

# impulsivity and compulsivity

## UNDERSTANDING THE DIFFERENCES

Impulsivity and compulsivity are natural behaviors controlled by brain mechanisms that are essential for survival in all species. Understanding these brain mechanisms may lead to targeted treatment strategies for these symptom domains when impulsivity and compulsivity become dysfunctional. Pathological impulsivity and compulsivity characterize a broad range of mental disorders, and are the core and most debilitating symptoms, at least phenotypically, in some of the disorders in which these behaviors occur. These illnesses, some of which are highly heritable, are currently classified across several *DSM-IV-TR* diagnostic categories. Obsessive-compulsive spectrum disorders include obsessive-compulsive disorder (OCD), body dysmorphic disorder, trichotillomania, Tourette syndrome, and hypochondriasis. Disorders involving deficits in impulse control include pathological gambling, externalizing disorders such as attention-deficit/hyperactivity disorder (ADHD), personality disorders such as borderline personality disorder, and substance and behavioral addictions.

### Impulsivity Versus Compulsivity

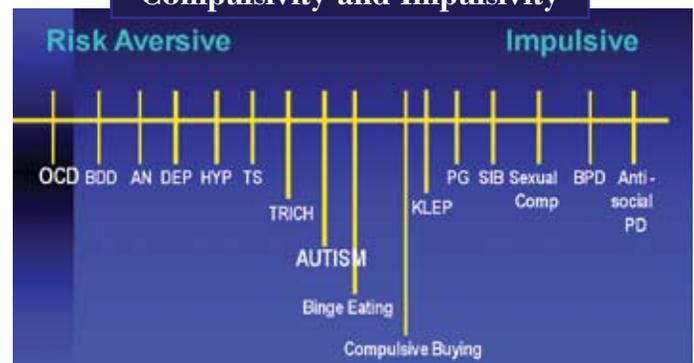
The concept of impulsivity has many different aspects and definitions, but in general it covers a wide range of “actions that are poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and that often result in undesirable outcomes,” or more simply put, a tendency to act prematurely and without foresight (*DSM-IV-TR*, 2000). Moeller and colleagues (Moeller, Barratt, Dougherty, Schmitz & Swann, 2001) defined impulsivity as “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others.” But, impulsivity is not always unplanned; for example, some pathological gamblers plan in advance to pursue their impulsive behavior. Impulsive behaviors can be conceptualized as being the core symptoms of a broad range of psychiatric disorders that are often comorbid with one another, including cluster B personality disorders, impulse control disorders, and bipolar disorder.

In contrast, compulsivity refers to repetitive behaviors performed according to certain rules or in a stereotypical fashion (Grant & Potenza, 2006). Compulsivity is a tendency to repeat the same, often purposeless acts, which are sometimes associated with undesirable consequences. Impulsivity and compulsivity may be viewed as diametrically opposed, or alternatively, as similar, in that each implies a dysfunction of impulse control (Stein, et al., 1996). Each involves alterations within a wide range of neural processes including, for example, attention, perception, and coordination of a motor or cognitive response. Objective neurocognitive tests hold potential for elucidating the mechanisms by which pharmacological agents exert their beneficial clinical effects, and for predicting clinical outcomes (Menzies, et al., 2007). Using sensitive and domain-specific neurocognitive tasks, we may also be able to fractionate impulsivity and compulsivity into separate and quantifiable neurobiologically specific domains (Fineberg, Hollander & Potenza, et al., In preparation).

Disorders characterized by impulsivity include impulse control disorders in the *DSM-IV-TR*, representing a failure to resist aggressive impulses (as in intermittent explosive disorder) and urges to steal (kleptomania), set fires (pyromania), gamble (pathological gambling), and pull one’s hair (trichotillomania). However, behaviors characteristic of these disorders may also manifest as symptoms of another mental disorder. A number of other disorders are not included as a distinct category but are categorized as impulse control disorders not otherwise specified in *DSM-IV-TR*. These include sexual compulsions, compulsive shopping, skin picking, and Internet addiction. Impulse control disorders share the feature of the irresistible urge to act in a given way, and may be considered as a subset of the obsessive-compulsive spectrum of disorders.

The obsessive-compulsive spectrum is a dimensional model of risk avoidance in which impulsivity and compulsivity represent polar opposite psychiatric spectrum complexes that can be viewed along a continuum of compulsive and impulsive disorders (*Figure 1*). Patients on the compulsive end of the spectrum tend to have an exaggerated sense of harm from the outside world, and engage in rituals/routines, such as obsessive-compulsive behaviors, to neutralize the threat or reduce the harm. This endpoint marks compulsive or risk-averse behaviors characterized by overestimation of the probability of future harm, exemplified by OCD. But some compulsive patients pursue unrewarding rituals for short-term gains (relief of tension) despite negative long-term consequences. Generally, however, OCD rituals are not pleasurable activities engaged in for their own sake, but rather are neutral or often irritating and unpleasant behaviors that are engaged in to reduce anxiety.

