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Pharmacological Treatments of Impulse Control Disorders

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Abstract

Impulse control disorders (ICDs) are commonly defined as the failure to resist an impulse, drive or temptation to perform harmful acts. In the Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision (DSM-IV-TR) the following disorders are included in the section 'Impulse control disorders not elsewhere classified': intermittent explosive disorder, pyromania, kleptomania, trichotillomania and pathological gambling. Common to all these disorders are repeated failures to resist impulses to perform harmful acts. This chapter gives a brief outline of clinical characteristic and pharmacological treatments of each of these disorders. Pharmacological trials with selective serotonin reuptake inhibitors, norepinephrine reuptake inhibitors, opioid antagonists, mood stabilizers and anti-epileptics have demonstrated potential effectiveness of pharmacological treatments for ICDs. However, several studies have limitations such as poor designs, relying on single cases, small sample sizes and lack of adequate control groups. More controlled studies in this area are needed to establish the effectiveness of pharmacological treatments for different ICDs.

Key Words

pharmacological treatment; impulse control disorders; intermittent explosive disorder; pyromania; kleptomania; trichotillomania; pathological gambling

15.1 Impulse control disorders

In the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV), five separate disorders are outlined in the section 'Impulse control disorders not

Clinical Trials in Psychopharmacology: A Better Brain, Second Edition Edited by M. Hertzman and L. Adler
 ISBN: 978-0-470-74076-7

elsewhere classified': intermittent explosive disorder (IED), pyromania, kleptomania, pathological gambling (PG) and trichotillomania (TTM). In the section 'Impulse control disorders not otherwise specified', skin picking, compulsive shopping and compulsive sexual behavior are included.

The essential feature of ICDs is the failure to resist an impulse, drive or temptation to perform an act that is harmful to the person him/herself or to others. Common in ICDs is that the individual feels an increasing tension or arousal before engaging in the behavior, and experiences pleasure, gratification or relief when committing the act. After the act is carried out, there may or may not be regret, self-reproach or guilt [1].

Many of the underlying behaviors in ICDs, such as gambling and shoplifting, are common in the general populations. However, only a small portion of the individuals performing these behaviors do it as a response to irresistible impulses or urges and can be classified as an ICD. In addition to the similarity of symptoms for these disorders, they also have similar ages at onset and courses [2]. However, the sex ratio seems to differ among the different disorders [1, 2]. With the exception of PG, all of these disorders are considered to be rare [2], and little research exists concerning epidemiology and treatment efficacy. Studies of phenomenology have shown that ICDs may be related to mood disorders, anxiety disorders and substance use disorders [2]. Although ICDs can often be significant disabling, these disorders often go undiagnosed and untreated [3].

Neurobiology of impulse control disorders

Apart from the possible phenomenological relationship with other disorders, the nature of the core symptoms of ICDs allows us to make assumptions about the underlying neurobiology. Serotonin, which is a central neurotransmitter in behavior initiation/cessation, has been suggested to be involved in the failure to resist impulses which is the core criteria of ICDs as serotonergic dysfunction is often related to impulsivity [4]. Norepinephrine is involved in arousal and excitement, and might be central in the increasing arousal experienced before committing the impulsive behaviors [4]. Dopamine is a neurotransmitter central in the reward and reinforcement systems in the Ventral Tegmental Area and the Nucleus Accumbens, and it has been hypothesized that this reinforcement system is likely to be involved in the rewarding feeling of pleasure when committing the act [4]. In addition, opioids are usually involved in feelings of pleasure and urges, and it has also been suggested that this substance is involved in the rewarding, pleasurable experience when committing the act. Due to the reinforcing properties, opioids have also been assumed to contribute to the urges experienced before committing the certain behavior. Since all the neurotransmitters and neurotransmitter systems mentioned here have been assumed to play a central role in ICDs, they have in clinical practice and in clinical studies been targets for pharmacological interventions of ICDs.

Most studies of the neurobiology of ICDs have focused on the hypothalamic-pituitary-adrenal axis, on serotonin, norepinephrine, glucose metabolism and on EEG data. The most consistent findings have suggested abnormalities, particularly in the serotonergic and, to a lesser extent, in the noradrenergic systems [2].

