Are executive function and impulsivity antipodes? A conceptual reconstruction with special reference to addiction

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Received: 7 May 2011 / Accepted: 5 March 2012 / Published online: 24 March 2012
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Abstract

Rationale Although there is considerable interest in how either executive function (EF) or impulsivity relate to addiction, there is little apparent overlap between these research areas.

Objectives The present paper aims to determine if components of these two constructs are conceptual antipodes—widely separated on a shared continuum.

Methods EFs and impulsivities were compared and contrasted. Specifically, the definitions of the components of EF and impulsivity, the methods used to measure the various components, the populations of drug users that show deficits in these components, and the neural substrates of these components were compared and contrasted.

Results Each component of impulsivity had an antipode in EF. EF, however, covered a wider range of phenomena, including compulsivity.

Conclusions Impulsivity functions as an antipode of certain components of EF. Recognition of the relationship between EF and impulsivity may inform the scientific inquiry of behavioral problems such as addiction. Other theoretical implications are discussed.

Keywords Executive function · Impulsivity · Addiction · Drug abuse · Substance abuse · Compulsivity

“Our mind is capable of passing beyond the dividing line we have drawn for it. Beyond the pairs of opposites of which the world consists, other, new insights begin.”

–Hermann Hesse

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Scientific thought often progresses by recognizing heterogeneity in phenomena formally considered homogenous. This evolution sometimes involves identifying small components and conceptualizing how they fit together to produce larger phenomena. Somewhat less frequently, science progresses by recognizing unity between seemingly diverse research areas (Wilson 1998). For example, recognizing similarity in principles common to genetic analysis and information theory led to the contemporary conceptual categories used in genetic studies (Gleick 2011). This paper explores executive function (EF) and impulsivity, two research areas generally considered conceptually distinct, and suggests that they may be antipodes (i.e., widely separated on a common continuum, upon which they are related).

EF has been defined in many ways (Bickel and Yi 2008). For example, some refer to EF as the functions associated with the frontal cortices (Stuss and Benson 1986) whereas others define EF by identifying its components, such as cognitive flexibility and strategic planning (Denckla 1994). Common across these diverse views, however, is the notion that an individual's EF system plays a role in the consideration of, and planning for, their future (Denckla 1994; Dennett 1995). We will employ a definition of EF that organizes and synthesizes the various notions of EF in an understandable and consistent fashion. Specifically, we will define EF as classes of self-directed behavior to change one's future reinforcement (Barkley 1997, 2004).

Impulsivity also has been defined and measured in a variety of ways. This diversity is evident in the classic review of Evenden (1999) on the impulsivity literature, which lists 28 terms that researchers have used to identify varieties of impulsivity. Twenty-eight is clearly an unwieldy number of terms for a single scientific concept. Although there is considerable overlap among the 28 terms, they do not appear to have a single meaning. For our present purposes, we will use the same classic definition used by Evenden (1999). Specifically, impulsivity will refer to actions that appear prematurely expressed, unduly risky, poorly conceived, and result in undesirable consequences (Durana and Barnes 1993).

EF and impulsivity, each defined in numerous ways, have both been important topics in the study of addiction. Addiction researchers have studied impulsivity for many years. This line of inquiry has focused on understanding a variety of the challenging and seemingly irrational behaviors of the addicted. Its considerable scientific achievements include greater understanding of aspects of the limbic and paralimbic brain regions, the role of dopamine, and the development of new procedures and measures. In the process of conducting these studies, however, researchers began to recognize the role of frontostriatal dysfunction in addiction (e.g., Jentsch and Taylor 1999). Unfortunately, the subsequent trend in drug research (with exceptions that we will review below) has been the development of two separate addiction research areas—one examining executive dysfunction and the other addressing impulsivity.

One factor contributing to the development of these distinct areas of addiction research may be the separation of these research areas in the broader, non-addiction, literature. Evidence for the development of the two separate research areas can be obtained from multiple databases. While the expansiveness of the literatures on each topic corresponds to the variety of definition, there is little overlap between these areas (see

Fig. 1 Venn diagrams demonstrating the paucity of overlap between Executive Function and Impulsivity. Proportional diagrams were created using the number of results from “Web of Science” searches on December 19, 2011 using the following search terms: “Executive Function” (14,516), “Impulsivity” (6,732), “Executive Function” and “Impulsivity” (384) “Executive Function and Drug Abuse” (157) “Impulsivity and Drug Abuse” (433) and “Executive Function and Impulsivity and Drug Abuse” (25). Similar results are obtained when conducting searches with these terms in other academic databases (e.g., Pubmed)

Fig. 1). The data portrayed in Fig. 1 could be interpreted to suggest that the notions of executive function and impulsivity are distinct in the sense that there is no basis for comparing them. Alternatively, the apparent distinction between these two research concepts may be due to them being notions at widely separated (opposite) ends of a shared continuum—in other words, antipodes. If the latter interpretation is more accurate, then each of the two research domains could benefit from the interrelation between the two fields, as scientific advances made in one field could benefit the other.

Determining the “opposite” is challenging when faced with terms like EF and impulsivity. How do we know an opposite? Moreover, because there are a variety of specific behaviors that are referred to as EF and/or impulsivity, how do you compare these as single constructs? A fruitful answer may be provided by determining the similarity between certain types of EF and impulsivity; namely, are certain types of executive dysfunction the equivalent of certain types of impulsivity? If a majority of impulsivity types have an antipode in the constellation of EFs typically studied, it may be safe to say what scientists call EF and impulsivity are generally antipodes. To address this, we will generally focus on addiction research to explore whether or not certain types of executive dysfunction and impulsivity are equivalent. Addiction is a research area wherein many studies have examined either executive

dysfunction or impulsivity. By examining those studies, we can understand the similarity between the impaired condition of one category (executive dysfunction) and the affirmative condition of another category (impulsivity). Because we are focusing on addiction, a condition often characterized by impairments in certain functions, our approach is similar to brain studies that impose functional impairments via lesions of the brain.

To assess the equivalence of executive dysfunction and impulsivity, we will first briefly codify: (a) types of EF and impulsivity, (b) the associated brain areas, and (c) studies in these two research domains that examine addicted populations. Second, we will examine the points of convergence and divergence across these two domains. Third, we will conclude by answering the question posed by the title of this paper. Namely, are EF and impulsivity antipodes? Given the vast sizes of these two research domains, reviewing them thoroughly is beyond the scope of this paper. Substantive reviews are available elsewhere (Evenden 1999; Hester et al. 2010). The current paper will instead primarily focus on addiction, because addicted individuals exhibit both impulsivity and executive dysfunction (e.g., Bickel and Marsch 2001; Bickel and Yi, 2008).

Executive function

As described above, EF is defined as behavior that is self-directed toward altering future outcomes (Barkley 1997, 2004). Many actions meet this definition, making the formulation of a comprehensive account of EF challenging. This challenge generally has been met by deconstructing EF into smaller units that are then grouped into larger categories, with the content and organization of these categories varying widely from author to author (e.g., Barkley 1997; Dalley et al. 2011; Miller and Cohen 2001). Despite the varied organization of these individual EFs, the general consensus is that EFs are associated with activity in the prefrontal cortex (e.g., Dalley et al. 2011; Luria 1966; Miller and Cohen 2001; Norman and Shallice 1986; Stuss and Benson 1986).

Classic accounts of EF were largely developed through interaction with clinical populations. For example, through his work with individuals with frontal lobe damage, Luria (1966, 1973) posited that the prefrontal cortex (PFC) was a supervisory attentional system (SAS) that governed the programming, regulating, and verification of behavior. Norman and Shallice (1986) subsequently incorporated this SAS into their account of EF. Observations of executive dysfunction in individuals with frontal damage led Stuss and Benson (1986) to propose a model emphasizing the PFC's role in the drive, control, and sequencing of behavior.

Many influential contemporary accounts emphasize one EF. For example, Stuss and Alexander (2007) emphasized the role of attentional control in energizing, task setting, and the monitoring of ongoing behavior; Baddeley (2003) emphasized the role of working memory; and Barkley (1997, 2004) emphasized the role of inhibition. Other contemporary accounts, however, focus on several EFs. For example, Robbins (1996) focused on planning, working memory, and attentional shifting; whereas Bechara (2005) focused on valuing future events and emotional aspects of decision making.

Recent accounts have also linked executive dysfunction to addiction. For example, George and Koob (2010) liken one's vulnerability to addiction to a breakdown of self-regulation, manifest as deficits in attention, decision making, and responses to reward, emotion, pain, and stress. Others have emphasized the roles of impaired response inhibition and the elevated salience of addiction-oriented cues in phenomena such as craving, loss of control, bingeing and relapse (Goldstein and Volkow 2002). Similarly, Garavan and Hester (2007) emphasized the role of attentional control, inhibitory control, and behavioral monitoring (i.e., error detection) as factors that predispose one to addiction.

With current accounts differing as to which EFs are included and/or emphasized (see Packwood et al. 2011, for an analysis), the adoption of any single account will surely exclude functions central to other accounts. Instead, we have focused on functions common to a variety of accounts. In doing so, we have restricted our focus to EFs central to prominent accounts of executive dysfunction in addiction. Our analysis will focus on attention (Garavan and Hester 2007; George and Koob 2010; Luria 1966, 1973; Norman and Shallice 1986; Stuss and Alexander 2007), behavioral flexibility (Robbins 1996), behavioral inhibition (Barkley 1997, 2004) planning (Robbins 1996), valuing future events (Bechara 2005), working memory (Baddeley 2003), emotional activation and self-regulation (Bechara 2005; George and Koob 2010), and metacognitive processes (Garavan and Hester 2007). These functions will be grouped into three broad categories: (a) the cross-temporal organization of behavior, (b) emotional and activation self-regulation, and (c) metacognitive processes. The procedures used to assess these functions are outlined in "Appendix", and we encourage interested readers to consult texts such as Lezak et al. (2004) for additional methodological information.

