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Reward drive and rash impulsiveness as dimensions of impulsivity:
Implications for substance misuse

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Key words: impulsivity, reward, personality, substance abuse, drugs

Abstract

One of the primary personality dimensions or traits that has consistently been linked to substance abuse is impulsivity. However, impulsivity is not a homogenous construct and although many of the measures of impulsivity are correlated, the most recent review of published factor analytic studies has proposed two independent dimensions of impulsivity; reward sensitivity, reflecting one of the primary dimension of J.A. Gray's personality theory, and rash impulsiveness. These two facets of impulsivity derived from the field of personality research parallel recent developments in the neurosciences where changes in the incentive value of rewarding substances has been linked to alterations in neural substrates involved in reward seeking and with a diminished capacity to inhibit behaviour due to chronic drug exposure. In this paper we propose a model that integrates the findings from research into individual differences with recent models of neural substrates implicated in the development of substance misuse.

Key words: impulsivity, substance abuse, dopamine, personality, reward

The extent to which personality traits may contribute to the development and maintenance of substance use disorders has been a vexed issue for many years in the addictions field. What began as a somewhat simplistic search for an addictive personality has been substituted by a model in which personality traits are considered as one of many risk factors that play a role in the development of substance misuse. One trait that has been the focus of research is broadly termed “impulsivity” and typically refers to the tendency to engage in behaviour that involves rashness, a lack of foresight or planning, or as a behaviour that occurs without reflection or careful deliberation. However, other theorists such as J. A. Gray have used the term to describe individual differences in the sensitivity to signals of reward. It would appear that impulsivity is not a homogenous construct but consists of at least two facets that in turn relate to broad conceptualisations of impulsivity defined as “rash impulsive” behaviour and heightened “sensitivity to reward”.

In parallel with the refinement of the nature and role of the construct of impulsivity in personality research generally, and substance abuse specifically, there has been an expansion in research focusing on the neurological processes and underlying mechanisms involved in substance use. In this paper a synthesis of these research literatures is provided and a model proposed that may be testable in clinical research.

Impulsivity and substance use

There are a number of different personality scales that measure a range of behaviours that are generally termed “impulsive” such as novelty seeking, behavioural undercontrol and disinhibition. The relationship between these measures and substance misuse has been investigated in a number of cross-sectional studies, and despite the variability in samples and the diversity in measures of impulsivity, a clear relationship between impulsivity and substance use has been consistently found (Baker & Yardley, 2002; Cloninger, Sigvardsson, & Bohman, 1988;

Johnson, Turner, & Iwata, 2003; Jorm et al., 1999; McGue, Iacono, Legrand, Malone, & Elkins, 2001; Shillington & Clapp, 2002; Simons & Carey, 2002; Soloff, Lynch, & Moss, 2000). For example, cross-sectional studies comparing substance misusers (typically alcohol) with non-clinical groups find that substance misusers score higher on measures of impulsivity such as novelty-seeking, sensation-seeking, Eysenck's impulsiveness scale (I₇) and a composite measure of "behavioural undercontrol" (which includes measures of novelty-seeking, extraversion, psychoticism, and the I₇; Battaglia, Przybeck, Bellodi, & Cloninger, 1996; Grau & Ortet, 1999; Pidock, Fischer, Forthun, & West, 2000; Sher, Bartholow, & Wood, 2000; Sher, Wood, Crews, & Vandiver, 1995; Wills, Vaccaro, & McNamara, 1994). There is also evidence from several prospective studies in which impulsiveness measured in childhood has been linked to the development of adult substance use disorders (Howard, Kivlahan, & Walker, 1997; Masse & Tremblay, 1997; Tarter, Kirisci, Habeych, Reynolds, & Vanyukov, 2004). Table 1 provides a brief overview of these studies with a summary of measures used and key findings.