Although Kim [5] suggests that symptoms of ICDs are generally refractory to psychotherapeutic or pharmacologic treatments, several pharmacological treatments have recently proven effective in the treatment of such disorders. Still, few controlled trials have been conducted and no empirically validated pharmacological treatments exist for these disorders. As little research investigating the efficacy of treatments for ICDs have been conducted, our understanding of efficacious and well-tolerated pharmacotherapies for ICDs lags behind those for other major neuropsychiatric disorders [3]. PG is the most common ICDs, and has received the most clinical and research attention. This chapter will give a brief outline of the clinical characteristics of ICDs, and present empirical research on pharmacological treatments for each of these.

15.2 Pathological gambling

Gambling can be defined as an activity that involves an attempt to win money by staking money on uncertain events [6]. For most people, gambling is a leisure activity without negative consequences. However, others develop excessive gambling behavior which has severe negative consequences for the gambler and his or her relationships with family members, friends or colleagues. According to DSM-IV-TR, the essential feature of PG is '*persistent and recurrent maladaptive gambling behavior that disrupts personal, family or vocational pursuits*' which is not better accounted for by a manic episode [1]. The lifetime prevalence of PG is between 1 and 3% in the adult population of North America. The prevalence is even higher for adolescents [7–9]. The prevalence of gambling is assumed to be on the rise due to expanding gambling opportunities and the general social approval of the gambling industry [10, 11]. Consequently, the need for effective treatment seems to be self-evident. Most of the treatments of PG have been conducted within the behavioral, cognitive and cognitive-behavioral spectrum [12]. Recently, however, several studies have been conducted investigating different pharmacological approaches to the treatment of PG.

Pharmacological treatments of pathological gambling

Evidence suggests the involvement of both serotonergic, noradrenergic, dopaminergic and opioidergic systems in the etiology of PG [13], and pharmacological treatments targeting these neurotransmitter systems have shown promising results in the early stages of understanding and treating PG [14]. These systems are related to the mechanisms that underlie behavioral disinhibition (serotonergic system), reward mechanisms (dopaminergic and opioidergic system) and arousal (noradrenergic system) associated with impulse control and addictive disorders [15]. Although PG is classified as an impulse control disorder, it has also been described as an obsessive-compulsive spectrum disorder within the impulsive cluster [16]. Potenza *et al*. conducted a functional magnetic resonance imaging (fMRI) study of gambling urges in pathological gamblers [17] and found that PG has neural features more similar to other ICDs and distinct from those of obsessive-compulsive disorders.

Hollander *et al*. [14] outline several psychopathological domains within PG which could conceivably be targeted for treatment: impulsive symptoms (arousal), compulsive symptoms (anxiety reduction) and addictive symptoms (symptoms of withdrawal). Pharmacological treatments of PG have usually involved the administration of either opioid antagonists, antidepressants or mood stabilizers [14]. Opioid antagonists block the effects of endogenous endorphins on central opiate receptors and inhibit dopamine release in the nucleus accumbens, involving reward, pleasure and urge mechanisms [14]. Several studies have also specifically indicated that pathological gamblers may be characterized by serotonergic dysfunction [18]. Most of the antidepressants drugs used in the treatment of PG are selective serotonin reuptake inhibitors (SSRIs) which appear to have anti-compulsive and anti-impulsive effects [14]. Mood stabilizers have proven effective in treating mania, and recent studies have also demonstrated effectiveness in treating other impulsive disorders such as borderline personality disorder, disruptive behavior and TTM [14]. It has been suggested that impulse control disorders and bipolar spectrum disorders may be related, and the impulsivity in PG seems to resemble that of bipolar disorder. The co-morbidity between bipolar disorder and PG has been estimated to be as high as 30% [19]. Mood stabilizers are assumed to have anti-impulsive effects [20], and hence are assumed to potentially be effective in the treatment of PG.

A recent meta-analysis of clinical trials using pharmacological interventions to treat PG identified 130 potential studies, but only 16 studies met the criteria for inclusion in the meta-analysis: (i) the target problem was PG, (ii) the treatment was pharmacological, (iii) the study was written in English and (iv) the study reported outcomes particularly pertaining to gambling [21]. A total of 597 subjects were included in the outcome analyses of these studies. Table 15.1 gives an overview of the included studies [19, 20, 22–35]. The analyses showed that at post-treatment the pharmacological interventions were more effective than no treatment/placebo, yielding an overall effect size (ES) of 0.78 (95% CI = 0.64, 0.92). A multiple regression analysis showed that the magnitude of ESs at post-treatment was lower in studies using a placebo-controlled condition compared to studies using pre-post design (without any control condition). No differences between the three main classes of pharmacological interventions (antidepressants, opiate antagonists and mood stabilizers) were detected.

