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Intermittent Explosive Disorder and the Impulse-Control Disorders

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Impulsivity has been defined as a predisposition toward rapid, unplanned reactions to either internal or external stimuli without regard for negative consequences. Given this definition of impulsivity, multiple psychiatric disorders might be characterized as exhibiting problems with impulse control. In the 5th edition of the Diagnostic and Statistical Manual of the American Psychiatric Association (DSM-5), the category of Impulse-Control Disorders Not Elsewhere Classified was dismantled and replaced with a new chapter on Disruptive, Impulse-Control, and Conduct Disorders. The classic impulse-control disorders still retained in the DSM-5 (Intermittent Explosive Disorder, Kleptomania, Pyromania) are unified by the presence of difficult, disruptive, aggressive, or antisocial behavior that is typically preceded by intense tension or arousal. Despite high prevalence rates in the general population (Kessler et al., 2005) and in psychiatric cohorts (Grant, Levine, Kim, & Potenza, 2005), Intermittent Explosive Disorder and the Impulse-Control Disorders have been relatively understudied, and the extent to which these disorders share clinical, genetic, phenomenological, and biological features is incompletely understood.

Intermittent Explosive Disorder (IED)

Human aggression consists in a behavioral act resulting in physical (or verbal) injury to self, others, or objects. It has several forms and may be defensive, premeditated (e.g., predatory), or impulsive (e.g., nonpremeditated), in nature. A converging pattern of data consistently points to critical differences between impulsive and premeditated aggression such that, while the two may appear in the same individual at different times, the underpinnings of the two are different (Barratt, Stanford, Felthous, & Kent, 1997; Raine et al., 1998). The most critical aspect of this phenomenon is that acts of impulsive aggression represent a quick—and typically angry— response, nearly always triggered by a social threat or frustration, that is out of proportion to the situation; premeditated aggressive acts, on the other hand, are thought out in advance and perpetrated for a tangible objective. Impulsive aggressive acts may include verbal attacks, temper tantrums (with or without property damage or harm to others), property assault, or assault to other living beings including animals. In fact, the severity of the aggressive outburst is less relevant than that the aggressive behavior is "explosive" in nature and that these acts cause distress to the individual or impairment in his or her psychosocial function, and are not due to another disorder (i.e., do not occur exclusively during that other disorder).

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While not widely known, a disorder of impulsive aggression has been included in the Diagnostic and Statistical Manual for Psychiatric Disorders (DSM) since the outset. It was called "Passive-Aggressive Disorder—Aggressive Subtype" and "Explosive Personality" in the original DSM and in DSM-II, respectively (American Psychiaric Association, 1952, 1968). With the publication of DSM-III (American Psychiatric Association, 1980), the disorder was codified as Intermittent Explosive Disorder (IED). At the time, IED was thought to be quite rare and was conceptualized as a disorder of exclusion (e.g., if some other diagnosis better explained the aggressive behavior that was the preferred diagnosis). In fact, DSM-III criteria were limited and poorly operationalized. Most importantly, it was not clear what constituted aggressive behavior, what type of aggressive behavior defined the disorder, and how often aggressive behavior should occur and in what time frame. At the same time that DSM-III was being embraced across the nation, new research suggested that aggressive behavior was associated with reduced functioning of the central serotonin (5-HT) system. Given that 5-HT is widely distributed across the brain, that 5-HT neurons fire at a relatively constant (tonic) rate, and that enhancing 5-HT function reduces aggression in animal models, the idea that 5-HT acts as a behavioral inhibitor, or "brake," became a popular model for human aggression (Coccaro, Fanning, Phan, & Lee, 2015). Consistent with this model, empirical data supports the idea that reduced 5-HT function is associated with impulsive, but not premeditated, aggression (Linnoila et al., 1983; Virkkunen et al., 1987). In this context of new data regarding the psychobiology of impulsive aggression, researchers began to modify DSM-III criteria in order to codify a disorder of impulsive (as opposed to generalized) aggression to allow for further research to occur. This led to research criteria which focused, first, on frequent outbursts of impulsive aggressive behavior and, then, incorporated DSM-IV IED criteria to create new diagnostic criteria for DSM-5.

