelle, l'hypocondrie, l'onychophagie, et l'excoriation psychogène, et certains avantages pour la trichotillomanie, le jeu pathologique, et l'achat compulsif. Il y a également des avantages suggérés pour plusieurs antipsychotiques atypiques dans des troubles comportant un degré élevé d'impulsivité, dont la trichotillomanie et le jeu pathologique, et dans une moindre mesure, la kleptomanie et l'excoriation psychogène. Les interventions cognitivo-comportementales se sont généralement révélées probantes lorsqu'utilisées comme traitement de base pour tout le spectre, avec une variation du degré d'utilité.

Conclusions : Parallèlement au TOC, certaines affections du spectre OC proposé bénéficient des IRS et (ou) des interventions cognitivo-comportementales. Cependant, la documentation sur les traitements est généralement limitée, et il faut plus d'essais randomisés contrôlés (ERC) afin d'évaluer les traitements individuels et combinés, pour l'usage à court terme et d'entretien.