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Impulsivity, Compulsivity, and Habit: The Role of Orbitofrontal Cortex Revisited

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Humans suffering damage to the orbitofrontal cortex (OFC) are often described as impulsive. The most famous example is Phineas Gage, a railway worker, who in 1848 suffered extreme frontal lobe damage when a long iron rod was projected through his skull after an accidental explosion. Gage survived, but was reported to have an extreme change in personality, including increased inappropriate behavior (impulsiveness) [1–2]. People with OFC lesions are more impulsive compared to both normal controls and people with non-OFC frontal cortex damage, as measured by self-report and by cognitive/behavioral tasks [3]. But, how is impulsivity defined and how can it be measured?

Impulsivity involves behaviors that are inappropriate for the context, premature, poorly planned and often resulting in adverse consequences. Impulsive behaviors have been described as having three dimensions: 1) an inability to use available information to reflect on the consequences of actions; 2) an inability to forego an immediate small reward in favor of a delayed larger reward; 3) a deficit in suppressing prepotent motor responses [4]. Taken together, these three dimensions of impulsivity reflect an inability to evaluate and subsequently respond flexibly in search of a specific goal or outcome under changing environmental conditions. In this commentary, we reflect on the similarities between impulsive, compulsive and habitual behavior and hypothesize a common neurobiological circuit that depends critically on the function of the OFC.

The ‘toggle’ between flexible, goal-directed actions and reflexive, stimulus-driven habits

In combination with its well-described involvement in inhibitory control [5;6] the prefrontal cortex (PFC) – including the OFC -- is critical for decision-making and response-selection. The PFC is impaired in disorders of impulsivity and compulsivity such as drug addiction, obsessive compulsive disorder, attention-deficit disorder and Tourette syndrome [5;7–12].

Distinct regions of the PFC work in concert with the striatum, forming a distributed network responsible for processing of reward information, reward-related learning, goal-directed actions and the formation of habits [13–20]. For example, during the acquisition of actions such as lever-pressing, performance is controlled by an expectation of the future consequences

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of that action (i.e., a R-O association is acquired). This form of goal-directed instrumental learning is flexible and is argued to be under PFC executive control, as is shown by a reduction in response performance when the outcome is made less attractive (outcome devaluation) [21]. Following repeated practice, however, such control is diminished and performance shifts to depend upon an association between antecedent environmental stimuli and the response (i.e., a S-R association is formed). Accordingly, as training progresses, instrumental performance becomes increasingly insensitive to outcome devaluation [22]. This criterion provides an objective means to discriminate between goal-directed actions and stimulus-driven habits [23], known to be dependent upon corticostriatal circuits [24].

OFC and the modulation of goal-directed behavior

Cognitive control of behavior – the ability to integrate thoughts, emotions and individual motor responses into coordinated, goal-directed behavior – is thought to depend upon the OFC [25]. Several studies have demonstrated that lesions of the OFC in both humans and rodents impair the ability to use outcome expectancies in guiding behavior [26–29]. Further, neuroimaging studies reveal changes in OFC activation during action selection following reinforcer devaluation [30–31]. Therefore, the OFC may be critically important for guiding behavior on the basis of available information about the consequences of one's actions. This ability is impaired both in individuals engaged in stimulus-driven habits and in those who have impulse-control disorders, suggesting that these behaviors may have similar neurobiological features. Accordingly, several studies investigating the role of OFC in impulsivity also suggest a role for OFC in habit.

OFC and impulsivity

One dimension of impulsivity is the inability to forego small, immediate rewards for larger, delayed rewards. This deficiency can be quantified in a behavioral task known as delay discounting. In general, the person or animal is given a choice between a response that produces an immediate, small reward, and a response that produces a larger reward after some temporal delay. If the delay is sufficiently short, normal subjects prefer the larger reward; as the delay increases, preference shifts to the small, immediate reward. People with psychiatric disorders such as attention deficit hyperactivity disorder (ADHD), drug addiction, and pathological gambling, which are associated with increased impulsivity, will discount a delay more readily than controls (i.e., they prefer the immediate reward) [32–33]. Likewise, rats chronically treated with cocaine respond more impulsively than saline treated rats in a delay-discounting procedure for food reward [34]. Interestingly, OFC lesioned rats, rather than behaving more impulsively, favor the larger, delayed reward at delays that make sham-lesioned rats switch their preference to the smaller reward [35]. This result suggests that the role of the OFC may not be to inhibit impulsive behaviors, but may be to assess and update the value of an outcome under changing conditions. The inability to alter behavior despite a decrease in the value of the outcome is reminiscent of habit behavior (see discussion above), and suggests an alternative role for the OFC in guiding behavior.