DSM-5 Criteria for IED

The new "A" criteria for IED in DSM-5 now clearly define the frequency and temporal nature of applicable aggressive behavior. The A_1 criteria require verbal, and/or non-assaultive and non-destructive, aggressive outbursts occurring at an average of twice weekly for at least three months while the A_2 criteria require assaultive, and/or destructive, aggressive outbursts occurring at least three times a year. About 70% of those meeting criteria for DSM-5 IED meet both the A_1 and A_2 criteria, while 20% meet the A_2 criteria only and 10% meet the A_1 only (Coccaro, 2011). Empirical studies have shown that those meeting only the A_1 criteria do not differ from those meeting only the A_2 criteria or those meeting both A_1 and A_2 (Coccaro, Lee, & McCloskey, 2014). The "B" criteria for DSM-5 do not differ from the similar criteria in DSM-IV and continue to require that the aggressive behavior be out of proportion to the situation. The remaining criteria have all been revised. The "C" criteria require that most of the aggressive outbursts be impulsive in nature so that IED may not be given to someone who predominately engages in premeditated aggressive behavior; the "D" criteria require that the aggressive outbursts cause distress and/or impairment for the individual so that the IED diagnosis is not made in the absence of clinically significant consequences; the "E" criteria require that the individual is at least six years of age before a diagnosis of IED is given so that typical aggressive behavior seen in children younger than six years of age are not considered pathological. The last criterion ("F") was revised so that the diagnosis of IED can be given as long as the aggressive outbursts do not occur only during the course of another disorder or exogenous factor known to be associated with aggression. With these changes came the immediate exclusion of individuals with selected disorders such as borderline and/or antisocial personality disorder. This was added because individuals with these disorders were frequently not particularly aggressive compared with those with only IED (Coccaro, 2012).

Epidemiology of IED

The first U.S. community survey (the National Comorbidity Study Replication: NCS-R) that included DSM-IV IED in its assessment of adult psychopathology reported a lifetime prevalence of IED at 7.3% and a past year prevalence of 3.9% by "Broad Criteria." While these prevalence rates appear high, "Broad Criteria" only require the presence of serious aggressive outbursts three times in a person's adult life, which is not very frequent and is unlikely to be associated with significant distress or impairment. The second definition used in the NCS-R study, "Narrow IED," required the presence of aggressive outbursts at least three times in a single year, and found meaningful differences between the two definitions with "Narrow IED" being far more severe than "Broad IED" (Kessler et al., 2006). The lifetime and past year prevalence of "Narrow IED" were 5.4% and 2.7% respectively. Recently, we revisited these data and have determined that lifetime and past year prevalence of DSM-5 IED are about 3.6% and 2.2%, respectively (Coccaro, Fridberg et al., 2016). The rates of IED vary across countries and range from an average of 2.9% for the broad definition and 2.1% for the narrow. Studies of adolescents estimate the lifetime and past year prevalence for the broad definition of IED at 7.8% and 6.2%, respectively; corresponding prevalence for the narrow definition of IED were 5.3% and 1.7%, respectively (McLaughlin et al., 2012)