Insert Table 1 about here

Although there are relatively fewer studies, the general pattern of heightened impulsivity in substance misusers is also found when comparing people with substance misuse and other disorders. For example, traits defined as impulsive or novelty-seeking have also been found in eating disordered women, most notably among those characterised by bulimic behaviour (Claes, Vandereycken, & Vertommen, 2002; Fassino et al., 2002; Vervaet, Audenaert, & van Heeringen, 2003). Women with bulimia who also misuse substances show even greater levels on measures related to impulsivity (e.g., Novelty Seeking) than women with bulimia only (e.g., Bulik & Sullivan, 1998; Bulik, Sullivan, Carter, & Joyce, 1997; Bulik, Sullivan, McKee, & Weltzin, 1994; Kane, Loxton, Staiger, & Dawe, 2004). Similar findings from cross-sectional studies on personality and comorbid substance use and schizophrenia have been reported with impulsivity

and sensation seeking associated with a lifetime history of substance abuse or dependence (primarily alcohol and cannabis use) amongst patients with psychotic disorders (Dervaux et al., 2001; Gut-Fayand et al., 2001; Liraud & Verdoux, 2000; Van Ammers, Sellman, & Mulder, 1997).

Thus, it would appear that at least one risk factor for the development and maintenance of substance misuse problems is a personality style characterised by heightened impulsivity. However, there is a growing recognition that definitions of impulsivity are many and varied. In part the model of personality from which the construct is drawn influences the definition of impulsivity. For example, impulsiveness is a core dimension within theoretical frameworks proposed by Eysenck, Cloninger, and Gray (see Dawe & Loxton, in press). There are, however, a number of measures of impulsivity that are not conceptually linked to a particular personality theory (e.g., Zuckerman's Sensation Seeking Scale; Zuckerman, Eysenck, & Eysenck, 1978).

Theoretical models that have influenced the measurement of impulsivity

Many of the measures that have been used in the studies cited above have been derived from models of personality that propose behaviour as being regulated by two (or more) independent systems (e.g., Eysenck, Cloninger, Gray, and to a lesser extent Zuckerman). In each of these models one system is associated with avoidance behaviour (or behavioural inhibition) whilst the other is broadly associated with appetitive motivation and approach behaviour (Carver, Sutton, & Scheier, 2000). Each of the two systems is proposed to have a different neural substrate and to reflect the broad personality traits of anxiety and impulsivity, respectively. Of particular relevance to the current review is Gray's theory in which two interacting systems of behavioural inhibition and approach correspond to the dimensions of anxiety and impulsivity (Gray, 1970; 1975; 1987a; 1987b; Gray & McNaughton, 2000). The former is believed to underlie activity in a conceptual brain system, referred to as the Behavioural Inhibition System (BIS) and reflects individual differences in reactions to conditioned aversive stimuli. Those with a more reactive BIS

are more likely to inhibit approach behaviour that is accompanied by subjective feelings of anxiety/frustration (Gray, 1987b). Gray and McNaughton (2000) propose that the major brain structure underlying the BIS is the septo-hippocampal system comprising the hippocampus proper, dentate gyrus, entorhinal cortex, subicular area (subiculum), posterior cingulate cortex, and the septum-diagonal band complex.

The other system, proposed to underlie the personality trait of impulsivity, is the Behavioural Approach System (BAS). The neural substrate of BAS involves the dopaminergic systems, particularly the mesolimbic dopaminergic pathways. It should be noted that this pathway is not linked solely to positive incentive motivational effects (i.e., approach behaviour), but responds equally to aversive stimuli that require goal directed behaviour (see Pickering & Gray, 1999). In essence, individuals with high BAS sensitivity are more likely to engage in approach and active avoidance behaviour, and to experience greater positive affect in situations containing cues for reward. The neural substrate of the BAS shares many similarities to neural pathways underlying the acutely reinforcing effects of natural reinforcers such as food, sex, and drugs of abuse. Further, the dopamine circuits have been found to activate in response to conditioned cues of reward, prior to the consummation of reinforcing substances (Childress et al., 1999).

Facets of Impulsivity – evidence for a two-factor model.

Measures of anxiety and behavioural inhibition, developed by Cloninger, Eysenck and Gray, appear to tap a single construct and are almost always highly correlated when used across different samples (e.g., Caseras, Avila, & Torrubia, 2003). However, measures of impulsivity or approach behaviours do not provide such uniformly consistent results. There have been a series of factor analytic studies in which measures derived from each of the theorists listed above have been compared to other measures of impulsivity with most studies supporting a two-factor structure. Scales such as Eysenck's Impulsivity scale - the I₇, Cloninger's Novelty Seeking scale and other measures of impulsivity (e.g., Zuckerman's Sensation Seeking Scale, Barratt Impulsiveness Scale;