15.3 Trichotillomania

TTM is defined as hair loss due to a patient's irresistible urge to pull out his/her hair [1]. The sites of hair pulling may include any part of the body on which hair grows, but the most common sites are the scalp, eyebrows and eyelashes. Hair pulling usually occurs in states of relaxation and distraction (e.g. when reading a book), but may also occur under stressful circumstances. Usually increased tension is present immediately before hair pulling, and the act is followed by gratification, pleasure or a sense of relief. According to the DSM-IV, the disturbance must cause significant distress or impairment in either social, occupational or other important

Table 15.1 Studies examining the effectiveness of pharmacological treatments for pathological gambling [21].

Study	Trial	Design	Mean dose at endpoint (mg/d)	Duration (wk)	N[a]	Mean age	Attrition[b] (%)	Proportion males (%)	Formal diagnosis	Treatment response
Black [22]	Bupropion vs. placebo	Open-label	400	8	10	44.6	0.0	40.0	Yes	70% partial/ complete remission
Blanco *et al.* [23]	Fluvoxamine vs. placebo	Double-blind	200	24	32	42.1	59.4	65.6	Yes	73% complete remission
Dannon *et al.* [24]	Topiramate vs. fluvoxamine	Single-blind	200 and 200, respectively	12	20	34.9	35.5	100.0	Yes	Topiramate and fluvoxamine: 75% complete remission 25% partial remission
Dannon *et al.* [25]	Bupropion vs. naltrexone	Single-blind	424 and 116, respectively	12	25	29.1	30.6	100.0	Yes	Bupropion: 75% complete remission 25% partial remission Naltrexone: 23% partial remission
Grant *et al.* [26]	Paroxetine vs placebo	Double-blind	20–60	16	71	45.4	44.7	60.5	Yes	59% partial/ complete remission

(continued overleaf)

Table 15.1 *(continued)*

Study	Trial	Design	Mean dose at endpoint (mg/d)	Duration (wk)	N[a]	Mean age	Attrition[b] (%)	Proportion males (%)	Formal diagnosis	Treatment response
Grant *et al.* [27]	Nalmefene (25, 50 and 100 mg) vs. placebo	Double-blind	25, 50 and 100, respectively	16	146	46.0	64.7	56.5	Yes	59% partial/ complete remission
Grant and Potenza [28]	Escitalopram vs. pre-treatment	Open-label	25.4	11	13	55.8	30.8	53.8	Yes[c]	62% partial/ complete remission
Hollander *et al.* [19]	Fluvoxamine vs. placebo	Double-blind	195	8 + 8 (crossover)	15	38.9	23.1	100.0	Yes	70% partial/ complete remission
Hollander *et al.* [29]	Lithium-carbonate vs. placebo	Double-blind	1150	10	29	44.5	27.5	58.6	Yes[d]	83% partial/ complete remission
Kim and Grant [30]	Naltrexone vs. pre-treatment	Open-label	157	6	17	44.6	17.6	41.2	Yes	Most patients improved
Kim *et al.* [31]	Naltrexone vs. placebo	Double-blind	187.5	11	45	48.6	19.6	33.3	Yes	75% partial/ complete remission
Kim *et al.* [32]	Paroxetine vs. placebo	Double-blind	51.7	8	45	49.3	8.9	33.3	Yes	48% complete remission 13% partial remission

Pallanti *et al.* [33]	Nefazodone vs. pre-treatment	Open-label	345.8	8	12	48.5	14.3	71.4	Yes	25% complete remission 50% partial remission
Pallanti *et al.* [20]	Lithium carbonate and Valproate vs. pre-treatment	Single blind	1200 and 1500, respectively	14	42	31.6	26.2	76.%	Yes	Lithium carbonate: 61% partial/ complete remission Valproate: 68% partial/ complete remission
Sáiz-Ruiz *et al.* [34]	Sertraline vs. placebo	Double-blind	95.0	24	60	38.9	38.3e	90.0	Yes	74% partial/ complete remission
Zimmerman *et al.* [35]	Citalopram vs. pre-treatment	Open-label	34.7	12	15	44.1	40.0	60.0	Yes	33% complete remission 54% partial remission

[a]The number for which the data analyses are based.
[b]Discontinued medication or withdrew from the study after randomization.
[c]In addition to the DSM-criteria for pathological gambling the patients also fulfilled the criteria for at least one anxiety disorder.
[d]In addition to the DSM-criteria for pathological gambling the patients also fulfilled the criteria for a bipolar spectrum disorder.

areas of functioning [36]. Although TTM has been sparsely studied and may be under-diagnosed, the lifetime prevalence is estimated to be 0.6–3.6% [37].

