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Behavioral and Neurological Foundations for the Moral and Legal Implications of Intoxication, Addictive Behaviors and Disinhibition

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Abstract

Disinhibition and addictive behaviors are related and carry moral implications. Both typically involve diminished consideration of negative consequences, which may result in harm to oneself or others. Disinhibition may occur on state and trait levels, and addictive substances may elicit disinhibitory states, particularly when intoxication is reached. Data suggest that trait disinhibition and addictions may be conceptualized as involving misdirected motivation with underlying biological bases including genetic factors, alterations in neurotransmitter systems and differences in regional brain function. The influences of intoxication on the brain share similarities with cognitive impairments in individuals with chronic substance abuse and those with trait disinhibition related to frontal lobe injuries. These findings raise questions about volitional impairment and morality. Although impaired volition related to disinhibition and addictive behaviors has been studied from multiple perspectives, additional research is needed to further characterize mechanisms of impairment. Such findings may have important implications in multiple legal and psychiatric domains.

Keywords

addiction; alcohol; disinhibition; impulsivity; intoxication; morality

Introduction

On a regular basis, people make decisions with moral implications, some of which are mundane (e.g., the decision whether or not to leave work a few minutes early when the boss is out of the office), while others are potentially life changing (e.g., choosing whether or not to pursue an extramarital affair). Moral judgments are made frequently and range from relatively small (e.g., a quip that a co-worker has used his or her looks to get ahead) to large in stature (e.g., the use

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of racist, sexist, or homophobic epithets). These situations involve making a distinction between “good” and “bad” behaviors. Accordingly, “morality” has been defined as a system of ideas of right and wrong conduct (American Heritage Dictionary, 2006). Among academicians, a clear consensus on a definition of “morality” is lacking (Damon, 1998; Frank, 2003; Kagan, 1993). In attempting to arrive at a definition of morality, theorists have tended to focus on either formal/structural (e.g., the universalizability of a moral principle) or material conditions (e.g., welfare or justice concerns) (Haidt, 2003).

Data suggest that a moral sense is present in both humans and primates (e.g., reciprocal altruism, deWaal, 1996), suggesting generalizability. Individual cultures, however, may differ with regard to the material conditions emphasized (Shweder, Much, Mahapatra, & Park, 1997). Volition is considered important in Western conceptions of morality. Accordingly, acts over which the actors have little to no control are typically thought to be outside of the moral realm (Gert, 2008). The American legal system places similar emphasis on volition, in that people tend not to be held legally responsible for unintentional acts (Morse, 2007). In addition to volition, arguably the other most significant point of emphasis in the Western notion of morality is that one should not harm others or impinge upon other people's individual liberties (Shweder et al., 1997).

Addiction, substance dependence, addictive behaviors, disinhibition and impulsivity are related terms and constructs, all of which have implications with respect to morality. “Addiction” to a substance describes a compulsive pattern of use, characterized by impaired control over the use of the substance and continued use in the face of negative consequences (O'Brien, Volkow, & Li, 2006). A related concept is “substance dependence,” a diagnosis given to individuals who meet criteria for problematic use delineated in the fourth edition of the Diagnostic and Statistical Manual (DSM-IV, American Psychiatric Association, 1994) or the tenth edition of the International Classification of Mental and Behavioural Disorders (ICD-10; World Health Organization, 1992). The extent to which addictions should include non-substance-related behaviors (e.g., gambling) has been debated (Potenza, 2006). Subsyndromal substance use behaviors with clinical or legal relevance are not encompassed within the term “addiction.” Multiple terms have been used to define subsyndromal behaviors. Here, we will use the term “addictive behaviors” to describe a broad range of actions that could be characterized by or potentially lead to impaired control or engagement despite negative consequences (e.g., gambling or intoxication resulting from substance use). Thus, the term “addictive behaviors” can refer to actions performed by addicted and non-addicted individuals. Addictive behaviors may also include behaviors encompassed by the term addiction. The use of the term addictive behaviors in this manner is not meant to represent the sole definition, but rather the one employed in this manuscript.