Patton, Stanford, & Barratt, 1995; Zuckerman et al., 1978) form one domain best described as a tendency to act rashly and without consideration of consequences (Caseras et al., 2003; Miller, Joseph, & Tudway, in press; Quilty & Oakman, in press). Measures of Gray's behavioural approach or impulsivity dimension such as Carver and White's (1994) BIS/BAS Scales (BAS-Drive, BAS-Reward Responsiveness) and Torrubia, Avila, Molto, and Caseras' (2001) Sensitivity to Reward scale (SR) from the Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ), load most strongly on a separate factor. Gray's BAS is proposed to reflect individual variation in sensitivity to rewarding stimuli in the environment. It has, in fact, been argued that the term impulsivity, which has been used to describe this dimension, is a misnomer and that the BAS is better conceptualised as an index of "reward sensitivity or drive" (Dawe & Loxton, in press). This both accords with the findings from factor analytic studies and with the items from each of the measures considered thus far.

However, there are some inconsistencies within the factor analytic studies, with scales such as Carver and White's (1994) Fun Seeking scale, one of the three BAS subscales, correlating with measures that load onto the two factors of rash impulsivity and reward sensitivity. However, while loading on both factors, the evidence to date suggests it to be more closely aligned with rash impulsiveness than reward drive (Caseras et al., 2003; Miller et al., in press; Zelenski & Larsen, 1999). Further, scores on Cloninger's Reward Dependence scale generally fail to load on either domain. We have noted previously that this scale assesses dependence on social approval, rather than a wider range of reinforcing stimuli (Dawe & Loxton, in press).

The neurobiology of impulsivity and substance misuse

The investigations of facets of impulsivity in the field of personality research have occurred somewhat independently of a body of research investigating underlying neural processes implicated in substance misuse. There has been a considerable research effort directed towards

identifying brain systems that mediate the reinforcing effects of addictive drugs. These pathways include dopaminergic projections from the ventral tegmental area and substantia nigra to the nucleus accumbens and the rest of the striatum, plus glutamate inputs from the prefrontal cortex, amygdala and hippocampus (see Robinson & Berridge, 2003 for a review). Long term exposure to drugs has been found to cause permanent changes to these pathways, which in turn later affect a range of brain functions including the reinforcement value of drug-related cues, hedonic drug effects, incentive motivation, drug cravings and executive functioning (e.g., Jentsch & Taylor, 1999; Kelley & Berridge, 2002; Koob & Le Moal, 1997; Robinson & Berridge, 2003). A number of researchers in the neurobiology field have proposed models of addiction that incorporate two interrelated neural processes. Key amongst these are Jentsch and Taylor (1999), Robinson and Berridge (2003) and Goldstein and Volkow (2002). Although these researchers have typically focussed on a specific site of drug action, they all refer to the interplay of neural circuits involved in acute and chronic drug use. We review the commonalities in their models below.

Incentive-salience and the dopaminergic pathways

The first process identified in each of these models involves an increase in the salience of the rewarding and/or reinforcing quality of the substance following an initial period of use. This occurs by two separate but related mechanisms. In the first, drug use produces a sensitisation of the mesolimbic dopaminergic system that results in an increase over time in the incentive-value of the drug itself and drug-related conditioned stimuli. This involves an increase in “wanting” drugs that occurs through a sensitising or hyper-responsivity of motivational processes to drug cues (Robinson & Berridge, 2003). This is possibly the result of hyperactivity in dopaminergic projections from the ventral tegmental area to the nucleus accumbens (Jentsch & Taylor, 1999). The second mechanism involves the “liking” or hedonic experience associated with drug use and is mediated by separate neural systems, notably the opioid receptors within the medial caudal portion of the nucleus accumbens shell (Robinson & Berridge, 2003) and occurs due to changes in

neural systems underlying associative learning (i.e., hyperactivity in dopaminergic projections from the ventral tegmental area to the amygdala; Jentsch & Taylor, 1999).