Pharmacological treatments of trichotillomania

The most common treatments for TTM are habit-reversal therapy (HRT), pharmacotherapy with fluoxetine or sertraline (SSRI) and pharmacotherapy with clomipramine (tricyclic antidepressant). A recent systematic review [38] compared the efficacy of behavioral treatment (HRT) and pharmacotherapy with either SSRI or clomipramine. Seven studies [39–45] met the criteria for inclusion in the study: (i) randomized clinical trial with control group or comparison group with active treatment, (ii) blinded assessment of the clinical outcomes, (iii) primary diagnosis of TTM and (iv) comparison of HRT, SSRI and clomipramine to each other or to a control condition. A total of 157 patients were included in the overall analysis in the systematic review (see Table 15.2). Six different outcomes were examined: (i) SSRI vs. control condition, (ii) clomipramine vs. control condition, (iii) HRT vs. control condition, (iv) HRT vs. SSRI, (v) HRT vs. clomipramine and (vi) clomipramine vs. SSRI.

A total of 72 completers from four different studies investigating the effect of SSRI vs. a control condition contributed to the first outcome. None of the four studies reported significant differences between SSRI and control conditions, and in the overall meta-analysis no significant differences between SSRI and the control conditions were found either ($z = 0.09$, $p = 0.93$). The overall estimated ES was 0.02 (95% CI = −0.32, 0.35).

Two studies investigated the effects of clomipramine. They comprised 24 completers, demonstrating a significant treatment effect favoring clomipramine when compared to control conditions (ES = −0.68, 95% CI = −1.28, −0.07). HRT was compared to control conditions in three trials, involving a total of 59 completers contributing to the analysis. The overall meta-analysis demonstrated beneficial effects of HRT compared to the control conditions (ES = −1.14, 95% CI = −1.89, −0.38) [38]. Two of these studies demonstrated a significant effect of HRT compared to the wait-list/placebo control condition [41, 44]. Only one study, Minnen *et al.* [45] compared HRT (n = 14) with SSRI (n = 11). There was no statistical significant difference between these two. However, there was a tendency toward a better effect of HRT compared to SSRI (ES = −0.73, 95% CI = −1.60, 0.14). One study [41] comparing the effects of HRT (n = 5) and clomipramine (n = 6) found a significant difference in favor of HRT (ES = −1.74, 95% CI = −3.23, −0.25). No blinded studies directly comparing the effects of clomipramine with SSRI were included in the meta-analytic review [38].

The results from the systematic review showed that HRT was the most effective treatment for TTM when practiced in this particular setting (by experienced clinicians in academic research settings). HRT demonstrated the largest ESs of these interventions. Compared to the most prevalent pharmacological treatments for TTM, clomipramine and SSRI, HRT demonstrated superiority. Clomipramine also demonstrated efficacy for TTM when compared to placebo or active control condition, while SSRI did not prove effective when compared to control conditions. This

Table 15.2 Studies examining the effectiveness of pharmacological treatments for trichotillomania.

Study	Trial	Design	Dose per day at endpoint	Duration (wk)	N[a]	Mean age	Attrition (%)	Proportion females (%)	Treatment response
Swedo *et al.* [43]	Clomipramine vs. placebo	Crossover trial, double-blind	Mean 180.8 mg Max 250 mg	10	13	31.6	0	100.0	23% complete remission 69% partial remission
Christenson *et al.* [39]	Fluoxetine vs. placebo	Crossover trial, double-blind	Mean 77.5 mg Max 80 mg	18	16	31.6	23.8	93.8	No response
Strichenwein and Thornby [40]	Fluoxetine vs. placebo	Crossover trial, double-blind	Mean 78.8 mg Max 80 mg	12	16	39.0	23.8	87.5	No response
Ninan *et al.* [41]	Clomipramine vs. palcebo HRT vs. waitlist control HRT vs. clomipramine	Randomized, parallel group trial, blinded assessment of outcome	Mean 116.7 mg Max 250 mg	9	16	33.4	30.4	81.3	HRT: 80% complete remission 20% partial remission Clomipramine: 67% partial remission
Van Minnen *et al.* [45]	HRT vs. waitlist controlHRT vs. fluoxetine	Randomized, parallel group trial, blinded assessment of outcome	Mean 60 mg Max 60 mg	12	40	31.3	7.0	95.0	HRT: 64% remission Fluoxetine: No response