In addition, impulsivity involves an inability to inhibit prepotent (well-established) motor responses. Differences in response inhibition can be measured using the 5-choice serial reaction time (5-CSRTT), go/no-go, or stop-signal tasks. Humans with impulsive disorders such as ADHD, trichotillomania (repetitive hair pulling), and drug addiction have been shown to have deficits in motor response inhibition in the go/no-go and stop-signal reaction time tasks [4]. The 5-CSRTT allows for measurements of response accuracy, response omissions, premature responses or impulsivity, and perseverative responses. OFC lesioned rats have increased omissions, premature, and perseverative responses, suggesting that the OFC dysfunction plays a role in impulsivity, but also in response flexibility [36]. OFC lesions in the rat also produce

deficits in the stop-signal reaction time task, again implicating the OFC in the inhibition of prepotent motor responses [37].

The most common finding in animals and humans with OFC lesions is a reversal-learning deficit. OFC lesions cause reversals to be acquired more slowly, and involve continued performance of the response that is no longer rewarded [38;39]. This result has often been interpreted as a failure to inhibit prepotent responses, but several lines of evidence suggest that the OFC may actually be important for encoding the outcome of the response (i.e., the presence or absence of the reward) [39]. Thus, the reversal-learning deficit observed with OFC dysfunction could be considered a failure to devalue the reinforcer (that is no longer presented), which could be interpreted as an increase in habitual responding.

Implications

Habits and impulsive behavior may intuitively appear to be on opposite ends of the behavioral spectrum, but the behaviors that define impulsivity and habit have some commonalities. Clinically speaking, the comparison between impulsivity and habit may be much like comparing impulsivity and compulsivity. Compulsivity is epitomized by obsessive-compulsive disorder (OCD) and Tourettes syndrome, where a person feels compelled to perform a behavior in order to relieve anxiety or stress, even if the behavior is inappropriate or counterproductive. These ritualistic behaviors are often described as habitual, and involve dysfunction in OFC [40]. However, the inappropriateness of the behavior, and the inability to inhibit a prepotent motor response also defines impulsivity. Impulsive disorders are often described as having compulsive features. For example, kleptomania and pathological gambling are considered impulsive disorders, yet those afflicted often describe obsessing about stealing or gambling and feeling compelled to do so [41].

Drug addiction also is a disorder that is certainly described as involving an impulsiveness to take drugs (especially in the initial phases of drug-taking), a compulsion to take drugs after chronic use, and the eventual development of a drug habit, characterized by automated responses to take the drug despite its adverse consequences [see review by Schoenbaum and Shaham in this issue 42; 43]. In addition, Diergaarde and colleagues [44] report in this issue that rats with increased impulsivity on the 5-CSRTT and delayed reward task will subsequently show increased motivation to self-administer nicotine and a resistance to stop responding for nicotine in extinction, suggesting that impulsiveness may yield vulnerability toward compulsion and habit ultimately leading to substance abuse disorders. The neural systems regulating impulsive, compulsive, and habitual behaviors likely have some differences; however, there may be overlapping neurobiology (e.g., activation of the OFC) that may explain why several psychiatric disorders have co-morbid impulsive and compulsive features.

Future studies aimed at disentangling the psychological constructs of impulsivity, compulsivity, and habit, and defining their neurobiological underpinnings within corticostriatal circuits will likely reveal a critical role for the OFC in adaptive behavioral regulation, as well as new insights into multiple psychiatric disorders characterized by maladaptive, inflexible, decision-making and response-selection processes. Such a focus has already become an area of intensive research within multiple disciplines relevant to Biological Psychiatry.

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