There is now considerable evidence that dopamine activation in the nucleus accumbens plays a specific role in the reinforcement value, or “attention grabbing” qualities of rewarding stimuli and events, and related cues (Kelley & Berridge, 2002). Specifically, Robinson and Berridge (2003) argue that it is the reinforcement value, rather than the hedonic effects of drug administration that contributes to drug dependence, cravings and relapse. Recurrent drug use sensitises the mesolimbic dopamine circuits to learned associations between drug cues and acute drug effects, making drug cues highly valued reinforcers. Such associations appear to be even more powerful than natural reward associations (Robinson & Berridge, 2003). In turn, hypersensitivity of the reward circuits leads to “excessive attribution of incentive salience to drug-related representations, causing pathological ‘wanting’ to take drugs” (Robinson & Berridge, 2003, p. 36). In essence, the incentive-salience model of addiction proposes that drugs and related-stimuli come to dominate the attention of drug-dependent individuals, leading to cravings in drug-related contexts and possible relapse, even after years of abstinence. Thus, the impulse to seek out drugs is influenced by the increased salience of rewarding and/or reinforcing qualities of the desired drug, related mostly to the dopaminergic pathways. However, contemporary models of addiction argue for the involvement of additional neural structures in the maintenance of drug dependence beyond the dopamine reward pathways. The focus of current research lies in the prefrontal cortex and the disinhibition of drug-taking behaviour in light of negative consequences.

Disinhibition and impaired functioning in the prefrontal cortex

Recent advances in the neuroimaging of brain functioning have shown activation of the frontal cortex following drug intake, implicating regions in the frontal cortex in drug use (Goldstein & Volkow, 2002; Tekin & Cummings, 2002). Furthermore, damage to the prefrontal cortex has been implicated in the inability to inhibit impulsive behaviour (Jentsch & Taylor, 1999;

Rolls, 1986). Thus, the second process implicated in continued drug use has been proposed as a tendency to engage in impulsive behaviour via an inability to inhibit behaviour once an approach response has commenced. This cognitive impulsivity is proposed to be a direct consequence of damage in cognitive inhibitory controls of the frontal cortex due to chronic exposure to drugs of abuse (although notably the majority of the evidence for this is derived from experiments involving the administration of psychomotor stimulants such as amphetamine which may not generalise to other drug classes).

The orbitofrontal cortex (OFC), in particular, is involved in disinhibited and compulsive behaviour, due to its involvement in regulating motivated behaviour, the correcting/updating of stimulus-reinforcement associations and the extinction of such associations (Rolls, 1986; Thorpe, Rolls, & Maddison, 1983). Damage to the OFC impairs the extinction of learned associations and the correction of inappropriate stimulus-response associations, resulting in perseverative behaviour (Rolls, 1986).

There is indirect evidence of impairment in this region in some substance-misusing individuals. For instance, Bechara, Dolan, and Hines (2002) found a subgroup of substance-dependent individuals who showed a marked preference of high, immediate payoffs, despite large, delayed losses, and an inability to inhibit behaviour in light of punishment in a financially-rewarded card-sorting game. The performance of this group was indistinguishable from individuals with impairment to the ventromedial prefrontal cortex, supporting the role of this region in the inability to inhibit behaviour in light of punishment. Thus, although in its infancy, there is growing evidence of impaired functioning in the prefrontal cortex and the inability of some individuals to inhibit drug-taking behaviour. Researchers such as Goldstein and Volkow (2002) have further explored the inter-relationships between the prefrontal cortex and the dopamine circuits in contributing to the maintenance of drug abuse and dependence.

Dysfunction and the implications for substance misuse risk

Goldstein and Volkow (2002) have proposed the striato-thalamo-orbitofrontal circuit as one link between the acute reinforcing effects of abused drugs and chronic compulsive drug use in drug dependent individuals. The OFC is connected both directly and indirectly to dopamine pathways and the limbic system, receiving projections from the nucleus accumbens via the mediodorsal nucleus of the thalamus, the ventral tegmental area, and other regions involved in drug reinforcement, such as the amygdala, cingulate gyrus and hippocampus (Volkow & Fowler, 2000). The OFC also projects back to the nucleus accumbens, the amygdala and hippocampus, resulting in a complex feedback system incorporating drug reward information from the limbic system and possibly modulating dopaminergic responses to drug intake.