Phenomenology

Aggressive outbursts in IED have a rapid onset and are short-lived (<30 minutes). These outbursts are frequent and of low intensity (i.e., verbal outbursts and/or non-destructive/non-assaultive outbursts occurring about twice a week for at least three months) and/or infrequent but of high intensity (i.e., physically assaultive/destructive outbursts occurring three or more times per year; Coccaro, 2011). Aggressive outbursts most commonly occur in response to a minor provocation by a close associate. Episodes are associated with substantial distress, impairment in social functioning, occupational difficulty, and legal or financial problems (Kessler et al., 2006). In a reanalysis of the National Comorbidty Study-Replication (Coccaro, Fanning, & Lee, 2016), the lifetime prevalence of DSM-5 IED was 3.6% and the past year prevalence was 2.2%. IED appears as early as childhood (e.g., prepubertal) and peaks in mid-adolescence with a mean age of onset ranging from about 13 to 18 years in adult samples (Coccaro, Posternak, & Zimmerman, 2005; Kessler et al., 2006) and lower in adolescent samples (McLaughlin et al., 2012). The average duration of symptomatic IED ranges from nearly 12 years to 20 years to nearly the whole lifetime. Clinical studies suggest that IED is more common in males than females (3:1 ratio). However, our reanalysis of the NCS-R data suggest that the lifetime prevalence rate for DSM-5 IED in males only approaches twice that in females [Odds Ratio: 1.90 (95% CI:1.50–2.38)] and that the past year prevalence rate for males is even less than that [Odds Ratio: 1.50 (95% CI: 1.14–1.98)]. IED is often comorbid with other lifetime disorders such as depressive disorder, anxiety disorder and substance use disorder (Coccaro, 2011, 2012; Coccaro, Kavoussi, Berman, & Lish, 1998; Coccaro, Posternak, & Zimmerman, 2005). However, in most cases, the age of onset of IED precedes that of these other lifetime comorbid disorders (Coccaro et al., 2005) suggesting that IED is not a consequence of these other disorders (Kessler et al., 2006).

Behavioral Genetic and Family Studies Like all personality traits, aggression has a strong genetic influence. Overall, the heritability of aggression is up to 50% (Miles & Carey, 1997). However, most studies do not distinguish between impulsive and premeditated aggression and/or did not use measures that correlate with impulsive aggression in particular. In a series of studies carried out in our group we have found significant heritability for "irritable

aggressiveness" (Coccaro, Bergeman, & McCleam, 1993), for "irritability," "verbal assault," "indirect assault," and "direct assault," in twins raised apart. In the latter study we found that the degree of genetic influence increases as one goes from "verbal" to "indirect" to "direct" assault suggesting that the more severe the form of aggression, the greater the underlying degree of genetic influence. Similarly, impulsivity has substantial genetic influence. There is strong genetic correlation between impulsivity and the three different forms of aggression (Seroczynski, Bergeman, & Coccaro, 1999). More recent studies from our group support these data by showing elevated rates of IED by Research Criteria (essentially the same as DSM-5) compared with controls (Coccaro, 2010).

Comorbidity

Psychiatric Because impulsive aggressive behavior appears in patients with many diagnoses, most clinicians have been reluctant to make a diagnosis of IED in the presence of other psychiatric diagnoses. In fact, impulsive aggressive behavior is manifest in all humans, early in life, and the onset can occur prior to many other psychiatric disorders. Clinical studies suggest significant co-occurrence of IED with mood, anxiety, and substance use disorders. In most cases, the age of onset of IED is earlier than that for the co-occurring disorder suggesting the independence of IED or suggesting that IED is a risk factor for the co-occurring disorder.

Medical A relationship between impulsive aggression, or "irritability," associated with cardiovascular illness has been reported for many years. A recent analysis of a large community sample data has noted that individuals with IED have an increased risk of coronary heart disease, hypertension, stroke, diabetes, arthritis, back/neck pain, ulcer, headaches, and other chronic pain (McCloskey, Kleabir, Berman, Chen, & Coccaro, 2010). Another study reports a significant relationship between IED and diabetes (de Jonge et al., 2014). A factor tying many of these conditions together may be abnormalities of immune function (e.g., coronary heart disease, stroke, arthritis, ulcer, and others). Recently, we reported elevated plasma inflammatory markers (C-Reactive Protein: CRP and Interleukin-6: IL-6) in individuals with IED compared with psychiatric and healthy controls (Coccaro, Lee, & Coussons-Read., 2014). In addition to elevated levels of these markers, a history of aggressive behaviors and an aggressive disposition correlate directly with plasma levels of CRP and IL-6 (Coccaro, 2006; Coccaro, Lee, & Coussons-Read., 2014) and CSF levels of CRP (Coccaro, Lee, & Coussins-Read, 2015a) and of IL-1R11 protein (Coccaro, Lee, & Coussons-Read, 2015a). Recently, we reported that the presence of a latent infection with *toxoplasma gondii* was associated with an increased rate of IED compared with healthy control volunteers (Coccaro, Lee, Groer, Can, Coussons-Read, & Postolache, 2016)