(continued overleaf)

Table 15.2 (*continued*)

Study	Trial	Design	Dose per day at endpoint	Duration (wk)	N[a]	Mean age	Attrition (%)	Proportion females (%)	Treatment response
Dougherty *et al.* [42]	Sertraline vs. HRT Sertraline and HRT	Randomized, parallel group, Double-blind	Max 200 mg	22	24	28.7	16.2	95.8	Single modality: 15.4% remission Dual modality: 54.5% remission
Woods *et al.* [44]	HRT vs. waitlist control	Randomized parallel group trial, blinded assessment of outcome	–	12	25	33.4	10.7	92.0	66% remission

[a]The number on which the data analyses are based.

finding is not in line with earlier reviews where SSRIs are recommended as the preferred pharmacological treatment for TTM [46].

However, methodological limitations of the trials included in this systematic review may have influenced these conclusions [38]. Firstly, no single clinical rating scale was used consistently in the included studies to assess severity and improvement of TTM symptoms, and it is possible that the different rating scales used have different sensitivity to detect changes in TTM severity. Secondly, most of the studies did not report the number of subjects with co-morbid Obsessive Compulsive Disorder (OCD), which quite frequently co-occur with TTM. Subjects with co-morbid OCD would probably respond to SSRI and clomipramine since these are both first-line treatment for OCD. Thirdly, all of the parallel-group trials included trial completers only in the analyses, which may have affected the results. Future studies should investigate whether HRT can demonstrate efficacy against more rigorous control conditions accounting for the non-specific effects of therapy, and determine if HRT is effective in treating TTM beyond the few sites where it is currently practiced [38].

15.4 Kleptomania

Kleptomania is characterized by recurrent failure to resist impulses to steal items even though the items are not needed for personal use or for their monetary value [1]. Increased sense of tension is usually experienced before the theft, and pleasure, gratification or relief when committing the theft. Many report guilt, remorse and depression afterwards. The stolen objects are often affordable and of little value to the individual and are often given away, discarded or secretly returned afterwards [1].

The disorder is disabling and often goes undiagnosed in clinical practice [47]. So far, no prevalence studies in general populations have been conducted. Hence the prevalence in these populations is unknown [48]. However, several studies of clinical samples suggest that the disorder is not uncommon. A recent study of psychiatric inpatients ($n = 204$) with multiple disorders found prevalence rates of 7.8 and 9.3% for current and lifetime diagnosis of kleptomania, respectively [49]. The fact that the current and lifetime prevalence rates are almost identical suggests that the condition is chronic if not treated [47]. One study of 107 patients diagnosed with depression found a prevalence rate of kleptomania of 3.7% [50] and, in a study of patients with substance abuse ($n = 79$), a prevalence of 3.8% was found [51]. Two studies of patients diagnosed with PG found that 2.1 and 5%, respectively, also met the criteria for kleptomania [52, 53]. The condition appears, however, to occur in less than 5% of shoplifters. Evidence suggests that approximately two-thirds of the individuals with kleptomania in clinical samples are female [1]. The onset of kleptomania usually occurs during adolescence, although early childhood onset and late adulthood onset have been reported [1].

Kleptomania was originally classified within the OCDs spectrum. However, recent evidence – such as clinical characteristics, familial transmission and treatment response – suggests that it has important similarities with addictive disorders and mood disorders. Kleptomania has also been shown to frequently co-occur with substance abuse [47].

Pharmacological treatments of kleptomania

The etiology of kleptomania is unclear, and little evidence concerning possible neurobiological correlates of the disorder exists [47]. It has been hypothesized that dysfunctions in the serotonergic system in ventromedial prefrontal cortex contribute to poor decision-making characteristics of individuals with kleptomania [54]. Evidence also suggests a non-specific serotonergic dysfunction as lower levels of platelet 5-hydroxytryptamine (5-HT) transporters (evaluated by means of binding of 3H-paroxetine) have been found in kleptomaniacs ($n = 20$) as opposed to healthy controls [55]. There have also been reports of kleptomania occurring after damage to the orbitofrontal–subcortical circuits of the brain [56]. Neuroimaging techniques have shown significantly decreased white matter integrity in inferior frontal regions of kleptomaniacs compared to controls [57]. This supports the hypothesis that kleptomaniacs may not be able to control and resist impulses to steal [48].