Given these neural connections between the mesolimbic dopamine circuits and the prefrontal cortex, Volkow and colleagues (Goldstein & Volkow, 2002; Volkow, Fowler, & Wang, 2002) propose dopamine-related reinforcement of drug effects as underpinning initial drug involvement which, with continued use, leads to disruption of the functioning of the striato-thalamo-orbitofrontal circuit; as well as circuits involved with stimulus-reinforcement learning and compulsive behaviour. Compulsive drug intake results in greater dopamine release, which in turn further disrupts the OFC system leading to loss of control over drug use. Thus, in concert with the incentive-salience model of addiction (Robinson & Berridge, 2003), the motivation for drug use moves from an initially pleasure-seeking activity to a compulsive drive response (Goldstein & Volkow, 2002; Volkow et al., 2002).

Integrative models of addiction now centre on neural interactions such as those between the prefrontal cortex (the OFC and the cingulate gyrus in particular) and the mesolimbic dopamine system. Both systems are proposed as acting in conjunction with, and parallel to, the other system resulting in salient attributions of, and heightened memories of, acute drug effects. This interaction between systems also results in decreased inhibitory control leading to cravings and relapse in response to drug-cues, even in long-term, abstinent, former drug and alcohol dependent

individuals. Thus, whilst abnormal activity in the mesolimbic dopaminergic pathways is likely to result in exaggerated incentive-processing, abnormal activity in the prefrontal cortex plays a role in diminished inhibition (Bechara et al., 2002). A number of researchers now discuss a possible synergistic effect between the heightened reinforcement value (incentive-salience) of conditioned drug-stimuli, via sensitisation of the mesolimbic dopamine system and the amygdala, and an inability to consciously inhibit impulsive behaviour, possibly due to prefrontal dysfunction (Jentsch & Taylor, 1999). Furthermore, given the feedback loops between the two regions (dopaminergic pathways and OFC), poor impulse-control and continued drug use, exacerbation of the incentive-salience of drug-cues is likely to occur (Berridge & Robinson, 2003). As Robinson and Berridge (2003) caution, “A loss of inhibitory control over behavior and poor judgement, combined with sensitization of addicts’ motivational impulses to obtain and take drugs makes for a potentially disastrous combination” (p. 46).

A proposed synthesis and model of substance abuse

The progression from drug and alcohol use to more severe abuse and dependence may reflect a wide variability in individual vulnerability to neural sensitisation, with some individuals showing rapid sensitisation to brief drug exposure, whilst others show very little drug-sensitisation (see Robinson, 1988). This may be influenced by a range of factors including genetic factors (see Young et al., this volume), exposure to chronic stress (Anton, 1999) or as we detail below may reflect individual differences in rash-spontaneous impulsivity and reward sensitivity.

As discussed, it would appear that the construct of impulsivity involves at least two related components, (i) a heightened sensitivity to unconditioned and conditioned rewarding stimuli that corresponds to Gray’s reward sensitivity and (ii) a tendency to engage in rash, spontaneous behaviour that has a cognitive component in which an individual has a tendency to disregard risk or consider future consequences. These two components may indeed reflect different underlying neural processes implicated in addictive behaviours, reviewed above. For instance, the concept of

a heightened sensitivity to the reinforcing effects of substances accompanied by a greater propensity to develop conditioned responses has many parallels with Gray's notion of an increased sensitivity to rewarding stimuli. That is, individuals prone to abuse drugs may have a more sensitive BAS. As a result, they are more receptive to the reinforcing effects of drugs and other rewarding stimuli (e.g., Blum et al., 2000). At the neurobiological level, this is reflected in the less efficient inhibitory (D2-like) dopaminergic synapses on striatal neurons believed to exist in persons with high BAS sensitivity (Pickering & Gray, 1999). Because these inhibitory synapses are less efficient there is a lowered output threshold of the nucleus accumbens, a core component of the mesolimbic dopamine reward system. This (likely innate) sensitivity to cues of reinforcement may play a key role in the vulnerability to rapid neural sensitisation and to develop overly-strong associations with conditioned drug cues, as per Robinson and Berridge's (2003) incentive salience hypothesis.