Psychological Features

Not surprisingly, individuals with IED demonstrate abnormalities in a number of psychological areas. Compared with controls, individuals with IED have elevations of: (a) relational aggression aimed at damaging interpersonal relationships (Murray-Close, Ostrov, Nelson, Crick, & Coccaro, 2010); (b) hostile attribution, and negative emotional responding, to socially ambiguous stimuli (Coccaro, Fanning, & Lee, 2016; Coccaro, Noblett, & McCloskey, 2009); (c) affective lability and affective intensity (Fettich, McCloskey, Look, & Coccaro, 2015); and (d) immature defense mechanisms including acting out, dissociation, projection, and rationalization (Puhalla, McCloskey, Brickman, Fauber, & Coccaro, 2016). Most recently, individuals with IED have been reported to have reduced emotional intelligence (Coccaro,

Solis, Fanning, & Lee, 2015). All of which provide a rationale for psychological intervention, particularly those that focus on emotional and social information processing.

Biological Features

Neurotransmitters Biological studies clearly show a bio-behavioral relationship between aggression and selected brain chemicals, such as 5-HT. To date, individuals with IED are reported to have altered 5-HT function compared with Non-IED, or Healthy Control, subjects (Coccaro, Lee, & Kavoussi, 2009b, 2010; New et al., 2004). In fact, most biological studies of aggression report an association with anomalies of 5-HT including reduced levels of cerebrospinal fluid metabolites of 5-HT, reduced responsiveness of 5-HT receptors to stimulation, and reduced numbers of 5-HT transporter sites both on circulating platelets and on neurons in the brain (Duke, Bègue, Bell, & Eisenlohr-Moul, 2013). In addition, reduction of 5-HT levels, after tryptophan depletion, is associated with increased aggression on laboratory aggression tasks in "aggressive" human volunteers and is associated with greater rantings of anger in IED compared with healthy individuals (Lee, Gill, Chen, McCloskey, & Coccaro, 2012).

In addition to 5-HT, several other neurotransmitter/modulators have been found to correlate with measures of aggression. Those that correlate inversely with aggression include oxytocin (Lee, Ferris, Van de Kar, & Cocarro, 2009) and those that correlate positively with aggression include dopamine (Coccaro & Lee, 2010), vasopressin (Coccaro, Kavoussi, Hauger, Cooper, & Ferris, 1998), neuropeptide Y (Coccaro, Lee, Liu, & Mathe, 2012), substance p (Coccaro, Lee, Owens, Kinkead, & Nemeroff, 2012), glutamate (Coccaro, Lee, & Vezina, 2013), and inflammatory proteins (Coccaro, 2006; Coccaro, Fanning, Phan, & Lee, 2015; Coccaro, Lee, & Coussons-Read, 2014; Coccaro, Lee, & Coussons-Read, 2015a, 2015b; Coccaro, Lee, Fanning et al., 2016) as well as measures of oxidative stress (Coccaro, Lee, & Gozal, 2016).

Neuroimaging Structural neuroimaging studies have reported that individuals with IED have reduced grey matter volume in fronto-limbic areas including the orbital prefrontal cortex, ventromedial prefrontal cortex, anterior cingulate cortex, amygdala, insula, and uncus (Coccaro, Fitzgerald, Lee, McCloskey, & Phan, 2016). In addition, measures of aggression were found to correlate directly with grey matter volume in these areas. The shape of the amygdala is also abnormal in individuals with IED compared with healthy controls. In this morphometry study, individuals with IED have significantly more areas of inward deformation in the amygdala compared with healthy controls (Coccaro, Lee, McCloskey, Csemansky, & Wang, 2015). Diffusion tensor imaging (DTI) reveals lower fractional anisotropy in two clusters located in the superior longitudinal fasciculus when compared with psychiatric and healthy controls (Lee et al., 2016). This suggests lower white matter integrity in long-range connections between the frontal and temporo-parietal regions of the brain and likely problems with connectivity between these brain regions.