Cross-temporal organization of behavior

The *cross-temporal organization of behavior* (CTOB) refers to EFs that organize behavior across time and enhance the consideration of, and planning for, future circumstances.

The CTOB includes several interrelated processes such as attention, behavioral flexibility, behavioral inhibition, planning, valuing future events, and working memory.

Attention refers to concentrating on one aspect of the environment while ignoring others (Barkley 1997), and is measured through vigilance tasks such as the Conners Continuous Performance Task (Conners 2000). Individuals with attentional strength concentrate on a task (e.g., participating in a therapy session) despite potential environmental distractions. Conversely, individuals with attentional deficits are easily distracted by other stimuli, making it difficult to complete required tasks.

Most brain activity can be modulated by attention because attention may be brought to bear arbitrarily on any concept, however abstract it may be. For example, monkeys can be trained to attend to lines of a specific orientation on a computer screen. As they do so, regions in early visual cortex that are sensitive to line orientation show elevated responding (McAdams and Maunsell 1999). If, instead, people must attend to faces, then regions associated with face processing, including the occipital lobe, lingual gyrus, and fusiform gyrus (de Fockert et al. 2001) are modulated. In general, attention is thought to be governed by behavioral goals, which then bias sensory processing. Goals are believed to be maintained by patterns of activity in the dorsolateral prefrontal cortex (DLPFC), which then modulate sensory activity via projections to the posterior parietal cortex (Miller and Cohen 2001). This theory has been supported by many neuroimaging studies (see Cabeza and Nyberg 2000 for a review).

Interestingly, cocaine-dependent/abusing individuals, a population with attentional deficits (Di Sclafani et al. 2002; Hester and Miller 2006; Kalapatapu et al. 2011; Verdejo-Garcia et al. 2006a), have less attention-task-oriented activation in the thalamus and more activation in the occipital lobe and PFC (Tomasi et al. 2007) than do controls. This suggests that the thalamus may be impaired, which makes the occipital lobe and PFC work harder to compensate. In turn, the greater PFC activation may render it less able to participate in other demanding activities (e.g., working memory).

Attention deficits are observed across a range of substance users, including alcohol abusers (Thoma et al. 2011a), amphetamine, and methamphetamine abusers (Iwanami et al. 1995; Johanson et al. 2006; Simon et al. 2000; Verdejo-Garcia et al. 2006b), cocaine-dependent/abusing individuals (Di Sclafani et al. 2002; Hester et al. 2006; Kalapatapu et al. 2011; Verdejo-Garcia et al. 2006c), and smokers (Spilich et al. 1992; Yakir et al. 2007). For example, Levine et al. (2006) compared the neuropsychological performance of HIV-positive patients that used stimulants

(i.e., cocaine and/or methamphetamine) to those that did not use stimulants. Although stimulant-using patients committed more errors of omission and had more varied reaction times on continuous performance tasks, they were not different on measures of intelligence or global cognition. Thus, the cognitive deficits associated with stimulant use were specific to attending. Furthermore, individuals with attention deficit hyperactivity disorder (ADHD; Gunther et al. 2011; Lundervold et al. 2011), the obese (Cserjesi et al. 2009; Maayan et al. 2011), and pathological gamblers (Kertzman et al. 2008) also have difficulties with attention.

Behavioral flexibility is the ability to adjust behavior appropriately in response to changing environmental contingencies (Hanna-Pladdy 2007) and is often measured by tasks such as the Wisconsin Card Sort Task (WCST) or the trail-making task B. Behavioral flexibility may be particularly relevant to addiction because “kicking the habit” requires much more than self-control in the presence of the addictive drug. To remain sober, behavioral flexibility is needed to avoid people, places, and things previously associated with drug use. Individuals with flexible behavior may seamlessly switch from one coping behavior to another as they encounter risky situations, whereas individuals without that flexibility may be unable to prevent or escape circumstances that lead to drug use.

Studies have identified brain regions responsible for behavioral flexibility. For example, studies using transcranial magnetic stimulation (TMS), have found that inhibition of activity in the medial frontal gyrus (MFG) disrupted behavioral flexibility (Moser et al. 2002). Because TMS directly manipulates brain activation, the findings of Moser et al. suggest that the MFG plays a causal role in behavioral flexibility. By contrast, patients with brain lesions in the ventromedial prefrontal cortex (VMPFC) and DLPFC have impaired behavioral flexibility (Bechara et al. 1994, 1997; Fellows and Farah 2005), suggesting that proper functioning of these regions may be necessary for flexible behavior.

Amphetamine abusers (Ornstein et al. 2000), chronic cocaine users (Bolla et al. 1999; Ersche et al. 2008; Fillmore and Rush 2006), marijuana users (Lane et al. 2007), and 3, 4-methylenedioxymethamphetamine (MDMA; von Geusau et al. 2004) users tend to be less flexible in the face of changing contingencies, than do non-using controls. For example, Lane et al. (2007) administered the WCST and a novel test of behavioral adaptability to heavy marijuana users and non-using controls. Similar to the WCST (described in the “Appendix”), the behavioral adaptability test establishes responding based on one set of contingencies before periodically altering them. Heavy marijuana users committed more perseverative errors on the WCST and were less able to adapt their responding to novel contingencies on the behavioral

adaptation test. This dysfunction across two tasks suggests that marijuana users may have a general deficit in behavioral flexibility. Moreover, deficits in behavioral flexibility are related to addiction. For example, Giancola et al. (1996) examined the relation between behavioral flexibility and negative consequences of drinking, as measured by the Drinker Inventory of Consequences. Relative to social drinkers with high (i.e., “good”) scores on the WCST, social drinkers with low scores on the WCST tended to experience more impulse-control-oriented negative consequences of drinking. Additionally, those with ADHD (Brewer et al. 2001; Lawrence et al. 2004; Pineda et al. 1999; Seidman et al. 1997; Shue and Douglas 1992), the obese (Cserjesi et al. 2009; Maayan et al. 2011; Verdejo-García et al. 2009), and pathological gamblers (Forbush et al. 2008; Goudriaan et al. 2006a; Marazziti et al. 2008) also show impairments in behavioral flexibility.

Behavioral inhibition “Behavioral inhibition refers to three inter-related processes (a) inhibition of the initial prepotent response to an event; (b) stopping of an ongoing response which thereby permits a delay in the decision to respond; and (c) the protection of this period of delay and the self-directed responses that occur within it from disruption by competing events and responses (interference control)” (Barkley 1997, p. 67). Behavioral inhibition is typically measured by tasks such as the stop signal reaction time (SSRT) task or go/no-go tasks. Relevant to addiction, an individual might think that joining friends at a bar is a good idea, but inhibit that action to reach his/her goal of remaining abstinent.

Neuroimaging studies have identified numerous brain regions associated with behavioral inhibition. This reflects the multifaceted definition of behavioral inhibition. Stopping an initiated behavior has been closely tied to activity in the right inferior frontal cortex (Aron et al. 2004). Of course, most instances of behavioral inhibition require some combination of inhibitory behaviors. Accordingly, a wider spectrum of brain areas is commonly implicated in inhibition. For example, Cai and Leung (2011) found that in addition to the inferior frontal cortex, the insula was robustly activated during behavioral inhibition tasks. Interestingly, populations with deficits in inhibition show less inhibition-task-oriented brain activation than do controls. For example, Norman et al. (2011) found that decreased activation in the left DLPFC, the right frontal gyrus, right medial gyrus, left cingulate, left putamen, medial temporal, and inferior parietal cortex during behavioral inhibition tasks predicted teenagers’ transition from moderate to heavy drug use. Similarly, obese females had lower levels of inhibition-task-oriented activation in the inferior parietal cortex and the cuneus than did healthy-weight women (Hendrick et al. 2011).

Compared to non-using controls, cocaine users (Colzato et al. 2007; Fillmore and Rush 2002; Kaufman et al. 2003), heroin addicts (Pau et al. 2002), and methamphetamine-dependent individuals (Salo et al. 2005) have deficits in behavioral inhibition. Impairments in behavioral inhibition, however, are not limited to addicted individuals. For example, the time taken to inhibit responding on the SSRT task is higher for recreational cocaine users than for abstinent individuals (Colzato et al. 2007), and this impairment increased as the level of exposure to cocaine increased. Furthermore, those with ADHD (for reviews, see Barkley 1997, 2004; Walshaw et al. 2010), the obese (Maayan et al. 2011; Verdejo-García et al. 2009), and pathological gamblers (Goudriaan et al. 2006b; Kertzman et al. 2008; Roca et al. 2008) also show deficits in behavioral inhibition.

Planning Defined as activities associated with choosing a future course of action, “planning requires the ability to identify and organize the necessary steps required to achieve a goal. These steps can include the ability to conceptualize (look ahead), view one-self and the environment in an objective fashion, generate alternatives, make decisions, and consider sequential and hierarchical ideas” (Hanna-Pladdy 2007, p. 120). Planning is typically assessed using tasks such as the Tower of London, and may be relevant to addiction. For example, aspects of cognitive behavioral therapy require identifying desirable outcomes and developing plans to obtain them. By contrast, poor planning could result in failure to complete therapeutically important tasks such as seeking employment or housing.