The second impulsivity component we have proposed (Dawe & Loxton, in press) to be involved in addictive behaviour is rash impulsiveness. We suggest that this component of impulsivity may be related to response disinhibition, or the inability to inhibit prepotent approach tendencies. While Jentsch and Taylor (1999) argue this form of impulsivity is the result of chronic drug use, we propose that it reflects individual differences in frontal cortex (specifically, the OFC and anterior cingulate cortex) functioning that may be exacerbated by chronic drug use. Recent studies provide some preliminary support for our hypothesis by linking functioning in the OFC in non-clinical samples with rash impulsiveness. For instance, Horn, Dolan, Elliott, Deakin, & Woodruff (2003) found greater activation of the OFC in participants low in rash impulsiveness, as measured by the Barratt Impulsivity Scale (Barratt, 1994) and the I₇, suggesting a possible vulnerability not connected with chronic drug abuse.

Disinhibition and reward sensitivity?

Despite our argument that reward sensitivity, as proposed by Gray, is different from rash impulsiveness, as measured by novelty-seeking and the I₇, response disinhibition has also been observed in research when using measures of reward sensitivity. For example, Newman and colleagues (Newman, 1987; Newman, Widom, & Nathan, 1985; Nichols & Newman, 1986) have demonstrated that participants high in extraversion (a proxy measure of reward sensitivity) are less able to inhibit an approach response in the presence of punishment cues during a dominant approach response set. Patterson and Newman (1993) attribute this form of disinhibition to a “response modulation deficit”, whereby impulsive persons encountering a punishment during a dominant approach response set are less able to stop and reflect on why they were punished. As a result, they fail to learn that switching to a passive response is more adaptive when faced with specific punishment cues. While Patterson and Newman do not link this response modulation deficit to any underlying neural structures, the disinhibition reported in their studies resembles that displayed by humans and non-human primates with damage to the OFC (Goldstein & Volkow, 2002; Jentsch & Taylor, 1999; Rolls, 1986). Thus, individuals high in rash impulsiveness are expected to be more likely to suffer from a response modulation deficit.

The findings from Newman and colleagues have been replicated using the Sensitivity to Reward (SR) Scale, a measure specifically designed to measure Gray’s BAS, indicating some involvement of reward sensitivity in response disinhibition, which we have attributed to rash impulsiveness (Avila, 2001). Notably, Avila (2001) details a series of studies in which BAS (reward sensitivity) was found to be involved in disinhibited behaviour. Specifically, and consistent with Patterson and Newman’s (1993) model, “BAS-mediated disinhibition” was most likely to occur in persons with high BAS sensitivity who had a reinforcement history of rewarded approach prior to the introduction of punishment cues, and when ‘shallow’ processing of punishment cues was required (i.e., punishment cues were easy to distinguish). This is not surprising as the involvement of reward sensitivity in response disinhibition would be expected

insomuch that there must be some prepotent approach tendency present to be inhibited (or disinhibited). It is also likely that persons high in reward sensitivity/drive will experience stronger prepotent approach tendencies and these would require greater levels of cognitive inhibition. Nevertheless, we propose that response disinhibition is more strongly associated with rash impulsiveness and frontal cortex dysfunction than it is with high BAS sensitivity. Firstly, neuroimaging studies consistently implicate frontal cortical dysfunction as being a core component of response disinhibition (Goldstein & Volkow, 2002; Horn et al., 2003; Jentsch & Taylor, 1999). Secondly, Avila (2001) noted in his studies that BAS-mediated disinhibition was more the result of difficulties in learning the aversive contingency than response inhibition per se. This conclusion is consistent with Patterson, Kosson, and Newman (1987) who found that forcing extraverts to reflect on their response disinhibitions (thus, increasing the likelihood of learning) resulted in later enhanced inhibition. However, this did not benefit neurotic extraverts, who are considered to be more impulsive than stable extraverts. Patterson et al. (1987) attributed this failure to their experimental design, such that they did not literally force participants to focus and reflect on their errors but rather only forced them to wait a fixed time interval before initiating the next trial. This allowed extraverts the opportunity to reflect on their mistake and focus their attention. The neurotic extraverts, however, did not appear to take advantage of this opportunity, or were unable to. For instance, drug addicted individuals are aware of the adverse consequences of their drug use yet they nonetheless cannot inhibit their approach behaviour. Similarly, Rolls (1986) cites an example of how some patients with frontal lobe damage are able to verbalise the correct response on a neuropsychological test but are still unable to correct their behaviour. Thus, depending on the severity of OFC dysfunction (i.e., how high they score on a measure of rash impulsiveness), reflection may be of benefit to some individuals more so than others. Future research would need to address this. The relative contributions of reward sensitivity and rash impulsiveness to predicting response disinhibition also require further empirical testing. In sum,

the evidence just presented suggests that impulsivity related to response disinhibition may in fact reflect a general trait that falls on a continuum that is in turn exacerbated by substance use.