“Disinhibition” can occur on either a state or trait level. Anderson, Smith and Fischer (2003) state that, “trait disinhibition has been theorized to be the mechanism underlying impulsive, sensation-seeking and risk taking behavior” (p. 384). Following the work of Patterson and Newman (1993) and according to Anderson et al., trait disinhibition is most closely characterized by a tendency to focus on and pursue reward, even in the face of punishment. Bond (1998) offers the following definition for state disinhibition: “the loss of restraint over some form of behaviour” (p. 42). When individuals experience state disinhibition, this is typified by patterns of brain activation and behavioral output that appear in some ways similar to what is observed in trait-disinhibited individuals (Bond; Lyvers, 2000). These findings may explain why some people exhibit impaired decision-making while using substances, particularly when intoxicated (Bond; Lyvers).

Impulsivity has been defined as “a predisposition toward rapid, unplanned reactions to internal or external stimuli with diminished regard to the negative consequences of these reactions to

the impulsive individual or others” (Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001; Potenza, 2006; 2007). As defined, impulsivity is a complex, multi-faceted construct with relevance to a broad range of psychiatric disorders including substance use disorders (Moeller et al.).

The links between disinhibition, impulsivity, addiction and addictive behaviors are apparent from the overlap in their definitions. All involve diminished restraint or control and continued behavioral engagement despite adverse consequences (Anderson et al., 2003; Moeller et al., 2001; O'Brien et al., 2006). Substance use is one of the most common stimuli leading to state disinhibition (Bond, 1998). While the links between trait disinhibition and addictive behaviors are arguably well established (Moeller et al.; Potenza, 2006), the nature of the association is unclear. Three non-mutually exclusive possibilities are that elements of trait disinhibition increase risk of addictive behaviors (Belin, Mar, Dalley, Robbins, & Everitt, 2008); that trait disinhibition and addictive behaviors have common underlying causes (Chambers, Taylor, & Potenza, 2003; Kreek, Nielsen, Butelman, & LaForge, 2005) and that characteristics associated with trait disinhibition are consequences of engagement in addictive behaviors (Beveridge, Gill, Hanlon, & Porrino, 2008).

There are multiple moral and legal implications relating to addictive behaviors and disinhibition. Adverse consequences may result from both. These consequences may involve circumstances in which another person could be harmed (e.g., physical aggression toward others, driving while intoxicated), and thus these actions may be morally (Shweder et al., 1997) and legally proscribed (Morse, 2007). For those who suffer from addictions and those characterized by disinhibition at the trait and/or psychopathological level (e.g., antisocial personality disorder), volition may be conceptualized as misdirected or impaired, given that both may be characterized by difficulties in planning and in weighing potential consequences (Moeller et al., 2001). Individuals with alcoholism often struggle to abstain from drinking (Witkiewicz & Marlatt, 2004). Impulsive people may have significant difficulty inhibiting responses (Moeller et al.). Alternatively, the volitional differences in individuals with addictions or increased impulsivity may reflect a deliberate preference for immediate gratifications or rewards at the expense of future consequences or potential gains (Rachlin, 2007). Given the importance of volition to Western conceptions of morality (Gert, 2008) and legality (Weil, 1989), the altered volition that characterizes addicted and trait disinhibited individuals raises questions of how their behaviors should be characterized on a moral continuum from highly immoral to amoral (i.e., outside of the moral realm). When volition is altered, this does not necessarily mean that all free will is suspended and the issue of free will is not supposed to be considered when determining legal responsibility (Morse). Therefore, alterations in volition does not imply that all moral and/or legal responsibilities for personal choices are nullified.

The overarching goal of the present review is to briefly summarize empirical findings in the relevant psychology, psychiatry and neuroscience literature related to these types of moral and legal issues. In doing so, our goal here is not to excuse “bad” behavior. Rather, our objective is to outline how processes of decision-making differ for individuals who are addicted or disinhibited and describe the neurobiology underlying these differences. The implications of these differences, including moral, legal and treatment issues, will be discussed.

Addictive behaviors

Moral conceptions of addictions have been considered from both lay and professional viewpoints. The following quotation taken from the Alcoholics Anonymous *Big Book* highlights moral notions of the addictions: “Those who do not recover are people who cannot or will not completely give themselves to this simple program, usually men and women who are constitutionally incapable of being honest with themselves ... They are naturally incapable

of grasping and developing a manner of living which demands rigorous honesty” (Alcoholics Anonymous, 2001, p. 58). Since this passage was first published in the earliest edition of the Big Book, research has offered strong evidence for an underlying biological basis to the onset and maintenance of addiction. Specifically, evidence has shown that addictions are highly heritable. Also, neural function underlying addictions has been examined with several key brain regions and neurotransmitter systems implicated.