The DLPFC is involved in establishing and maintaining plans; for example, patients with damage to the DLPFC are unable to perform basic planning skills such as shopping for a small list of items (Shallice and Burgess 1991). Brain imaging studies suggest the DLPFC coordinates activity in other regions of the brain that subserve attentional and motivational aspects of planning. de Ruiter et al. (2009) implicated the DLPFC the VMPFC, parietal cortex, and striatum in planning. Dockery et al. (2009) manipulated levels of DLPFC activity via transcranial direct current stimulation (tDCS), and improved tower task performance, suggesting that the DLPFC plays a causal role in planning.

Individuals dependent on amphetamines (Ersche et al. 2006), cigarette smokers (Yakir et al. 2007), cocaine and/or heroin abusers (Davydov and Polunina 2004; Fernandez-Serrano et al. 2010) ketamine users (Morgan et al. 2009), and opioid-dependent individuals (Ersche et al. 2006) perform poorer on planning tasks than do non-using controls. Impairments, however, may be most prominent during periods of active drug use. For example, Morgan et al. (2006) compared the Stockings of Cambridge task (a modification

of the Tower Task) performance of 30 frequent ketamine users, 30 infrequent ketamine users, 30 ex-ketamine users, 30 polysubstance abusers, and 30 nondrug-using control subjects. Relative to the control subjects, the frequent ketamine users required significantly more moves to complete the task. By contrast, the polysubstance users', ex-ketamine users', and infrequent ketamine users' performance was similar to that of the control subjects. Additionally, those with ADHD (Nigg et al. 2002), the obese (Lokken et al. 2010), and pathological gamblers (Goudriaan et al. 2006b) also show impaired planning.

Valuing future events The *valuing of future events*, defined as the propensity for future reinforcers to maintain current responding, is often measured through delay discounting tasks. Individuals that value future events often make sacrifices for delayed gains (e.g., education). By contrast, those that do not value future events often engage in activities that provide immediate reinforcers (e.g., drug use) at the expense of their future wellbeing.

Many brain regions may be associated with the valuation of future events. For example, McClure et al. (2004) found elevated activation in limbic and paralimbic regions (i.e., ventral striatum, medial orbitalfrontal cortex, medial PFC, posterior cingulate cortex, left posterior hippocampus) when individuals chose immediate rewards and relatively high levels of activation in prefrontal areas (i.e., left intraparietal cortex, right DLPFC, right ventrolateral PFC, right orbitalfrontal cortex) when individuals chose delayed rewards (see also, Bickel et al. 2009; Kable and Glimcher 2007, 2010; McClure et al. 2007b). Furthermore, applying TMS to the DLPFC (Figner et al. 2010) or lateral PFC (Cho et al. 2010) altered individuals' valuation of future events.

Individuals addicted to alcohol (Petry 2001a; Vuchinich and Simpson 1998), cigarettes (Bickel et al. 1999; Johnson et al. 2007), cocaine (Bickel et al. 2012b; Coffey et al. 2003; Heil et al. 2006), and heroin (Kirby et al. 1999; Madden et al. 1997) all devalue future reinforcers more rapidly than do non-using controls (see Bickel et al. 2012b; Bickel and Marsch 2001; Madden and Bickel 2009). For example, Madden et al. (1997) found that heroin addicts devalued delayed reinforcers at a significantly higher rate than did the non-using controls. Furthermore, those with ADHD (Barkley et al. 2001), obese individuals (Davis et al. 2010; Weller et al. 2008; Zhang and Rashad 2008), and pathological gamblers (Dixon et al. 2003; Mackillop et al. 2006a; Petry 2001b) also discount at higher rates than controls.

Individuals who discount at low rates respond better to treatment (e.g., MacKillop and Kahler 2009; Sheffer et al. 2012; Washio et al. 2011). For example, among cocaine-dependent individuals receiving contingency management, those with lower discounting rates were abstinent for greater

durations compared to those with higher rates (Washio et al. 2011). This relation was significant for individuals receiving low magnitude vouchers, suggesting that higher magnitude vouchers may be needed to treat cocaine dependence in individuals with higher discounting rates.

Working memory "the ability to retain some information active for further use, and to do so in a flexible way allowing information to be prioritized, added or removed" (Bledowski et al. 2010, p. 172), is often measured through tasks such as the *n*-back or O-span. Relevant to addiction, individuals may use working memory in learning new skills during treatment. By contrast, individuals with dysfunctional working memory may be unable to transfer the new skills emphasized during treatment to their long-term behavioral repertoire.

Neuroimaging studies find that the DLPFC, VMPFC, dorsal cingulate, frontal poles, medial inferior parietal cortex, frontal gyrus, medial frontal gyrus, and precentral gyrus, are active during working memory tasks (de Fockert et al. 2001; Glahn et al. 2002). Moreover, MDMA users have more working memory-task-oriented activation in the superior frontal gyrus and its thalamic projections than healthy controls (Moeller et al. 2004); and cocaine-dependent individuals have less caudate, putamen, cingulate gyrus, middle and superior frontal gyri, inferior frontal gyrus pars triangularis and pars opercularis, precentral gyrus, and thalamus working memory-task-oriented activation than healthy controls (Moeller et al. 2004, 2010). Furthermore, TMS and tDCS studies have found that the DLPFC (Mottaghy et al. 2000; Mulquiney et al. 2012) and inferior frontal junction (Zanto et al. 2011) are causally related to working memory.

Alcoholics/alcohol abusers (Beatty et al. 1995; Thoma et al. 2011a), cocaine addicts/abusers (Beatty et al. 1995; Berry et al. 1993; Di Sclafani et al. 2002; Hoff et al. 1996; Kalapatapu et al. 2011; Kubler et al. 2005; Mittenberg and Motta 1993; O'Malley 1990; Verdejo-Garcia et al. 2006a), marijuana users (Block and Ghoneim 1993; Bolla et al. 2002), methamphetamine-using/dependent individuals (McKetin and Mattick 1997; Rippeth et al. 2004; Simon et al. 2000), amphetamine-using/dependent individuals (Ersche et al. 2006; Ornstein et al. 2000), and opioid-using/dependent individuals (Ersche et al. 2006; Ornstein et al. 2000) often perform worse than controls on working memory tests. Moreover, Patterson et al. (2010) demonstrated that reaction times on the *n*-back task predicted cigarette smokers' relapse after 14 days of abstinence. Specifically, after undergoing 14 days of varenicline or placebo treatment, participants were required to smoke for 1 day, after which their abstinence was measured for a week. Individuals in the placebo group that abstained during this 7-day test had significantly lower reaction times on the *n*-back test than those who

relapsed (see also Teichner et al. 2001). Furthermore, those with ADHD (McInerney and Kerns 2003; McInnes et al. 2003; Rucklidge and Tannock 2002; Seidman et al. 1997) the obese (Gunstad et al. 2007; Maayan et al. 2011), and pathological gamblers (e.g., Leiserson and Pihl 2007; Roca et al. 2008) also have impaired working memory.

Emotional and activation self-regulation

The second major type of EF is *emotional and activation self-regulation* (EASR). These functions, which we will define as the behavioral processes involved in controlling affective states, are sometimes considered “hot” EFs (e.g., Bechara 2005; Metcalfe and Mischel 1999; Miller and Cohen 2001; Steinberg 2010; Urcelay and Dalley 2011). “The self-regulatory role of the executive system is stressed here in that emotions, once elicited, come to be moderated or regulated by self-directed, executive actions. Included in the component is also the self-generation of drive or motivational arousal states that support the execution of goal-directed actions and persistence towards the goal” (Barkley 1997 p.74). Hence, EASR involves (1) the processing of emotional information and (2) initiating and maintaining goal-related responding. Individuals proficient in EASR remain calm in the face of confrontation, whereas those with poor EASR “lose control” of their emotions, resulting in poor decision making. EASR is often measured using the Iowa Gambling Task (IGT).

Imaging studies show that IGT performance is associated with activation of the medial PFC (Fukui et al. 2005) and lateral PFC (Hartstra et al. 2010), the ACC and the orbito-frontal cortex (Hartstra et al. 2010). The same areas are implicated in the up- or down-regulation of emotions (Ochsner and Gross 2005). The DLPFC is believed to suppress activity in regions such as the VMPFC and amygdala that are closely associated with the experience of emotion (Ochsner and Gross 2005). This interaction is supported by the observations that individuals with VMPFC lesions perform poorly on the IGT (e.g., Bechara et al. 2003) and that applying tDCS to the DLPFC disrupts IGT performance in older adults (Boggio et al. 2010).

The somatic marker hypothesis posits that the VMPFC is responsible for integrating emotional biasing signals based on previous experience. While performing the IGT, healthy controls develop physiological responses (e.g., galvanic skin response and/or heart rate) in anticipation of choosing decks associated with reward and punishment. These signals (i.e., somatic markers) guide the participant’s current selection based on their previous experiences with the decks. Individuals with impaired VMPFC, however, fail to develop these biasing signals (Bechara et al. 1994, 1996, 1997; Crone et al. 2004; Goudriaan et al. 2006b).