A hypothesised temporal sequence of addiction is as follows: individual differences in reward sensitivity (i.e., BAS sensitivity), mediates initial drug use and incentive salience of drug-related cues, thus leading to continued drug use. The drug-taking behavior is also further reinforced by stress (i.e., increases in corticosteroids), which has been shown in animal studies to sensitize BAS reward pathways (particularly in those highly reactive to stress), and further enhances the reinforcing effects of drug use (Koob & Le Moal, 2001). Drug use is more likely to persist at this point in persons who display rash impulsiveness due to their diminished ability to inhibit prepotent approach tendencies, thereby continuing drug use despite the negative feedback of acute drug withdrawal and other problems associated with substance dependence. Drug taking becomes compulsive, exacerbated by further dysfunction in the OFC and anterior cingulate due to chronic activation of the dopaminergic pathways and further increasing the inability to inhibit behaviour (i.e., increased rash impulsiveness) including drug use (Goldstein & Volkow, 2002; Jentsch & Taylor, 1999). Thus, whilst an impulsive, reward sensitive temperament may increase the probability of experimenting with psychoactive substances, other characteristics (i.e., high rash impulsiveness) may also play a role in the progression from occasional use to more chronic abuse and dependence.

Priorities for future research in testing a two-factor model of impulsivity

It is now evident that impulsivity is not a homogenous construct. However, the model we have proposed has not been tested in clinical groups and, in particular, in people with substance misuse problems. Therefore, in the first instance it is necessary to determine whether the model has empirical support by using measures conceptually related to both reward drive and rash impulsivity in determining whether these are indeed independent constructs. This model could be further tested in a series of cross-sectional studies in clinical groups with specific predictions made

regarding the role of reward drive and rash impulsivity in the initiation and maintenance of substance misuse. The relationship between heightened reward drive and substance misuse may be associated with the initial use of substances, since people with greater sensitivity to reward, as measured using BAS-related scales such as the Sensitivity to Reward (SR) Scale, are more likely to approach novel stimuli (Avila, 2001). Similarly, those high in reward sensitivity are perhaps more sociable and therefore more inclined to try substances in the presence of peer pressure (Claridge & Davis, 2003; Knyazev, Slobodskaya, Kharchenko, & Wilson, in press). Further, the psychoactive effects of the substance may be perceived as having greater reinforcement/reward value in those with heightened sensitivity to reward resulting in a subsequent augmentation of conditioning to drug-related cues, thus increasing the likelihood of further substance use.

The role played by the rash impulsiveness construct is somewhat more complex. It has occurred to us that young people with impulsive behaviour difficulties (i.e., conduct disorder, attention deficit hyperactivity disorder) tend to perform poorly on tasks that require a modification of previously rewarded behaviour following the introduction of a punisher (Avila, 2001). That is, once in an approach response set they are unable to shift behavioural response despite negative feedback. It is also notable that there are particularly high rates of comorbidity between conduct disorder and substance misuse in young people and equally high rates in adults with antisocial personality disorder (ASPD) and substance misuse. While heightened reward drive may be common to both those with substance misuse only and those with comorbid substance misuse and conduct disorder or ASPD, heightened rash impulsiveness may distinguish the comorbid group. Some preliminary data to support this is reported by Moeller and colleagues (Moeller et al., 2002) in which those with both cocaine dependence and ASPD scored higher than those with a diagnosis of cocaine dependence on the Barratt Impulsiveness Scale (a measure of rash impulsiveness in our proposed model) but did not differ on a delayed reward task. Further investigation using a combination of laboratory based tasks and self-report measures could test this further.