Functional magnetic resonance imaging studies report greater amygdala response to exposure to angry faces in IED compared with healthy controls. This finding is true for implicit (Coccaro, McCloskey, Fitzgerald, & Phan, 2007; McCloskey, Fitzgerald, Lee, McCloskey, & Phan, 2016) and explicit emotional processing. Life-time history of aggression correlates with amygdala response to the angry faces. Finally, acute activation of 5-HT receptors by a single dose of citalopram has been associated with an enhanced fMRI signal response to angry faces in the left temporal parietal junction (TPJ) of IED compared with healthy control individuals (Cremers, Lee, Keedy, Phan, & Coccaro, 2015). Since the TPJ is associated with social-cognitive processes, such as perspective taking and empathy, it is

possible that SSRIs administration may reduce aggressive tendencies towards other people by enhancing these social-cognitive processes.

Treatments for IED in Adults

Subsequent chapters in this book (Chapters 25 through 28) address intervention programs focusing on conduct disorder and oppositional defiant disorder, and on the symptomatic behaviors associated with those disorders, including aggression. This chapter provides an overview of treatment considerations for intermittent explosive disorder, and then, in later sections, for impulse-control disorders (kleptomania; pyromania). Because of the limited literature on treatment of adolescents with these disorders, the literature on research with adults is primarily summarized, and carries implications for future research with youth.

Pharmacological While there are no medications approved by the Food and Drug Administration (FDA) for the treatment of IED, several psychopharmacologic agents appear to have effects on aggression. Double-blind, placebo-controlled, clinical trials in patients with impulsive aggression have been conducted over the past decade. The first studies reported a reduction in impulsive aggressive behavior by the serotonin-activating antidepressant, fluoxetine, in impulsive aggressive individuals with personality disorders (Coccaro, Kavoussi, & Lesser, 1992; Salzman et al., 1995) and this has been replicated in three other studies (Coccaro & Kavoussi, 1997; George et al., 2011; Silva et al., 2010) and in a study with individuals with IED (Coccaro, Lee, & Kavoussi, 2009a). Effective doses for fluoxetine are in the 20–40 mg qd range (Coccaro, Lee, & Kavoussi, 2009a).

Other classes of agents shown to have "anti-aggressive" effects in double-blind, placebo-controlled trials of individuals with "primary" aggression (i.e., not secondary to psychosis, severe mood disorder, or organic brain syndromes) include mood stabilizers (lithium: Sheard, Marini, Bridges, & Wagner, 1976) and anticonvulsants (phenytoin: Barratt et al., 1997; carbamazepine: Gardner & Cowdry, 1986; oxcarbazepine: Mattes, 2005; divalproex: Hollander et al., 2003). While NE beta-blockers (e.g., propanolol/nadolol: Mattes, 1990; Ratey et al., 1992) have also been shown to reduce aggression, these agents have exclusively been tested in patient populations with "secondary" aggression (e.g., mental retardation, organic brain syndromes, etc.). Classes of agents that may have "pro-aggressive" effects include tricylic antidepressants (amitriptyline: Soloff, George, Nathan, Schulz, & Perel, 1986; benzodiazapines: Gardner & Cowdry, 1985), and stimulant and hallucinatory drugs of abuse (amphetamines, cocaine, phencyclidine: Fishbein & Tarter, 2009). Findings from double-blind, placebo-controlled, clinical trials suggest that anti-aggressive efficacy is specific to impulsive, rather than non-impulsive, aggression (Barratt et al., 1997).