Addicted individuals often show deficits in EASR. For example, IGT performance is impaired among individuals abusing or dependent on alcohol, cannabis, cocaine, MDMA and methamphetamine (Bechara et al. 2001; Fein et al. 2004; Grant et al. 2000; Hanson et al. 2008; Quednow et al. 2007; Rogers et al. 1999; Verdejo-Garcia et al. 2007a; Whitlow et al. 2004), and opioids, as well as individuals with ADHD (Masunami et al. 2009), pathological gamblers (Forbush et al. 2008; Goudriaan et al. 2005; Lakey et al. 2007; Roca et al. 2008), and the obese (Horstmann et al. 2011; Verdejo-García et al. 2009). Furthermore, EASR task performance may predict treatment outcome. For example, Passetti et al. (2008) administered neuropsychological tests (i.e., IGT, the Cambridge Gambling Task, Tower of London, a delay discounting task, and the go/no go task) prior to initiating opioid replacement therapy. Three months into treatment, individuals with substandard scores on the gambling tasks, but not the other measures, were significantly more likely to have continued to use illicit drugs.

Metacognitive processes

The EFs described above are relatively simple classes of behavior. The social contexts in which decisions are made, however, are complex. When interfacing with others, it is important to choose courses of action that are appropriate from both your perspective and theirs. *Metacognitive processes* (MP), which include social cognition and insight, are the processes whereby we discriminate our and other’s perspectives, and act accordingly.

Social cognition, sometimes referred to as theory of mind, is “the ability to correctly attribute mental states such as intentions, thoughts and beliefs to other people” (Pickup 2008, p. 206). Individuals with these skills know when things that they have said upset others, whereas those without these skills may be surprised to hear that they offended others.

Being aware of our thoughts and actions is complex. Traditionally called metacognition, these skills include “awareness, beliefs, and knowledge about one’s self, including abilities, weaknesses, knowledge, skills and personal history” (Wasserstein and Lynn 2001, p. 379). Sometimes called insight (see Goldstein et al. 2009 for a review), these processes are inferred by individuals’ awareness of their errors on a range of cognitive tasks. Because individuals with insight know that certain decisions or behaviors may be maladaptive, they can choose to behave in alternative ways. By contrast, those without these skills may be unaware that their decisions are leading directly to poor outcomes (Goldstein et al. 2009).

MP for perceiving one’s own versus others’ perspectives can be dissociated via fMRI. Specifically, insight is

associated with activation in the insula and ACC (Craig 2009; Hester et al. 2009). By contrast, social cognition is associated with activation in the medial PFC, right superior temporal gyrus, left temporal parietal junction, left somatosensory cortex, and the right DLPFC (e.g., Germine et al. 2012; van den Bos et al. 2011), and impaired social cognition is seen in individuals with VMPFC lesions (Bechara 2004).

Addicted individuals have deficits in these skills (Goldstein et al. 2009). For example, poor scores on the Meta-Cognitions Questionnaire are associated with elevated levels of alcohol consumption (Moneta 2011; Spada and Wells 2005) and cigarette smoking (Spada et al. 2007) in college students. Similarly, alcoholics have deficits in social cognition skills as revealed by their overestimation of the emotions in facial expressions (Clark et al. 2007; Foisy et al. 2007; Oscar-Berman et al. 1990; Philippot et al. 1999; Salloum et al. 2007) and their failure to identify the perspective of cartoon characters (Uekermann et al. 2007). Similar deficits are seen in individuals with ADHD (e.g., Corbett and Glidden 2000; Fine et al. 2008; Semrud-Clikeman 2010).

Conclusions regarding executive function

Overall, executive dysfunctions are clearly evident among the addicted and could contribute to the poor outcomes associated with addiction. For example, the incidence of mild to severe neurocognitive impairments is estimated to be between 50 and 80 percent among individuals with alcohol use disorders (Bates et al. 2006) and cocaine and methamphetamine dependence (Aharonovich et al. 2006; Gonzalez et al. 2004). The prevalence of these EF impairments may provide an explanation for treatment failure among patients (Lundqvist 1995). Indeed, diminished EF may interfere with the ability to assimilate and participate in treatment (Verdejo-Garcia et al. 2004). The inability to inhibit behavior, or having a short temporal perspective may lead addicted individuals to exhibit impatience and frustration with the treatment process and therapeutic sessions. Difficulty in planning may also render patients unresponsive to several aspects of cognitive behavioral therapy (CBT) that require planning and other executive skills. Lapses of memory may prevent patients from implementing CBT strategies designed to avoid relapse, while a lack of cognitive flexibility may make it difficult to implement novel modes of behaving (Lundqvist 2005; Verdejo-Garcia et al. 2004).

The EFs considered above comprise many seemingly interrelated classes of behaviors. In fact, one may posit that these functions represent nested abilities, in which some EFs are dependent upon others (e.g., inhibition is a prerequisite to attention, attention is a prerequisite for working memory,

etc.). This is an interesting prospect, which may flow naturally from current and future data, but is beyond the scope of the present discussion.

Impulsivity

Impulsivities entail behaviors that are prematurely expressed, unduly risky, appear poorly conceived and result in undesirable consequences (Durana and Barnes 1993). These features are all disadvantageous, but impulsive behavior is not always dysfunctional (Conners 2000; Dickman 1990; Gullo and Dawe 2008). The undesirable consequences of impulsivity, however, attract the most attention of impulsivity researchers (Evenden 1999), particularly in addiction (Crews and Boettiger 2009; Perry and Carroll 2008). By focusing on dysfunctional impulsivities, our approach echoes that of most addiction researchers. This section will consider varieties of impulsivity seen as important to understanding human addiction (Crews and Boettiger 2009; Dalley et al. 2011; de Wit 2009; Evenden 1999; Moeller et al. 2001; Perry and Carroll 2008). After first discussing trait impulsivities, we will consider four state impulsivities (i.e., behavioral disinhibition, attention deficit impulsivity, reflection impulsivity, and impulsive choice).

Trait impulsivities

Many types of impulsivity, which are seen as personality traits (i.e., behavioral characteristics that are relatively stable across the lifespan of the individual), have been integrally tied to personality theory (Cloninger 1987; Corr 2004; Dawe and Loxton 2004; Eysenck and Eysenck 1991; Nigg 2000; Pickering and Gray 1999; Whiteside and Lynam 2001; Zuckerman 1994). An ongoing issue in personality theory is the extent to which the existence of a specific trait in an individual's personality destines that person to a particular behavior pattern. Higher scores on trait impulsivity questionnaires distinguish alcoholics (Mitchell et al. 2005), cocaine-dependent individuals (Coffey et al. 2003), MDMA users (Butler and Montgomery 2004; Morgan 1998; Parrott et al. 2000) opioid addicts (Kirby et al. 1999; Madden et al. 1997), stimulant users (Leland and Paulus 2005), and pathological gamblers (Fuentes et al. 2006; MacKillop et al. 2006b) from healthy controls. These findings support characterizing impulsivities as disadvantageous traits. On the other hand, if trait impulsivities were associated only with behaviors that are detrimental to evolutionary fitness, then these traits would have long ago been selected out of our species' genetic makeup. This suggests that variation in trait impulsivities exists within our species' population, and that some variants are functional.

Sensation-seeking (i.e., personal effect of novelty), a trait impulsivity that is often characterized positively, has also been posited as dysfunctional in explanations of addiction phenomena. Individuals' sensitivity to reward may help explain their persistent drug taking despite their awareness of the negative consequences of doing so (Corr 2004; Dawe and Loxton 2004; Gullo and Dawe 2008). For example, Gullo and Dawe (2008) note that adolescents susceptibility to developing drug abuse problems (cf. Chambers et al. 2003) is related to brain development. Specifically, the mesolimbic and orbitofrontal brain regions are staggered in human development, with the mesolimbic system maturing during adolescence and the orbitofrontal system maturing after adolescence. Gullo and Dawe associated this staggered development with age-based differences in the trait measures of "reward drive" and "rash impulsiveness", suggesting that reward drive and rash impulsiveness reflect observations in neuroscience that predict the greater susceptibility of adolescents to substance misuse (see also Bickel et al. 2012c; Steinberg et al. 2008).

Trait impulsivities impact patterns of brain activation. For example, Sripada et al. (2010) found that activation in the medial PFC during a delay-discounting-type task was inversely correlated with scores on the Barratt Impulsiveness Scale. Similarly, Hinest et al. (2011) found that when selecting delayed rewards during delay-discounting-type tasks, activation in the pregenual anterior cingulate cortex and ventrolateral prefrontal cortex was positively correlated with impulsivity on the IVE (Eysenck et al. 1985) whereas venturesomeness scores correlated positively with activation in the right lateral orbitofrontal cortex, subgenual anterior cingulate cortex, and the left caudate nucleus.

Behavioral disinhibition

Behavioral disinhibition defined as the inability to control a response that has already been initiated (Dalley et al. 2011), results in behavior that is "prematurely expressed" and is likely to result in "undesirable consequences." Because drug use is often influenced by powerful internal or external stimuli (e.g., craving, cue reactivity), individuals who can inhibit their responses to these stimuli are more likely to stop using.

Compared to healthy controls, individuals addicted to alcohol (Kaufman et al. 2003; Lawrence et al. 2009; Noel et al. 2007), cigarette smokers (Spinella 2002), cocaine-using/dependent individuals (Fillmore and Rush 2002; Hester and Garavan 2004; Kaufman et al. 2003; Li et al. 2006; Verdejo-Garcia et al. 2007b), and methamphetamine abusers (Monterosso et al. 2005) display behavioral disinhibition. For example, Monterosso et al. (2005) found that SSRT, which measures the time until response inhibition, was significantly longer for methamphetamine abusers than for controls. Similar deficits have been seen in persons with

ADHD (Passarotti et al. 2010) and pathological gamblers (Goudriaan et al. 2006a; Kertzman et al. 2008).