Finally, whilst there may well be individual differences in the propensity towards behaving in a rash impulsive manner, it may be that there are specific substances in which this heightened propensity is compounded by damage to specific pathways involved in cognitive inhibitory controls. The process has been well documented for the psychomotor stimulants such as cocaine and amphetamine, but does the use of other substances such nicotine have an effect on cognitive inhibition? The role of individual differences in the treatment of substance misuse also warrants some attention. For example, many drug and alcohol treatment programs include cue-response prevention strategies to prevent relapse following abstinence. However, given the evidence cited above of high levels of sensitivity to conditioned cues of reward in substance misusing populations and the proposal that chronic use exacerbates the salience of conditioned drug cues and reduced ability to inhibit approach behaviour in light of such cues, it seems reasonable to question the utility of such strategies in highly reward-driven clients. Most treatment models in the addiction field do not incorporate an individual differences component in which personality features or traits are taken into account when conceptualising a treatment plan - although Linehan's recent work is one exception (Linehan, Dimeff, & Reynolds, 2002; Van den Bosch, Verheul, Schippers, & Van den Brink, 2002). It is possible that understanding individual differences in reward drive and rash impulsiveness will assist further in elucidating potential mechanisms of change for individual clients and enhance treatment effectiveness.

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Table 1
Summary of studies on impulsivity and substance misuse

Authors	Sample	Measure of impulsivity	Measure of substance use	Results
Cloninger, Sigvardssen, & Bohman (1988)	431 Swedish participants: assessed at age 11 years and again at age 27 years	TPQ	Interview	While all personality variables predicted adult alcohol abuse, high NS scores and low HA scores were most predictive of early-onset alcohol abuse
Grau & Ortet (1999)	149 non-alcoholic Spanish women	KSP (Spanish)	Frequency & Quantity of use	Stepwise regression found the Monotony Avoidance/Sensation Seeking subscale of the KSP and the Impulsiveness subscale of the KSP were the most significant personality predictors of frequency and quantity of alcohol use, respectively.
Howard, Kivlahan, & Walker (1997)	Review of the literature applying Cloninger's Tridimensional Theory of personality to substance abuse	TPQ	Various measures	NS scores predicted presence of alcoholism and type of alcoholism (e.g., Type I), early onset alcohol abuse, cigarette use, criminality, and presence of antisocial personality disorder.
Johnson, Turner, & Iwata (2003)	Representative community sample of 1803 participants aged 19-21	BIS/BAS Scales	Interview	Participants with a lifetime diagnosis of drug abuse/dependence or noncomorbid alcohol abuse/dependence were found to have significantly higher BAS-FS scores
Knyazev, Slobodskaya, Kharchenko, & Wilson (in press)	4501 Russian high school students	GWPQ-R-Short, EPQ	Frequency of use	BAS was best personality predictor of substance use and 2 nd best predictor out of all variables examined (e.g., drug offer, SES). BAS also moderated the relationship between peer drug offer and drug use.
Masse & Tremblay (1997)	1034 boys aged 6-10 years and followed up each year until 10-15 years old	TPQ	Author's own questionnaire-yes/no	High NS scores and low HA scores predicted early-onset substance use
McGue, Iacono, Legrand, Malone, & Elkins (2001)	2670 parents of twins; 666 boys and 702 girls assessed at age 11 and followed-up at age 14	Parents: Constraint (MPQ) Adolescents: TRF scales	Age of first drink	In parents, age of first drink was significantly predicted by Constraint scores; In adolescents, scores on TRF scales of oppositionality, hyperactivity/impulsivity, and inattentiveness (i.e., disinhibition) at age 11 predicted drinking onset by age 14
Sher, Bartholow, & Wood (2000)	457 undergraduates	TPQ, EPQ	Diagnostic Interview Schedule III-A	NS and EPQ-P were the most consistent predictors of each substance use disorder examined at initial testing and 6-yr follow-up, some even after controlling for the presence of other concurrent substance use disorders
Sher, Walitzer, Wood, & Brent (1991)	490 children of alcoholics and nonalcoholics	TPQ, EPQ, EPI, MMPI	Frequency of use, DSM-III criteria	"Behavioural undercontrol" scores (aggregate of impulsivity subscales of measures used) were significantly higher in children of alcoholics (who also reported more alcohol/drug problems) and also mediated (along with alcohol expectancies) the relationship between paternal alcoholism and child's alcohol use
Shillington & Clapp (2002)	1612 adolescents aged 15-21 yrs	Scale constructed by researchers (6 items)	Frequency of alcohol & marijuana use	Adolescents who used alcohol & marijuana were more impulsive than those who only used alcohol. Impulsivity score was also a significant predictor of severity of alcohol-related problems.