Psychological A variety of cognitive-behavioral treatments have been found to be moderately (or more) effective in the treatment of anger and/or aggression in adults or youth. Specifically, some cognitive-behavioral techniques have demonstrated a reduction in anger or the aggressive behaviors of classroom children, juvenile delinquents, residentially placed adolescents, college students, drivers, abusive parents and spouses, and prison inmates (Beck & Fernandez, 1998; Deffenbacher, Huff, Lynch, Oetting, & Salvatore, 2000; Edmondson & Conger, 1996; Novaco, 1977). Many treatments have been found to be effective at follow-up of up to 15 months, often with additional improvement gains noted at follow-up relative to post-treatment (Deffenbacher, 1988; Deffenbacher, McNamara, Stark, & Sabadell, 1990; Deffenbacher, Oetting, Huff, Cornell, & Dallager, 1996; Deffenbacher, Oetting, Huff, & Thwaites, 1996; Deffenbacher & Stark, 1992; Hazaleus & Deffenbacher, 1986). Specific

treatments have included relaxation training, social skills training, skill assembly social skills training, problem solving, negative thought reduction, self-instruction, cognitive therapy, and combined cognitive-relaxation or cognitive-behavioral treatment. Notably, however, the anger treatment literature does not discriminate between clinical anger problems without aggression and pathological aggression and so these findings may not generalize to more severely aggressive individuals with IED.

The first, and only, study of cognitive-behavioral therapy (CBT vs. wait-list control) in DSM-5 IED demonstrated that impulsive aggression, anger, and hostile thoughts are significantly reduced by a CBT package that includes relaxation training, cognitive restructuring, and coping skills training (McCloskey, Noblett, Deffenbacher, Gollan, & Coccaro, 2008). Fluoxetine and CBT demonstrated similar therapeutic responses and given that both treatments are likely working through different mechanisms, combination of the two kinds of treatments may be more effective than either alone.

Kleptomania

Epidemiology

Although the precise prevalence rates of Kleptomania remain unknown, rates in treatment-seeking samples suggest that it is fairly common. Kleptomania is also experienced by a broad range of psychiatric patient populations including 3.7% of depressed patients (n = 107; Lejoyeux, Arbaretaz, McLoughlin, & Adès, 2002) and 24% of those with bulimia (Hudson, Pope, Jonas, & Yurgelun-Todd, 1983). A study of psychiatric inpatients (n = 204) with a range of admitting disorders revealed that 7.8% (n = 16) endorsed current kleptomania and 9.3% (n = 19) met lifetime criteria (Grant et al., 2005). Approximately two-thirds of individuals with kleptomania, in clinical samples, are women (McElroy, Pope, Hudson, Keck, & White, 1991).

Phenomenology

Kleptomania is characterized by repetitive, uncontrollable stealing of items that people do not need for their personal use. Although kleptomania typically has its onset in early adulthood or late adolescence (McElroy et al., 1991), the disorder has been reported in children as young as 4 years old and in adults as old as 77 (Grant, 2006). The literature clearly suggests that the majority of patients with kleptomania are women (McElroy et al., 1991). Items stolen are typically hoarded, given away, returned to the store, or thrown away (McElroy et al., 1991). Many individuals with kleptomania (64% to 87%) have been apprehended at some time due to their stealing, and 15% to 23% report having been incarcerated (Grant, 2006). Most individuals with kleptomania try unsuccessfully to stop. The diminished ability to stop often leads to feelings of shame and guilt. Of married subjects, less than half had disclosed their behavior to their spouses due to shame and guilt (Grant, 2006). High rates of other psychiatric disorders (depression, bipolar disorder, anxiety disorders, substance use disorders, and eating disorders) are common in individuals with kleptomania (Grant, 2006; McElroy et al., 1991).

Treatments for Kleptomania

Pharmacological There have been only two placebo-controlled trials for kleptomania. One study investigated the antidepressant, escitalopram, in the treatment of kleptomania. Subjects were all given open-label escitalopram for 7 weeks and those who responded (n = 15) were then randomized to either continue on the medication or be switched to placebo (Koran,

Aboujaoude, & Gamel, 2007). After randomization, 43% of those on escitalopram relapsed compared to 50% on placebo, thereby showing no drug effect in terms of response.