Neuroimaging studies have revealed deficient inhibition-related brain activation in cocaine users compared to drug-naïve controls. Kaufman et al. (2003) found that, compared to non-using controls, cocaine users exhibited less activation in the cingulate, presupplementary motor area, and insula during successful no/go responses and errors of commission. Hester and Garavan (2004) found that cocaine-dependent individuals' compromised ability to inhibit prepotent responses in a go/no/go task was associated with deficient activity in anterior cingulate and right prefrontal cortices. Thus, diminished functionality of the anterior cingulate and prefrontal cortices is currently suggested as the primary neurologic locus of behavioral disinhibition.

Attention deficit impulsivity

Attention deficit impulsivity defined as diminished ability to persist in engagement of relevant rather than irrelevant stimuli, results in behavior that appears "unduly risky, or inappropriate to the situation," and that results in "undesirable consequences".

Addicted individuals often have attention deficit impulsivity, which sometimes interacts with excesses in behavioral disinhibition. For example, Salgado et al. (2009) found that alcohol-dependent patients made more errors of commission on the Continuous Performance Task than healthy controls (see also Rubio et al. 2010). Moreover, those with ADHD, who by definition have attention deficit impulsivity, are disproportionately smokers (Lambert and Hartsough 1998; Laucht et al. 2007). Alcoholics that grew up with undiagnosed ADHD symptoms are more likely to have smoked, be dependent on nicotine, or have impaired concentration during nicotine withdrawal (Heffner et al. 2010). Similarly, Rodriguez-Jimenez et al. (2006) found that pathological gamblers with a history of ADHD had poorer scores on the stop signal task than pathological gamblers without a history of ADHD, although no such differences were seen on a continuous performance task. By contrast, Kertzman et al. (2008) found that pathological gamblers performed poorer than controls on both the go/nogo task and continuous performance task. Kertzman et al. however, did not assess the pathological gamblers childhood ADHD symptoms. Further research is needed to clarify the relation between behavioral disinhibition impulsivity and attention deficit impulsivity in ADHD and in addictive behaviors.

Attentional deficits may impact individuals' perception of drugs. McCloskey et al. (2010) administered three doses of D-amphetamine to nondrug users and asked them about their liking of, and desire for the drug. They found that

individuals who had committed many attentional lapses on a reaction time task in the absence of drug did not show the dose-dependent increases in drug-liking and drug-desiring that were seen in individuals with few attentional lapses. The authors concluded that attention deficits might result in less sensitivity to the euphoria produced by stimulants, driving persons with attention deficits to consume higher amounts.

Individuals with attention deficit impulsivity may have disrupted patterns of brain activation. For example, Schneider et al. (2010) found that, relative to controls, adults with ADHD had abnormally low continuous-performance-task-related brain activity in the caudate nuclei, the anterior cingulate cortex, parietal cortical structures, as well as abnormally high activity in insular cortex. Additionally, Cao et al. (2008) found that boys with ADHD performing a cued target detection task had less activation in the frontal (middle and superior frontal gyrus), parietal (inferior parietal lobe, precuneus) and putamen regions (see Tamm et al. 2006 for comparison) than did healthy controls. Further, Konrad et al. (2007) used fMRI imaging before and after a year of methylphenidate treatment of ADHD to assess the effects of the stimulant on three aspects of attention—alerting, reorienting, and executive control. Matched controls were also imaged, and the ADHD patients stopped medication 1 week before the second imaging session. The treatment was beneficial in that after treatment, the ADHD patients alerting and orienting performance was comparable to that of the control group. Over this 1-year period, however, controls showed a greater functional improvement in task-related brain activation in the temporoparietal junction (critical to reorienting of attention) and the anterior cingulate cortex (critical for executive control) than did the ADHD group. Furthermore, only the ADHD group showed a decrease in activity in the insula and putamen. Konrad et al. suggested that this reflected a reduction of compensatory brain activation in the patient group despite a year-long course of treatment (see Shafritz et al. 2004 for comparison).

Reflection impulsivity

Reflection impulsivity defined as a deficit in the tendency to gather and evaluate information before making a decision (Clark et al. 2009) results in behavior that appears “poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation” and often has “undesirable consequences.” Failure to gather information about drugs may be a common characteristic of behavior patterns that devolve into drug addiction.

The matching familiar figures task (MFFT) is one measure of reflection impulsivity. Poorer performance on the MFFT than controls has been observed in alcoholics (Alterman et al.

1984), current and past cigarette smokers (Yakir et al. 2007), MDMA users (Morgan 1998; Morgan et al. 2006; Quednow et al. 2007), opioid addicts (Cohen et al. 2010), and pathological gamblers (Kertzman et al. 2008). For example, Quednow et al. (2007) found that the MFFT performance in abstinent MDMA users was significantly more impulsive than in abstinent cannabis users or healthy controls, whereas the go/nogo test performance did not distinguish between the groups. This supports the idea that reflective impulsivity is a construct distinct from other varieties of impulsivity. Similarly, Clark et al. (2006) recently used their information sampling task (IST) to assess reflective impulsivity among opioid- and amphetamine-dependents (Clark et al. 2006) and among those populations plus cannabis users (Clark et al. 2009). In each case, the substance users showed higher levels of reflection impulsivity. Furthermore, Lawrence et al. (2009) used the IST to distinguish alcohol-dependent and problem-gambler groups from control subjects.

In the only study relating reflection impulsivity to brain function that we found, Chevalier et al. (2000) investigated the cognitive functioning of children suffering from benign focal childhood epilepsy, which may affect cognitive functions that depend on late development of the frontal lobes. Chevalier et al. found that scores on the MFFT were among the neuropsychology measures that were poorer compared to healthy controls. All of the measures that distinguished participants with childhood epilepsy from controls reflected poor frontal lobe functioning.

Impulsive choice

Impulsive choice can be defined as preference for smaller, sooner rewards in choices between them and larger, later rewards. Because many benefits in life (e.g., gainful employment, pleasing and productive social relations, a healthy diet) can be viewed as larger later rewards, impulsive choices often appear “poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation,” with “undesirable consequences.” This behavior pattern is the converse of that presented in our section entitled “Valuing Future Events.” The measures of impulsive choice and the phenomena in support of this being a variety of impulsivity relevant to understanding drug abuse were therefore presented in that section. To save space, we will not repeat that exposition here.

Conclusions regarding impulsivity

We have (a) noted the remarkable diversity of conceptions of impulsivity, (b) acknowledged that the interest of science is

largely focused on the potentially dysfunctional consequences of impulsive behavior, (c) considered some of impulsivities that have been operationally defined and studied, and (d) considered the brain regions whose activity may subserve these varieties of impulsivity. In all varieties of impulsivity, a behavior-regulating process is terminated “prematurely,” with the result that behavior often appears “poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation” and resulting in “undesirable consequences.” In our consideration of varieties of impulsivity, we found that neural correlates of each variety was constituted of a balance between the activation of mesolimbic dopamine regions of the brain and activation of prefrontal cortical regions of the brain. In varieties of impulsivity, this balance of brain-region activations favors the behavioral output of the mesolimbic regions. The role of the prefrontal brain regions in this balancing act is stated well by Diekhof and Gruber (2010): “prefrontal influences on activation in the NAcc and VTA may decouple behavior from the impact of immediately rewarding stimuli” (Diekhof and Gruber 2010, p. 1492). Or in other words, prefrontal influences on the NAcc and VTA “supervene the reflexive desire to exploit immediately available rewards” (Diekhof and Gruber 2010, p. 1492).

Comparing and contrasting

The impaired EFs and excessive impulsivities described above occur across addictive disorders (e.g., drug addiction, gambling, etc.) and psychiatric conditions (e.g., ADHD, antisocial personality disorder, bipolar disorder), suggesting that they may be trans-disease processes: that is, processes that operate in multiple diseases (Bickel et al. 2012d; Bickel and Mueller 2009). Importantly from this perspective, advances in the understanding a trans-disease process evident in one disorder may be relevant to the understanding and treatment of other disorders. If impaired EFs and/or excessive impulsivities are trans-disease processes, empirical and theoretical advances from one disorder can shape the theoretical lens through which we view other related disorders. For example, there is variability of conceptual approaches in the ADHD field, wherein different researchers view the same impairment as either executive dysfunction (e.g., Barkley 1997, 2004) or as impulsivity (e.g., Malloy-Diniz et al. 2007), and correlations between these constructs have been demonstrated (Malloy-Diniz et al. 2007; cf. Weatherly and Ferraro 2011), suggesting that executive dysfunction and impulsivity may overlap, could inform addiction research. This understanding of the inter-relations between EF and impulsivity may prove beneficial if it were transferred to addiction research.

Comparisons and points of divergence between impulsivity and EF will be considered below. Because our basic concern is to improve the lives of those suffering from addiction, our comparisons of EFs and impulsivities will focus on the changeable (i.e., state) rather than immutable (i.e., trait) impulsivities.

Points of convergence

In this section, (a) definitional and methodological overlap, (b) overlap in the populations affected, and (c) overlap in neurobiological bases of EFs and impulsivities will be explored.