### A Dimensional Approach to Compulsivity and Impulsivity



Note: OCD, obsessive-compulsive disorder; BDD, body dysmorphic disorder; AN, anorexia; DEP, depression; HYP, hypochondriasis; TS, Tourette's syndrome; TRICH, trichotillomania; KLEP, kleptomania; PG, pathological gambling; SIB, self-injurious behavior; Comp, compulsion; BPD, borderline personality disorder; PD, personality disorder (Source: Modified from Hollander & Wong, 1995)

Patients on the impulsive end of the spectrum tend to underestimate the harm associated with behaviors such as aggression, excessive gambling, or self-injury. This endpoint designates impulsive action

generally characterized by a lack of complete consideration of the negative results of such behavior, exemplified by borderline and antisocial personality disorders (Hollander & Rosen, 2000). Some impulsive patients do recognize and assess the harm associated with the impulsive behavior but nonetheless still engage in it because they find that the thrill or arousal they experience in response to the behavior outweighs the negative consequences.

Impulsive behaviors generally have an element of pleasure, at least initially, although they may lose their pleasurable quality over time. Some patients with impulse control disorders may engage in the behavior to increase arousal, but there may be a compulsive component to their behavior in which they continue to engage in the behavior to decrease dysphoria. So, in general, while compulsivity may be driven by an attempt to alleviate anxiety or discomfort, impulsivity may be driven by the desire to obtain pleasure, arousal, or gratification. Both types of behaviors however, share the inability to inhibit or delay repetitive behaviors. Over time, impulsive behaviors may become compulsive (driven behaviors without arousal), and compulsive behaviors may become impulsive (reinforced habits).

### **Contributing Factors**

There are many contributing factors to impulsivity and compulsivity, such as genes, sex, environment, psychiatric disorders, and substance abuse. The neurobiology of impulsivity and compulsivity may involve inhibitory neurotransmitters such as serotonin and gamma-aminobutyric acid (GABA); excitatory neurotransmitters such as glutamate, norepinephrine, and dopamine; and prefrontal cortex and/or limbic dysfunction. Convergent evidence suggests that a failure in top-down cortical control mechanisms, leading to striatal overdrive, may constitute a unifying pathophysiological model underpinning an “impulsive-compulsive spectrum” of mental disorders (Chamberlain & Sahakian, 2007). Increased frontal lobe activity may characterize the compulsive disorders, such as OCD. In contrast, decreased frontal lobe activity may characterize the impulsive disorders, such as pathological gambling and borderline personality disorder (Hollander & Rosen, 2000).

Impulsive and compulsive features may present at the same time or at different times during the same illness (Hollander & Wong, 1995). Although both compulsive and impulsive disorders may be related to prefrontal cortex dysfunction, compulsive disorders would be related to hyperactivity and impulsive disorders to hypoactivity of the prefrontal cortex. Compulsiveness appears to be associated with increased frontal lobe activity, while impulsiveness appears to be associated with reduced frontal lobe activity.

### **Treatment Targets**

The impulse control disorders may be conceptualized in addictive, affect-driven, and compulsive models. Targeted treatments of impulsivity in impulse control disorders may influence the motivational circuitry, or work via addictive, affect-driven, and compulsive systems. Treatments should also target comorbid bipolar spectrum, ADHD, and compulsive and addictive disorders for maximal anti-impulsive effects. There is some evidence that different symptom dimensions within the impulse control disorders are particularly responsive to different medication classes (Coccaro, 1998; Berlin, Rolls & Iversen, 2005). It is therefore important to individualize treatment decisions based on the limited evidence base, the patient’s presenting problems, history, and comorbid conditions.

For example, a patient with borderline personality disorder with prominent cognitive/perceptual distortion may respond to

neuroleptics, while those with depressed mood may respond best to antidepressants. Some symptom dimensions (e.g., antisocial traits) may be less responsive to medication, and some classes of medication, including the benzodiazepines, do not appear particularly effective for the treatment of impulse control disorders and should therefore generally be avoided (Stein, Harvey, Seedat & Hollander, 2006).