In an open-label study of naltrexone, 12 subjects received doses ranging 50–150 mg per day (mg/d). Naltrexone resulted in a significant decline in the intensity of urges to steal, stealing thoughts and stealing behavior (Grant & Kim, 2001). In a follow-up double-blind, placebo-controlled study of naltrexone, 25 kleptomania subjects received naltrexone or placebo for 8 weeks. The study found naltrexone significantly reduced urges to steal and stealing behavior compared to placebo (Grant, Kim, & Odlaug, 2009).

Psychological No controlled studies of psychological treatments exist for kleptomania. Case reports suggest that cognitive and behavioral therapies may be effective in treating kleptomania. Covert sensitization, where a person is instructed to imagine stealing as well as the negative consequences of stealing (e.g., being handcuffed, feeling embarrassed), has been successful in reducing kleptomania symptoms (Grant, 2006). Imaginal desensitization, where a person images the steps of stealing but also imagines her ability to resist the behavior, has also successfully reduced kleptomania behavior (Grant, Odlaug, & Donahue, 2012).

Pyromania

Epidemiology

As with Kleptomania, the precise prevalence rate of Pyromania is unknown, though studies in treatment-seeking samples suggest that pyromania is also fairly common. One study of 107 patients with depression found that 3 (2.8%) met current DSM-IV-TR criteria for pyromania (Lejoyeux et al., 2002), and a study of 204 psychiatric inpatients revealed that 3.4% (n=7) endorsed current and 5.9% (n=12) had lifetime symptoms meeting DSM-IV-TR criteria for pyromania (Grant et al., 2005). The gender ratio in pyromania is unknown.

Phenomenology

The DSM-5 describes Pyromania as a preoccupation with fire setting and characterizes the behavior with the following diagnostic criteria: (a) deliberate and purposeful fire setting on more than one occasion; (b) tension or affective arousal before the act; (c) fascination with, interest in, curiosity about, or attraction to fire and its situational contexts; and (d) pleasure, gratification, or relief when setting fires or when witnessing or participating in their aftermath.

Although long thought to be a disorder primarily affecting men, recent research suggests that the gender ratio is equal in adults and may be slightly higher among females in adolescence (Grant & Kim, 2007). Mean age of onset is generally late adolescent, and the behavior appears chronic if left untreated (Grant & Kim, 2007). Urges to set fires are common in individuals with this behavior and the fire setting is almost always pleasurable. Severe distress follows the fire setting and individuals with pyromania report significant functional impairment (Grant & Kim, 2007).

Treatment

There is no controlled pharmacological or psychological treatment data regarding pyromania. However, cognitive-behavioral treatment has been found to be useful in reducing deviant fire-related behaviors among children who had set recent fires (Kolko, 2001), suggesting such treatments could be explored and tested for these symptoms of pyromania.

Conclusions

Clinicians evaluating patients with intermittent explosive disorder and impulse-control disorders should assess the circumstances that led them to seek help. In most psychiatric disorders, patients seek treatment because they are troubled by their symptoms. Patients with impulse-control disorders, however, continue to struggle with the desire to engage in the behavior and their need to stop because of mounting social, occupational, financial, or legal problems.

In the area of intermittent explosive disorder and impulse-control disorders, the systematic study of treatment efficacy and tolerability is in its infancy. With few studies published, it is not possible to make treatment recommendations with a substantial degree of confidence. No drugs are currently approved by the FDA for the treatment of any of these disorders.

Clinicians should be aware of the limitations of our treatment knowledge. Most published studies employed relatively small sample sizes, were of limited duration, and involved possibly nonrepresentative clinical groups (e.g., those without co-occurring psychiatric disorders). Heterogeneity of treatment samples may also complicate identification of effective treatments. At present, issues such as which medication to use and for whom, or the duration of pharmacotherapy or cognitive behavior therapy cannot be sufficiently addressed with the available data.

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