Definitional and methodological overlap Our four types of impulsivity appear to have definitions antipodal to four types of EF. Attention deficient impulsivity (i.e., diminished capacity to persist in engagement with relevant rather than irrelevant stimuli) is the opposite of attention (i.e., concentrating on one aspect of the environment while ignoring other aspects of the environment). Specifically, low levels of the EF “attention” would manifest as attention deficit impulsivity. Similarly, behavioral inhibition and behavioral disinhibition also share definitional features. In fact, behavioral disinhibition impulsivity (i.e., the ability to control a response that has already been initiated) is implicit in the second of the three inter-related processes that were identified in the EF, behavioral inhibition (i.e., stopping of an ongoing response which thereby permits a delay in the decision to respond). Additionally, reflection impulsivity (i.e., a deficit in the tendency to gather and evaluate information before making a decision) is by definition similar to planning (i.e., activities associated with choosing a future course of action) in that gathering and evaluating information are activities entailed in planning. Lastly, impulsive choice (i.e., choice of small, sooner rewards over larger, delayed rewards) is often the result of a subject’s failure to value future events.

EF and impulsivity also entail overlapping measurement. For example, the Conners Continuous Performance Task has been used to measure both attention-deficit impulsivity (Powell et al. 2010; Rubio et al. 2010; Sacco et al. 2005) and attention (Beebe et al. 2004; Bennett et al. 2008; Harrison et al. 2009; Loeber et al. 2008). Similarly, the go/no go task has been used to measure both behavioral disinhibition and behavioral inhibition (Goudriaan et al. 2006a; Monterosso et al. 2005). The methods used to measure reflective impulsivity (i.e., the MFFT and the IST) and planning (e.g., tower tasks), however, are less similar. Specifically, the planning tasks focus on the consideration of multiple steps beyond the present decision whereas the MFFT and IST focus on prematurely making single-step decisions. Lastly, delay-

2010) or decreases (i.e., decreased rates of discounting; Cho et al. 2010) rates of discounting, depending on the parameters of the stimulation. Thus, the PFC appears causally related to both choice impulsivity and the valuation of future events.

Although overlap between the neural substrates of reflection impulsivity and planning may be expected, little data are available regarding this overlap. The finding that patients with frontal damage due to seizures exhibit high levels of reflection impulsivity (Chevalier et al. 2000), however, is consistent with the DLPFC and dorsal medial PFC involvement in planning (de Ruiter et al. 2009). Furthermore, current research does not show direct overlap between the neural substrates of attention and attention deficit impulsivity. These relations may be clarified by future research.

There do not appear to be impulsivity antipodes to working memory, EASR, or metacognitive processes, although dysfunction of each of these EFs is evident in addiction. The PFC involvement in these skills, however, suggests that they may relate to impulsivity. Thus, although these constructs lack distinct antipodes, the patterns of brain activation seen are consistent with the notion of EF and impulsivity as antipodes.

Points of divergence

Although all four state impulsivities considered above have antipodes in EF, there are EFs not paralleled by any type of impulsivity. Notably, there is no impulsivity equivalent to working memory. Interestingly, exposing subjects to working memory training decreases rates of delay discounting (Bickel et al. 2011). The improvement in valuing future events suggests that both working memory and valuing future events are EFs. Conversely, taxing working memory during delay discounting tasks increases discounting rates (Hinson et al. 2003, suggesting a symmetrical effect. With impulsive choice being the opposite of valuing future events, the parallel effect of working memory training suggests that the impulsivity may be subsumed by the EF.

Metacognitive processes and EASR also lack antipodes in impulsivity. Although not part of impulsivity, dysfunction of such processes has been documented in addiction (Goldstein et al. 2009). Moreover, Mischel et al. showed that individuals low in impulsivity (i.e., having greater ability to delay gratification) also have strong metacognitive abilities (reviewed in Mischel and Underwood 1974). Awareness of strong temptations may be central to self-initiating distraction techniques needed to overcome impulsive drives. Such EFs aid in individuals' navigation of the social landscape wherein behavior occurs (e.g., Shoda et al. 1989). Nonetheless, these EFs may explain a broader range of behavioral phenomena than impulsivity.

Behavioral flexibility may be an important point of divergence between EF and impulsivity. Specifically, behavioral

flexibility (i.e., the ability to change behavior appropriately in response to changes in environmental contingencies), may have a more valid oppositional relation to compulsivity (i.e., persistence or perseverance of behavior in spite of nonreinforcement or punishment; Dalley et al. 2011) than to impulsivity (i.e., actions which are poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and that often result in undesirable consequences; Durana and Barnes 1993). Compulsivity differs from impulsivity in that compulsivity entails behaviors that persist without an obvious relation to the overall goal whereas impulsivity entails poorly conceived behaviors that are prematurely expressed and/or unduly risky (Dalley et al. 2011; cf. Franken et al. 2008).

Behavioral flexibility may relate to “response perseveration” and “reversal learning.” In fact, response perseveration is measured by responding on the WCST (e.g., Finn and Hall 2004; Lane et al. 2007; Smillie et al. 2009) or other tasks that require that participants adapt to changing rules (e.g., de Ruiter et al. 2009; Ersche et al. 2008). Thus, response perseveration may be a failure of behavioral flexibility (i.e., a functional antipode). Similarly, studies of reversal learning, sometimes referred to as “cognitive inflexibility” (e.g., Laughlin et al. 2011; Stalnaker et al. 2009; Vocci 2008), entail changing the contingencies that govern reward and punishment (Fellows and Farah 2005; Franken et al. 2008; Koditwakku et al. 2001; Smillie et al. 2009). As the term “cognitive inflexibility” implies, failures in reversal learning can be seen as failures of behavioral flexibility. Although these constructs may be related to impulsivity (Franken et al. 2008), most accounts treat reversal learning and response perseveration as separate from impulsivity (e.g., Ersche et al. 2008; Lane et al. 2007; Smillie et al. 2009).

Although some regard compulsivity as a core component of impulsivity (Fellows 2007; Patterson and Newman 1993), impulsivity is often associated with the positive reinforcement entailed in the early novelty-driven phases of addiction, whereas compulsivity is associated with the negative reinforcement entailed in the later habit-driven phases of addiction (Brewer and Potenza 2008; Dalley et al. 2011; Fineberg et al. 2010; Koob 2009). Because the theoretical relation between compulsivity and impulsivity remains unclear, we have conservatively opted to view these constructs as distinct. If consensus later suggests that compulsivity should be considered as one of the constituent components of impulsivity, then our observation that compulsivity may be the opposite of behavioral flexibility would strengthen our argument that impulsivity and EF are antipodes.

Competing neurobehavioral decision systems theory

The overlapping brain areas associated with the impulsivities and EFs reviewed above are consistent with the competing neurobehavioral decisions systems (CNDS) theory

(Bechara 2005; Bickel et al. 2007; Bickel et al. 2012c; Jentsch and Taylor 1999). The CNDS holds that overt behavior is influenced by the relative activation in two neurobiological systems (i.e., the executive and impulsive systems). The executive system, made up of various areas of the prefrontal cortex, supports deliberative processes such as the valuation of future events. By contrast, immediate reinforcers primarily activate the impulsive system, made up of evolutionarily older limbic and prelimbic structures.

The CNDS theory seemingly indicates that impulsivities and EFs are independent, insofar as they are linked to separate neural systems. These areas, however, are closely and densely interconnected such that, functionally, they act on a continuum defined by the relative activation of two brain systems (Alexander and Crutcher 1990; Haber et al. 2000). In everyday decisions, behavior seems to result from the interaction and integration of these systems in the VMPFC, such that lesions to this region render people severely impaired (Bechara and Damasio 2005).

The continuity between the impulsive and executive systems is observed in discrete choices. Impulsive choice is associated with relatively high levels of activation in the impulsive system whereas the valuation of future events is associated with relatively high levels of activation in the executive system (Hare et al. 2009; McClure et al. 2004). Similarly, attention deficit impulsivity is associated with elevated activation in the caudate nucleus (i.e., impulsive system) and decreased activation in PFC areas (i.e., executive system; Schneider et al. 2010). Correspondingly, attention is associated with PFC activation (Tomasi et al. 2007).

According to the CNDS theory, numerous factors could predispose one to dysregulation between the executive and impulsive systems. First, there could be pre-existing damage to the PFC that could diminish the effectiveness of the executive system. Second, the earlier maturation of the impulsive system could be the reason that drug experimentation begins in adolescence. Third, unfavorable environments, such as growing up in poverty or with considerable stress, adversely affect EF (see Hackman and Farah 2009, for a review).

Figure 3, inspired by a figure in Taub et al. (2002), outlines how drug abuse may interact with these CNDS, leading to addiction. Once a drug reinforcer is received, two additional factors influence responding. First, drugs are intense and immediate reinforcers that interact with and activate the impulsive brain regions. Second, toxicological affects may result from drug consumption.

Feedback and non-feedback functions then result in use-dependent neuroadaptation. Specifically, drugs reinforce drug seeking and consumption, which become increasingly

Fig. 3 Factors influencing the balance of activation between the executive and impulsive systems in drug addiction. The activity of the executive function system and the impulsive system are represented as “Prefrontal” and “Limbic” actions, in balance within the central, receiving component of the model. Five non-feedback factors, three of them independent and two of them in a potential sequence chain, are pictured in the left third of the figure. The rightmost two-thirds of the figure pictures eight independent factors, some of which may interact with each other as feedback mechanisms that result in drug-use-dependent changes in the brain

effective. Concurrently, increasingly ineffective executive system use may be punished. These two processes may result in use-dependent changes in these two brain regions: elaboration of the limbic brain region, and functional atrophy or learned disuse of the executive system. This model is broadly consistent with the reviewed studies that implicate increased impulsive system activation or low levels of executive system activation in executive dysfunction/impulsivity. For example, deficient behavioral inhibition (Norman et al. 2011) or behavioral disinhibition (Hester and Garavan 2004) are linked to depressed executive system activation.