Antidepressants, mainly SSRIs, have been considered the treatment of choice for kleptomania as for other ICDs [58]. However, evidence from case reports of responses to serotonergic medication in kleptomania have shown inconsistent results [47]. In one study using open-label escitalopram in the treatment of kleptomaniacs ($n = 20$), 79% reported improvement in stealing behavior. The responders were randomized to continue medication or receiving placebo. After the double-blind phase, 43% of those receiving medication and 50% in the placebo group no longer remained abstinent. There was no statistical difference in the treatment effect between the escitalopram and the placebo condition [59]. Still, it has been argued that there may exist patients suffering from a subtype of kleptomania sharing common features with OCD who may respond well to SSRIs [47]. Grant [47] suggests that kleptomaniac behaviors may be far more heterogeneous than initially thought, and that antidepressants or mood stabilizers may be beneficial for those kleptomania subjects with significant mood symptoms who may shoplift due to subsyndromal mania or depression. However, more well-controlled studies in this area are needed.

According to Grant [47], emerging evidence suggests that SSRIs may lack effectiveness in treating kleptomania but that lithium, anti-epileptic and opioid antagonist seem to show promising results. No controlled studies of mood stabilizers or anti-epileptic medications in the treatment of kleptomania have been published, but case reports of lithium, valproate and topiramate have shown that these medications may be effective [47]. A case series of three patients treated for kleptomania showed that treatment with topiramate was effective [58]. The biological mechanism of this effect is unknown, but is hypothesized to be related to the disinhibition of GABA input in the nucleus accumbens area, targeting the arachidonic acid cascade [58]. Studies with controlled designs are needed to confirm these preliminary findings. Lithium alone, or in combination with fluoxetine, has been associated with improvement in kleptomania in several reports [2, 58, 60]. However, some case studies of lithium as monotherapy or lithium augmentation have shown no effects in the treatment of kleptomania [47].

The efficacy of opioid antagonist in kleptomania has recently been examined because of the possible relationship and similarities to addictive disorders. The urge or craving state that people with kleptomania experience before engaging in the

problematic behavior, and the hedonic experiences during the behavior, much resemble that of addictive behaviors. Opioid antagonists are assumed to work indirectly on dopamine reducing the subjective experience of reward and urges seen in kleptomania [61]. In one open-label study with naltrexone (n = 10) for 12 weeks (mean effective dose was 145 mg/day), 80% reported significant reduction in urges to steal and 20% reported complete remission of the symptoms [61]. A longitudinal study of naltrexone as monotreatment for kleptomania (n = 17) found that 76.5% had reduction in the urges to steal and 41.1% ceased to steal [62] at the most recent follow-up, where the mean duration of follow-up was 481.9 ± 280.9 days after baseline. Table 15.3 summarizes controlled studies of pharmacological treatments of kleptomania [59, 61, 62].

15.5 Pyromania

The essential feature of pyromania is repeated episodes of deliberate and purposeful fire setting, where the patient experiences tension or affective arousal before committing the act. There is also a fascination with, interest in, curiosity about or attraction to fire and its situational contexts. The patient usually experiences pleasure, gratification or release of tension when setting the fire and witnessing its effects and participating in its aftermath [1]. Research on pyromania has mainly focused on the criminal population. Although pyromania is considered to be a rare disorder [1], a recent study of psychiatric inpatients (n = 204) revealed that 3.4% met the DSM-IV criteria for current pyromania, whereas the lifetime prevalence was 5.9% [49]. Fire setting during adolescence does not necessarily reflect symptoms of pyromania, but may be a symptom of various psychiatric disorders [63]. A recent study of adolescent psychiatric inpatients (n = 102) found that, after excluding patients who set fire due to other disorders such as conduct disorder, bipolar disorder, psychotic disorders, substance use disorders and developmental disorders, seven patients met the criteria for current pyromania [64].