Estimates of heritability for drug addictions range between 30-60% (Kreek et al., 2005). The genetic propensity for addiction appears two-pronged in that there are contributions common across addictions and specific addictions appear associated with particular genetic variants (Kreek et al.). For instance, a recent report from the Collaborative Study on the Genetics of Alcoholism (COGA; Dick et al., 2007), indicated possible involvement of a gene variant corresponding to ankyrin repeat and kinase domain containing 1 (ANKK1) in association with alcoholism. This gene has a role in signal transduction (i.e., the process by which cells convert one kind of stimulus or signal into another) and response to external stimuli. According to the authors, there is reason to believe that ANKK1 may contribute to dopaminergic reward processes.

The COGA group has investigated a factor termed a low level of response (LR) to alcohol. People with LR report relatively little effect of alcohol even after consuming several drinks. Several longitudinal studies have established that LR—first assessed during young adulthood, presumably before the onset of tolerance—predicts the subsequent onset of alcohol dependence and other alcohol-related problems later in life (Heath et al., 1999; Rodriguez, Wilson, & Nagoshi, 1993; Schuckit & Smith, 1996; Volavka et al., 1996). While the COGA group is beginning to identify genes underlying LR, prior studies indicate that correlations between levels of self-reported subjective effects of alcohol are considerably stronger between siblings than between non-related individuals, suggesting that the apparent risk presented by LR is heritable (Wilhelmsen et al., 2003). Schuckit and Smith have reported that LR mediates associations between a positive family history of alcoholism and the outcome of developing alcohol dependence.

These data suggest that some risk for the development of alcohol dependence is present relatively early in life, before alcohol is first consumed, and that there are biological mechanisms underlying such propensity. Other important factors concern the onset and maintenance of addiction once substances have begun to be consumed, and the extent to which there are biological underpinnings to these processes. These factors have been considered through research that has identified roles for multiple neurotransmitters, such as dopamine and serotonin, in addictive behaviors. Although it is beyond the scope of the present article to describe in detail these neurobiological mechanisms, some discussion is warranted.

Dopamine is considered to mediate rewarding and reinforcing effects of drugs (Goldstein & Volkow, 2002). Continued seeking and taking of substances may be perpetuated, at least in part, by reduced numbers of dopamine D2 receptors in the brain (Volkow, Fowler, & Wang, 2003). According to Volkow et al., a reduced number of receptors leads to altered dopamine signaling, which could lead to an impairment on the part of individuals susceptible to addiction to be adequately reinforced by everyday activities considered by most people to be enjoyable. Dopamine levels, however, are not sufficient to account for the onset of addiction since dopamine increases can be observed in addicted and non-addicted individuals following the introduction of drug (Goldstein & Volkow). In fact, the dopamine release observed in response to cocaine administration is typically larger in non-addicted individuals as compared to addicted ones (Volkow et al., 1997). Although multiple studies have implicated dopamine systems in drug addictions, drugs influencing dopamine neurotransmission (e.g., dopamine antagonists like haloperidol) have not demonstrated much success in clinical settings in the

treatment of substance dependence or addictive behaviors like pathological gambling (Potenza, 2006).

The neurotransmitter serotonin has also been associated with heavy alcohol use and addiction (Herman, Philbeck, Vasilopoulos, & DePetrillo, 2003; Kreek et al., 2005). LeMarquand and colleagues (1999) found that a sample of non-alcoholic young men (ages 18-25) with strong multi-generational family histories of alcoholism responded differently than a sample of young men of similar age with a negative family history to a tryptophan depletion procedure. Tryptophan is the amino acid precursor to serotonin. Following tryptophan depletion, central serotonin levels are reduced and serotonin neurotransmission altered. Low levels of serotonin or its main metabolite have also been implicated in addictive and impulsive behaviors (Kreek et al., 2005; Rogers et al., 1999; Verkes et al., 1998). While in a tryptophan-reduced state, family-history-positive young men in the LeMarquand et al. study made more commission errors on a go/no-go behavioral task—indicative of greater difficulty in inhibiting responses—than did family-history-negative young men, suggesting that the family-history-positive group was more sensitive to the effects of reduced serotonin neurotransmission on impulsive behavior. These findings suggest associations between impulsive behavior and low levels of serotonin in individuals with greater propensities for developing substance addictions. While serotonin may be involved in the onset and maintenance of addiction, it is unlikely that dysfunction in serotonin neurotransmission is solely or predominately responsible for addictive behaviors. Limited efficacy has been demonstrated in clinical trials of selective serotonin reuptake inhibitors (SSRIs) for the treatment of alcohol problems (Torrens, Fonseca, Mateu, & Farre, 2005), suggesting limitations with respect to a central or solitary role for serotonin in addictive behaviors.