The CNDS view suggests that disrupted regulatory balance between the two decision systems relates more closely to drug reinforcement’s rapid onset than to a drug’s pharmacological actions (e.g., Wise and Kiyatkin 2011). First, rapid onset is evident in most addictive commodities. Second, therapeutic medications for opioid and nicotine dependence have the same pharmacological profile as the drugs abused, except that the onset is slower and the effects last longer. Third, findings from nonhuman’s delay discounting of drug reinforcers are heterogeneous; they do *not* suggest that drug administration, in and of itself, is sufficient to change this discounting (e.g., Harty et al. 2011; but see Pine et al. 2010). Fourth, nonpharmacological stimuli result in addiction-like responding, suggesting that pharmacological effects are not necessary to engender addiction-like behavior.

One implication of viewing impulsivities and EFs as points along the CNDS continuum is that therapeutic approaches deemed effective for the remediation of executive dysfunctions may reduce impulsivities. This may be particularly fruitful because cognitive rehabilitation programs can remediate executive dysfunction in ADHD (e.g., Klingberg et al. 2002), stroke (Westerberg et al. 2007), and aging (Richmond et al. 2011). Furthermore, Bickel et al. (2011) found that training-based improvements in EF (i.e., working memory) generalized to delay discounting (i.e., impulsive choice/valuation of future events).

Conclusions

In this review, we asked if EF and impulsivity are antipodes. Within addiction science, the answer is a qualified yes. We based this answer on five sets of observations. First, each of our four state impulsivities had a definitional overlap with an EF—i.e., (a) attention deficit impulsivity and attention, (b) behavioral disinhibition and behavioral inhibition, (c) reflection impulsivity and planning, and (d) impulsive choice and valuing future events. Second and relatedly, compulsivity, which many addiction researchers frequently treat as distinct from impulsivity (Dalley et al. 2011), appears to be the antipode of behavioral flexibility. Third, operational measures of EFs and impulsivities also are similar. Fourth, the populations with impulsivity also exhibit executive dysfunction. Fifth, the same brain regions are associated with impulsivity and with executive dysfunction.

Our answer is qualified for two reasons. First, there are EFs (e.g., working memory, metacognition, and EASR) that have no antipode in impulsivity. Second, unlike impulsivity, few speak of EF in terms of traits versus states. Whether related constructs such as IQ should be considered a trait (e.g., Shamosh et al. 2008) or a state (e.g., Muraven and Baumeister 2000), however, is still debated. Hence, it is unclear how much we must temper our conclusions due to this lack of “trait EFs.”

There are three implications of the qualified antipodal relation between impulsivity and EF. First, given that all state impulsivities are encompassed within EF, we could consider eliminating state impulsivity and merely refer to the various executive dysfunctions. Second, the subcomponents outlined above could be explicitly identified as points along a continuum ranging from impulsivity to EF. The CNDS view of addiction (Bickel et al. 2007), reviewed above, may serve as a conceptual grounding for this continuum. This theory suggests that there are two neurobehavioral decision

systems—an impulsive decision system embodied in the limbic and paralimbic brain regions, and an executive decision system embodied in areas of the prefrontal cortices. This model suggests that addiction results from the impulsive decision system exhibiting more control than the executive system (Bechara 2005; Bickel et al. 2007, 2012a, b; McClure et al. 2004, 2007a). Thus, the impulsivities we observe in addicted individuals may result from a failure or dysfunction of the executive system. Third, we could imbed both conceptual categories as examples of a different conceptual category. The notion of reflection impulsivity intimates that all failures of self-regulation may be grounded upon the failure to process information (Clark et al. 2006). We could consider placing state impulsivity and all of EFs into a conceptual category of information processing. This would expand the influence of computational theory (e.g., Niv and Montague 2008) and thereby extend the possible purview of information theory into still another domain (Gleick 2011).

Our success in modifying EF/impulsivity should guide our adoption of EF and/or impulsivity as our conceptual framework. If the techniques such as training EFs (Bickel et al. 2011) medication, or technologies such as TMS, strengthen the executive system, then addiction-related problems should be viewed as examples of executive dysfunction. Conversely, if specific therapies only affect impulsivity (e.g., by *not* also increasing EF or activity in the PFC), then these behavioral challenges should be framed as “impulsivity.”

Regardless of how our conceptualization evolves as a practical result of this review, we need to recognize that a strong component of science is continuity. Scientists today stand upon a foundation built from of prior results and advances as they plan and conduct future studies to understand their subject matter. Unfortunately, given that conceptual similarities of the EF and impulsivity concepts are little recognized, these scientists may stand upon considerably narrower foundation than they could if they recognized that EFs and impulsivities operate as antipodes. We hope that this paper will broaden the conceptual foundation of research conducted in both domains in general, and within the study of addiction, specifically.¹

Acknowledgments This work was funded by NIDA grants R01 DA 12997, R01 DA 024080, R01 DA 024080-02S1 (NIAAA), R01 DA 030241, R01 DA 022386. The authors would like to thank Patsy Marshall for assistance with manuscript preparation.

¹ When describing other researchers' findings, we have made an effort to retain language consistent with that used in the original report.

Appendix

Table 1 Outline for the procedures used to assess functions

Description of assessment	Dependent measure(s)	Exec. function measured (e.g., publication)	Impulsivity measured (e.g., publication)
<p><i>Conners continuous performance task</i> (CCPT; Conners 2000). A computer-based task that briefly displays various individual letters separated by brief interstimulus intervals. The subject's task is to press a key or click a mouse button as quickly as possible when a previously specified target letter appears in the series. In one version of the task, the target is defined as "any letter other than X". In another version of the task, the target is the appearance of a two-letter sequence (e.g., "X" following an "A")</p> <p><i>Simple reaction time test</i> (McCloskey et al. 2010). Subjects are required to press a key as quickly as possible upon computer-screen presentation of a symbol, which may happen after various inter-stimulus interval lengths have passed. For each subject, the mean deviation of the reaction times from the modal reaction time is calculated</p> <p><i>Trail making task, part A</i>. This task (Bowie and Harvey 2006) presents to the subject a page of paper with numbers from "1" through "25" randomly positioned on the page. The subject is required to as quickly and accurately as possible draw lines that connect the numbers in sequence, thus making a trail that begins at "1" and ends at "25"</p> <p><i>Trail making task, part B</i>. The task (Bowie and Harvey 2006) is similar to part A, except that the subject is presented with a page on which the numbers "1" through "13" and the letters "A" through "L" are randomly positioned on the page. The task requires the making of a trail that alternates numbers and letters in ascending sequence (e.g., "1" to "A" to "2" to "B" to "3" to "C" to "4" and so forth)</p> <p><i>Digit span tasks</i> (Miller 1956). These tasks present in each trial a sequence of individual digits (letters and/or numbers) aurally or visually (on a computer screen) with one second between digits. The subject is required to repeat back the sequence in the order presented. Across trials, the number of digits in the presented sequence is increased. The subject's digit span is the maximum number of digits that the subject can repeat back without a mistake on 50 % of trials</p> <p><i>Wisconsin Card Sorting Test</i> (WCST; Berg 1948). This task first shows to the subject a number of stimulus cards with symbols on them. The symbols on a card are all the same per card, but they can vary across cards on the dimensions of their shape, their color, or the number of them on the card. The subject is then presented individual cards and required to place each card into one pile. The criterion (e.g., color versus number versus shape) for correct placement of cards into categories is determined by the procedure. It is initially unknown to the subject; however the subject is given immediate feedback as to whether each particular placement is right or wrong. During the course of the test, the rules that determine whether a card placement is correct are occasionally</p>	<p>Reaction time, # of errors of omission, # of errors of commission</p> <p>Proportion of unusually long reaction times</p> <p>completion time, # of errors</p> <p>Completion time, # of errors</p> <p># of digits in maximum span</p> <p>Categories achieved, trials needed, incorrect matches, perseverative errors</p>	<p>Attention (Kertzman et al. 2008)</p> <p>Spencer et al. 2009</p> <p>Attention (Kalapatapu et al. 2011)</p> <p>Behavioral flexibility (Di Scialfani et al. 2002)</p> <p>Attention (Di Scialfani et al. 2002) working memory (Patterson et al. 2010)</p> <p>Behavioral flexibility (Verdejo-Garcia et al. 2006a)</p>	<p>Attention deficit (Salgado et al. 2009)</p> <p>Attention deficit (McCloskey et al. 2010)</p>

Table 1 (continued)

