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Addictive Behaviors

Impulsive action and impulsive choice across substance and behavioral addictions: Cause or consequence?

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HIGHLIGHTS

- Behavioral addictions associated with impulsive actions.
- Findings are similar to those seen in chronic substance use disorders.
- Whether cognitive deficits are cause or effect remains unclear.

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ABSTRACT

Substance use disorders are prevalent and debilitating. Certain behavioral syndromes ('behavioral addictions') characterized by repetitive habits, such as gambling disorder, stealing, shopping, and compulsive internet use, may share clinical, co-morbid, and neurobiological parallels with substance addictions. This review considers overlap between substance and behavioral addictions with a particular focus on impulsive action (inability to inhibit motor responses), and impulsive choice (preference for immediate smaller rewards to the detriment of long-term outcomes). We find that acute consumption of drugs with abuse potential is capable of modulating impulsive choice and action, although magnitude and direction of effect appear contingent on baseline function. Many lines of evidence, including findings from meta-analyses, show an association between chronic drug use and elevated impulsive choice and action. In some instances, elevated impulsive choice and action have been found to predate the development of substance use disorders, perhaps signifying their candidacy as objective vulnerability markers. Research in behavioral addictions is preliminary, and has mostly focused on impulsive action, finding this to be elevated versus controls, similar to that seen in chronic substance use disorders. Only a handful of imaging studies has explored the neural correlates of impulsive action and choice across these disorders. Key areas for future research are highlighted along with potential implications in terms of neurobiological models and treatment. In particular, future work should further explore whether the cognitive deficits identified are state or trait in nature: i.e. are evident before addiction perhaps signaling risk; or are a consequence of repetitive engagement in habitual behavior; and effects of novel agents known to modulate these cognitive abilities on various addictive disorders.

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1. Introduction

Several behavioral problems (for example, gambling, stealing, shopping, and use of the internet) have been hypothesized to have similarities to substance addictions and there is interest in whether they could be usefully conceptualized as 'behavioral addictions' (i.e. people compulsively and dysfunctionally engage in an activity without exogenous drug administration) (Holden, 2001). Support for such a conceptualization would ideally arise from several complimentary perspectives, including evidence of overlapping phenomenology, comorbidity, neurobiology, and treatment response. While evidence of such overlap is in many cases lacking (e.g. Kor, Fogel, Reid, & Potenza, 2013), the latest version of the Diagnostic and Statistical Manual (DSM-5) recognized the utility of this conceptualization for the purposes of gambling disorder, which now sits within the category of Substance-related and Addictive Disorders.

From a phenomenological perspective, substance addictions are typically characterized by repetitive habitual engagement in drug use (escalation in quantity and/or frequency of use over time), unsuccessful attempts to cut back, craving (intense desires to obtain substances), persistence despite negative functional impact (e.g. in terms of relationships or health consequences), narrowing of repertoire (less functionally appropriate behavior), increased engagement to produce a given effect (tolerance), and withdrawal symptoms (unpleasant physical consequences when use is reduced or stopped). Many of these phenomenological aspects are shared with putative behavioral addictions (e.g. see Leeman & Potenza, 2012). For example, individuals who gamble occasionally may experience initial pleasure and be able to control gambling-related urges, but over time, this behavior may become ingrained, more 'habitual' than 'pleasurable', and difficult to resist, with a profound negative impact on everyday functioning (el-Guebaly, Mudry, Zohar, Tavares, & Potenza, 2012). Exposure to gambling-related environmental cues can trigger craving, in much the same way that drug-related cues can trigger craving in substance-addicted individuals. Pathological gamblers often make unsuccessful attempts to cut back and experience symptoms akin to withdrawal when resisting the behavior (Cunningham-Williams, Gattis, Dore, Shi, & Spitznagel, 2009).

There is an ongoing search in psychiatry for neurobiological markers implicated in given behavioral domains that cross specific diagnostic categories. Neurobiological models of addiction emphasize the likely involvement of excess activity of the basal ganglia reward and habit forming system coupled with a lack of top-down control or inhibition (Bari & Robbins, 2013; Cardinal & Everitt, 2004; Robbins, Everitt, & Nutt, 2008). For substance addiction, there is a translational evidence of a postulated shift over time from a behavior that is initially rewarding (implicating the ventral striatum) to one that becomes habitual and compulsive (implicating the dorsal striatum) (Everitt & Robbins, 2013). However, diminished control over such actions (impulsive action) and preference for a small immediate gratification rather than a larger delayed gratification (impulsive choice) may serve to augment both aspects of striatally-mediated behaviors. While indubitably not capturing all facets of these illnesses, just as someone with alcoholism cannot seem to suppress the habitual act of consuming alcohol and seeks a short-term rewarding 'hit', so, too, do individuals with behavioral addictions report difficulties in stopping their habits and in averting their desire for short-term reward. While cognitive deficits have been reported across various domains in individuals with substance addiction and certain behavioral addictions versus healthy controls (e.g. Dom, De Wilde, Hulstijn, van den Brink, & Sabbe, 2006; Durazzo & Meyerhoff, 2007; Kalechstein, De La Garza, Mahoney, Fantegrossi, & Newton, 2007; Nnadi, Mimiko, McCurtis, & Cadet, 2005; Riggs & Greenberg, 2009; van Holst, van den Brink, Veltman, & Goudriaan, 2010), the domain of behavioral inhibition may represent a particularly fruitful arena for the search for candidate cognitive vulnerability markers. This primer focuses on findings using tests quantifying aspects of behavioral inhibition in this context.

2. Cognitive tasks fractionating aspects of behavioral inhibition: neural and neurochemical substrates

With respect to modeling behavioral inhibition, several potentially dissociable cognitive domains have been proposed (Bari & Robbins, 2013). For the purposes of this selective review, we focus on deficient response inhibition (impulsive action), and deficient deferment of gratification (impulsive choice).

2.1. Deficient response inhibition (impulsive action)

Response inhibition refers to the ability to suppress a given response when signaled to do so in response to environmental cues and is typically operationalized by go/no-go and stop-signal test (SST) paradigms (e.g. see Logan, Cowan, & Davis, 1984; Schachar et al., 2007; Eagle, Bari, & Robbins, 2008). Both types of test require participants to make simple motor responses (such as pressing a left or right button) in response to cues (such as left and right directional arrows appearing on a computer screen) — these are referred to as 'go' trials. On a minority of trials, participants attempt to withhold their usual response due to the presence of a stop-cue (referred to as 'stop' trials). The relative excess of go trials renders the go response 'pre-potent'. For go/no-go tasks, the stop-cue is presented alongside (at the same time) as the go-cue: therefore, the response has not already been triggered; crucially, on stop-signal tasks, the stop-cue is presented a variable time after the go-cue. As such, stop-signal tasks assess the ability of the participant's brain to suppress already triggered responses. Stop-signal tasks may be more sensitive to inhibitory dyscontrol since they use tracking algorithms that flexibly adapt to the individual's performance. Via