Pharmacological treatments of pyromania

To our knowledge, only one relatively large study of pharmacological treatment of pyromania has yet been published. The study recruited 14 adults and 7 adolescents with lifetime DSM-IV pyromania from inpatient and outpatient studies of impulse control disorders. Of the 21 subjects, 14 had previously received treatment for psychiatric disorders, and only two had received treatment specific for pyromania. All 14 had received psychotropic medication, but only two had received medication specifically prescribed for pyromania symptoms. Partial or complete remission of pyromania urges and behavior were reported in 6 of the 14 cases (see Table 15.4). The medications used included topiramate, escitalopram, sertraline, fluoxetine and lithium. In three of the cases, pyromania symptoms recurred when the medication was discontinued. In the cases not responding to psychopharmacology, different medication had been tried: fluoxetine, valproic acid, lithium, sertraline, olanzapine, escitalopram,

Table 15.3 Studies examining the effectiveness of pharmacological treatments for kleptomania.

Study	Trial	Design	Mean dose at endpoint (mg/d)	Duration	N[a]	Mean age	Attrition (%)	Proportion women (%)	Treatment response
Abiujaiude *et al.* [59]	Escitalopram vs. placebo	Open label	20	4–7 wk	11	46.0	15.4	81.8	79% improved 43% partial remission 57% complete remission
Grant and Kim [61]	Naltrexone	Open label	148	12 wk	10	37.0	33.3	70.0	20% partial remission 70% complete remission
Grant [62]	Naltrexone	Retrospective longitudinal study	135.3	3 yr	17	39.6	0	70.6	36% partial remission 41% complete remission

[a]The number of subjects included in the analyses

Table 15.4 Pharmacological treatments of pyromania: an overview [63].

Pharmacologic treatment for pyromania (N = 14)								
SSRI				Mood stabilizer	Antipsychotic	Anti-epileptic		
Citalopram n = 1	Escitalopram n = 3	Sertraline n = 2	Fluoxetine n = 2	Lithium n = 2	Olanzapine n = 1	Valproic acid n = 1	Clonazepam n = 1	Topiratmate n = 1
No response	2 partial/ complete remission 1 no response	1 partial/ complete remission 1 no response	1 partial/ complete remission 1 no response	1 partial/ complete remission 1 no response	No response	No response	No response	Partial/ complete remission

citalopram and clonazepam [63]. Treatment with escitalopram, sertraline, fluoxetine and lithium has demonstrated inconsistent results and, beyond the above-mentioned study, the rest of the pharmacological treatment literature on pyromania comprises single case studies. More research in this area is therefore needed in order to draw conclusions about the effectiveness of pharmacological treatment for pyromania.

15.6 Intermittent explosive disorder

IED is characterized by repeated episodes of serious assaultive acts or destruction of property that are out of proportion to any provocation or precipitating psychosocial stressor, and are not due to the direct physiological effects of a substance or better accounted for by another mental disorder [1]. Thus, IED must be distinguished from episodes of aggressive behavior that are due to antisocial personality disorder, borderline personality disorder, psychotic disorder, manic episode, conduct disorder or attention-deficit/hyperactivity disorder. IED usually starts during adolescence [65], and is more prevalent among males [1]. A recent prevalence study showed that the lifetime and last year prevalence rates of IED in the United States were 7.3 and 3.9% respectively [65]. High levels of co-morbidity with mood, anxiety and substance use disorders are reported [65, 66].

Pharmacological treatments of intermittent explosive disorder

Results from an epidemiologic study in the US showed that the majority (60.3%) of those diagnosed with lifetime IED had received treatment for emotional problems, but only 28.8% had received treatment specifically for IED [65]. It has been hypothesized that dysregulation of the serotonergic system and mild brain injuries may be central in the etiology of IED. The medications offered in treatment of patients with IED are mainly SSRIs, mood stabilizers and beta-blockers. However, the efficacy of these medications has mainly been determined through case reports, and controlled trials are needed to confirm the utility of these medications [66]. To the best of our knowledge, no randomized controlled trial of pharmacotherapy for IED has been conducted.

15.7 Conclusions

Common features of ICDs include urges, pleasure-seeking and inability to resist impulses. Several studies have demonstrated dysfunctions in neurotransmitter systems involved in these mechanisms, which may be targeted through pharmacological treatment. Pharmacological trials of different ICDs have shown promising results and demonstrated potential effectiveness of pharmacological treatments for these disorders. Still, several studies have limitations such as poor designs, relying on single cases, small sample sizes and lack of adequate control groups. More controlled studies in this area are needed to establish the effectiveness of pharmacological treatments for different impulse control disorders.

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