Description of assessment	Dependent measure(s)	Exec. function measured (e.g., publication)	Impulsivity measured (e.g., publication)
<p>changed, requiring the subject to learn the new rules for matching new cards to the correct category</p> <p><i>Stop signal reaction time task</i> (e.g., Loeber and Duka 2009). The task entails many trials and a “go signal” is presented on every trial, so that the required “go response” to it becomes prepotent. A “stop signal” follows the “go signal” in a small proportion of trials (“stop trials”). The individual is required respond to go signals as quickly as possible but also to stop the go response as quickly as possible, or emit a different “stop response,” in trials in which a stop signal appears. Although the procedure assesses the absence of a response (i.e., not responding), the capability to inhibit responding is generally greater with shorter latencies between the go and stop signals. The procedure titrates to a measure of this latency value at which the subject successfully inhibits the response on 50 % of stop trials; this is the stop signal reaction time (SSRT) for the individual subject. Shorter SSRTs indicate a weaker inhibitory repertoire</p> <p><i>Go/nogo test</i>. (e.g., Patterson et al. 1987). In this task various stimuli, is/are presented to the subject (for humans, often in a discrete trial format). Responding in the presence of some specified stimuli is reinforced (“go” stimuli) and responding in the presence of other stimuli goes unreinforced or is penalized (“nogo” stimuli). The subject is required to respond as quickly as possible. Go stimuli may be presented in a greater proportion than nogo stimuli so that responding to go stimuli is prepotent</p> <p><i>Stroop Task</i> (Stroop 1935). This task entails different conditions in which the subject is asked to correctly respond as quickly as possible to stimuli presented in a series. Dependent measures are taken within conditions and compared across conditions. In some conditions, the correct response is unchallenging—to state the color of a presented geometric form, for example. In other conditions, the presented stimuli have features which cognitively interfere with the correct response o the stimulus. In the classic version of this task, for example, the correct response is to state the ink-color of a presented stimulus; however, the stimuli are words that refer to colors and the words are printed using colors that are incongruent with the semantic meaning of the word (e.g., the word “RED” printed in blue ink). Performance scores in interference conditions are lower than in non-interference conditions. In modified versions of the task, interference is introduced to some conditions by other means. For example, if consumption of a particular drug by the subject is being investigated, words with semantic meanings related to that drug may be used in the interference condition</p> <p><i>Tower tasks</i> (Kaller et al. 2011; Sullivan et al. 2009). These tasks require the subject to solve puzzles involving the rearrangement of geometric forms in the context of rods or pegs onto which the geometric forms may be placed. In the task, predefined starting configurations are presented, rules for allowable movements of the forms are stipulated, and terminal configurations are provided as puzzle goals. For example, the Tower of Hanoi test consists of three rods, and a number of disks of different sizes, which can slide onto any rod. A trial in the assessment begins with the subject being shown an ending configuration, which has the disks arranged on one rod with smaller disks always positioned</p>	<p>SSRT</p> <p># of errors of commission (“false alarms”), # of errors of omission, reaction time</p> <p>Completion time, # of stimuli responded to in a fixed duration, # of errors</p> <p>Completion of task within time limit, time to complete task, # of rule violations, # of moves required</p>	<p>Behavioral inhibition (Colzato et al. 2007)</p> <p>Behavioral inhibition (Verdejo-Garcia et al. 2006a)</p> <p>Behavioral inhibition (Rippeth et al. 2004)</p> <p>Planning (Ersche et al. 2006)</p>	<p>Behavioral disinhibition (Fillmore and Rush 2002)</p> <p>Behavioral disinhibition (Spinella 2002)</p> <p>Behavioral disinhibition (Hester et al. 2006)</p>

Table 1 (continued)

Description of assessment	Dependent measure(s)	Exec. function measured (e.g., publication)	Impulsivity measured (e.g., publication)
above larger disks. Then a beginning configuration is presented, in which disks are arranged on one or more rods, always with smaller disks on top of larger disks. The subject is instructed to rearrange the disks from the starting configuration to the ending configuration as quickly as possible. Difficulty of a trial is function of the number of disks and their positions in the starting configuration. The following rules must be obeyed to avoid "violations": (a) only one disk may be moved at a time, (b) each move consists of taking the upper disk from one of the rods and sliding it onto another rod (on top of the other disks that may already be present on that rod) and (c) no disk may be placed on top of a smaller disk			
<i>Delay discounting assessments.</i> These assessments obtain the subject's valuation of a larger-magnitude reward whose receipt is to be delayed. An assessment procedure obtains one such subjective evaluation for each of numerous delays until the larger-magnitude reward. From these several subjective values, a discounting function and discounting rate can be determined. In a commonly used behavioral delay-discounting assessment procedure, the subject is asked in each trial a question about their preference for a smaller-sooner (SS) versus a larger-later (LL) reward. Across trials, the SI reinforcer magnitude is adjusted so that the procedure ultimately determines the smallest SI reinforcer for which the subject is indifferent between the two reinforcers (i.e., the indifference point).	Delay discounting rate (<i>k</i>)	Valuation of future events (Bickel et al. 2011)	Impulsive choice (Madden et al. 1997)
<i>N-back tasks</i> (Kirchner 1958; Kane et al. 2007). These tasks present a sequence of stimuli to the subject, and require that the subject indicate when the current stimulus matches the stimulus presented <i>n</i> steps earlier in the sequence (i.e. the current stimulus is a "target" stimulus). The subject is typically required to emit either of two responses indicating "yes" or "no" regarding a match, and to do so as quickly as possible. The task is more difficult as the parameter <i>n</i> is increased.	<i>N</i> , targets correct, nontargets correct, reaction time	Working memory (Verdejo-Garcia et al. 2006a)	
<i>Gambling tasks</i> (Verdejo-Garcia and Bechara 2009; Rogers et al. 1999). These tasks measure the effects of risk-taking. For example, the Iowa Gambling Task presents four virtual decks of cards on a computer screen. Subjects are told that each time they choose a card they will be told of the same game money they won; however, some choices also result in the announced loss of money. Each of the decks has a different distribution of gains and losses over time (i.e., some are "good" decks and some are "bad" decks). Subjects can choose which deck to play over the course of numerous trials	Relative number of advantageous deck selections compared to disadvantageous deck selections	Emotional activation and self-regulation (Lehto and Elorinne 2003)	
<i>Tests of social cognition</i> "theory of mind". These processes are assessed via various tests, such as the first- and second-order false belief stories (Wimmer and Perner 1983), the Faux Pas test (Baron-Cohen, 1999), Happe's stories (O'Hare et al. 2009) the Mind in the Eyes task (Baron-Cohen et al. 2001), and Cartoon's tasks (Corcoran et al. 1997). For example, one cartoon task presents the initial three frames of a cartoon sequence and requires that the subject choose the fourth frame. This task requires that the subject understand the way one character would react to the other character's affective state (e.g., Sebastian et al. 2011)	Age-appropriate social acceptability of response to stimuli	Social cognition (see description of assessment)	
<i>Assessments of metacognition.</i> Metacognition (cognition about thinking itself) is typically inferred from responses on questionnaires, such as the Thought Control Questionnaire (Wells and Davies 1994), the Meta-Cognitions Questionnaire (Cartwright-Hatton and Wells 1997), and the	Subscores and total score	Metacognitive function (see description of assessment)	

Table 1 (continued)

Description of assessment	Dependent measure(s)	Exec. function measured (e.g., publication)	Impulsivity measured (e.g., publication)
<p>Short Meta-cognitions Questionnaire (MCQ-30; Wells and Cartwright-Hatton 2004). For example, the MCQ-30 (Wells and Cartwright-Hatton 2004) measures individual differences in maladaptive metacognition. It yields five subscores and a total score that reflect maladaptive cognitions such as lack of confidence in cognitive abilities and inability to control emotions regarding cognitive abilities</p> <p><i>Matching familiar figures test</i> (e.g., Zehnick et al. 1972). In the MFFT, the subject is presented a standard picture of a familiar item (e.g., a bicycle) and six other pictures, five of which are similar to the standard and one of which is identical. The participant is required to identify the identical match to the standard. When errors are made, this is indicated to the subject and the subject is allowed to respond to the same template again. Low accuracy and short latency indicate reflection impulsivity</p> <p><i>Information sampling task</i> (Clark et al. 2009). This computerized task commences with the subject being presented a 5 X 5 grid of gray boxes. The gray color initially displayed on each box hides one of two colors, yellow or blue, that can be revealed by touching the box. Removal of the gray from all the boxes would reveal a random assortment of yellow and blue cells making up the 25 cells of the grid. Touching a gray box turns the cell either yellow or blue, thus providing the subject with one item of information. The required task for the subject is to decide which of the two colors is in the majority for that trial of the test; the subject indicates their decision by touching a cell of that color. The subject is able to “open” as many of the boxes as they wish before indicating their decision. Opening higher numbers of boxes indicates reflectiveness while opening fewer boxes shows impulsiveness</p> <p><i>Barratt impulsivity scale</i> (BIS-11; Patton et al. 1995). This questionnaire assessment yields an overall impulsiveness score and three subscores which have been labeled attentional impulsiveness, motor impulsiveness, and non-planning impulsiveness</p> <p><i>Impulsivity-Venturesomeness-Empathy Scale</i> (IVE; Eysenck et al. 1985). This questionnaire assessment yields three scales—impulsiveness, venturesomeness, and empathy</p> <p><i>SSS-V Sensation Seeking Scale</i>. This subscale of the Zuckerman-Kuhlman Personality Questionnaire (SSS-V; Zuckerman 1994; Zuckerman et al. 1993) is comprised of four subscales—thrill and adventure seeking, experience seeking, disinhibition, boredom susceptibility</p>	<p>Accuracy in selecting exact match, latency to respond</p> <p># of boxes opened before deciding, latency to decision</p> <p>Subscores and overall score</p> <p>Impulsivity scale score</p>	<p>Exec. function measured (e.g., publication)</p>	<p>Reflection impulsivity (Alterman et al. 1984)</p> <p>Reflection impulsivity (Clark et al. 2006)</p> <p>Trait impulsivity (Kirby et al. 1999b)</p> <p>Trait impulsivity (Madden et al. 1997)</p> <p>Thrill and adventure seeking, experience seeking, disinhibition, boredom susceptibility (Steinberg et al. 2008)</p>

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