There may be several unique developmental trajectories to impulsivity and compulsivity (e.g., ADHD, bipolar spectrum, trait impulsivity, obsessive compulsive personality disorder) and various routes to altering motivational circuitry, such as modulators of cortico-striatal-limbic circuits. We suggest that core symptoms within disorders should be treated and appropriate outcome measures should be used to determine targeted treatment response. Interventions should be directed at the brain circuitry that modulates core symptoms, which may be shared across disorders rather than *DSM* diagnoses (Berlin & Hollander, 2008).

Although the neurobiological basis of OCD (symptoms and related cognitive impairments) is unclear, lesion, functional neuroimaging, and neuropsychological studies have suggested that structural and functional dysfunction of limbic or affective cortico-striato-thalamo-cortical circuitry, which includes the orbitofrontal cortex, plays a key role (Swedo & Snider, 2004). These circuits, first identified in nonhuman primates, have also been identified in human lesion and imaging studies of patients who have OCD (Menzies, et al., 2007).

### **Treatment Approaches**

Intervention can occur at the symptom, syndrome, or behavioral level. Effective treatment of impulsivity and compulsivity depends on determining the cause(s) of these behaviors and selecting treatments accordingly. Pharmacological and nonpharmacological treatment, such as behavioral strategies aimed at reducing impulsive and compulsive behavior, may be most effective for the long-term treatment of the underlying chronic or recurrent illness.

There is no standardized treatment for complex disorders involving impulsivity, although a range of different medication classes have been investigated (Stein, et al., 2006). Pharmacological treatments may reduce impulsivity and normalize arousal by reducing dopaminergic activity, enhancing serotonergic activity, shifting the balance of amino acid neurotransmitter from excitatory (glutamatergic) toward inhibitory (GABAergic) transmission, decreasing glutamatergic conduction, and/or reducing or stabilizing nonadrenergic effects. Medications used to treat disorders involving impulsivity, including impulse control disorders and cluster B personality disorders, which have been shown to be effective in some clinical trials, include selective serotonin reuptake inhibitors (SSRIs), lithium, and anticonvulsants (Berlin et al., 2008; Hollander & Berlin, 2008; Hollander, et al., 2008). CBT and psychodynamically informed psychotherapy have a useful role in the management of a number of impulse control disorders. More specific details of the pharmacotherapy and psychotherapy approaches to each of the individual impulse control disorders can be found elsewhere (Simeon & Berlin, 2007).

With regard to compulsive behavior, the most common treatment approaches for OCD are pharmacological and psychological. Cognitive-behavioral therapy (CBT) was the first psychological treatment for which empirical support was obtained. A recent review compared psychological treatments with treatment as usual and found that psychological treatments derived from cognitive-behavioral models are effective for adults with OCD (Gava, Barbui & Aguglia, 2007).

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Based on the hypothesized underlying neurobiology of OCD and observed treatment effects, SSRIs are considered first-line treatments for OCD. However, SSRIs are often associated with delayed onset of therapeutic effect (8 to 12 weeks), only partial symptom reduction, and response failure or intolerability in 40 to 60 percent of patients. Pharmacological options for SSRI-refractory cases include increasing drug dosage, changing to another SSRI or clomipramine, combining SSRIs, or changing the mode of drug delivery. Augmentation with second-generation antipsychotics appears promising, as does augmentation or monotherapy with some anticonvulsants (Hollander, Baldini, Sood & Pallanti, 2003; Berlin, Koran & Jenike, et al., In review; Koran, Hanna & Hollander, 2007).

### Alternative Interventions

Some patients with OCD remain refractory to all standard pharmacological and psychological treatments. Several alternative medical interventions have been considered for these severe cases, including ablative neurosurgery and brain stimulation techniques such as electroconvulsive therapy, transcranial magnetic stimulation (TMS), and deep brain stimulation (DBS-the nonablative neurosurgical procedure). Studies exploring these techniques for OCD treatment are limited by small sample sizes and scarcity of double-blind trials, and none of these alternative interventions are FDA approved for treatment of OCD. However, given the promising efficacy findings thus far, reversibility, noninvasiveness or minimal invasiveness, tolerability, and possibility of double-blind trials, additional research should be conducted with TMS and DBS to refine these techniques, better establish their efficacy, and offer more options to patients who have exhausted almost all other available treatments (Berlin, Hamilton & Hollander, 2008).

### Conclusion

In general, evidence suggests that mood stabilizers appear to be effective for treating the symptom domains of impulsivity and compulsivity across a wide range of psychiatric disorders and for impulse control and cluster B personality disorders in particular. We suggest that clinicians target and treat core symptoms of impulsivity and compulsivity based on the underlying neurobiology of these behaviors, instead of the overall diagnosis, while taking into account comorbid disorders, associated symptoms, developmental trajectory, and family history. ▼

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