In addition to findings regarding genetics and the roles of specific neurotransmitters, another valuable line of research has sought to localize particular areas of the brain where activation has been found to be altered in addicted individuals. Early research focused on limbic brain regions and more recent research has focused on the frontal cortex (Jentsch & Taylor, 1999). Frontal cortical function influences higher order, executive functions (Tancredi, 2005) through cortical-striatal circuits (Robbins, 2007). Frontal lobe volume losses have been observed in individuals who are dependent on multiple drugs including cocaine, alcohol and heroin (Franklin et al., 2002; Pfefferbaum, Sullivan, Mathalon, & Lim, 1997), suggesting the possibility of compromised executive functioning. The orbitofrontal cortex and anterior cingulate gyrus are two frontal structures that appear particularly relevant to addiction and impulsivity because they are neuroanatomically connected to “reward-related” limbic areas, and thus may have an important role in reward-related impulse control. These areas tend to be activated in addicted individuals during the processes of intoxication, craving and heavy episodic use (Goldstein & Volkow, 2002). Altered brain function can be observed in addicted individuals even at a young age. Tapert and colleagues (2003) found that adolescents with alcohol use disorder diagnoses displayed greater brain activation in response to alcohol-related cues compared to adolescents without alcohol-use disorders. Differential activation was observed in limbic and cortical areas including the left anterior prefrontal cortex, anterior cingulate, parahippocampus, amygdala, hypothalamus, and the posterior cingulate. These same regions have also been implicated in studies of addiction among adults.

These types of studies have helped characterize the neural dysfunction that may result from ongoing engagement in addictive behaviors. These findings do not exclude the possibility that compromised function in these areas predates addiction (see section on trait disinhibition below), nor do they exclude the possibility that factors other than drug use contribute. The issue of causation is typically difficult to address in human studies. Animal studies allow the opportunity to carefully control exposure to drug over an extended period of time and to observe the influences of this exposure on the animal's behavior and neural circuitry in ways that may

not be possible in humans due to logistical or ethical constraints (Tabakoff & Hoffman, 2000). Using a non-human primate model of cocaine exposure, Beveridge et al. (2008) reported neural changes occurring following prolonged drug exposure, including significant declines, first in the ventromedial prefrontal cortex, but later extending into the orbital and dorsolateral prefrontal cortices. Given that animal investigations can be controlled in manners not typically possible in human studies, these types of animal findings offer strong support for the notion that prolonged drug exposure can induce significant changes in brain function.

In summary, data from genetic studies suggest that elements contributing to the risk of addiction are present before alcohol or other drugs are first consumed. Once substance consumption begins, biological factors also contribute to the onset and maintenance of addiction. Multiple neurotransmitters contribute to addiction, including but not limited to dopamine and serotonin, and multiple brain regions may become compromised in addicted individuals.

Trait disinhibition

Like addictive behaviors and addictions, trait disinhibition appears relevant to moral issues. Individuals high in trait disinhibition are thought to be less likely to reflect on and learn from punishment after it occurs and thus may be less likely to alter their behaviors accordingly (Anderson et al., 2003; Patterson & Newman, 1993). A failure to learn from punishment may lead to antisocial actions. Such failure could result in harm being brought upon other people, a morally proscribed action (Schweder et al., 1997). Another similarity with addictions is that recent research questioned the level of volition with which highly disinhibited individuals engage in some behaviors, particularly those diagnosed with psychopathology related to trait disinhibition (Moeller et al., 2001). Due to space constraints, a decision was made to focus on impulsivity in the present review. Many considerations regarding state and trait disinhibition may also pertain to the theoretically related constructs of risk-taking, sensation seeking and extraversion, though.

When one discusses “impulsivity” it can be as either as a trait—which typically begins to manifest itself early in the life span and may often have an impact on multiple areas of functioning in one's life—or as a more transient occurrence, resulting from acute biological and/or environmental perturbations, which may or may not be far reaching in impact (Dougherty, Mathias, Marsh, Moeller, & Swann, 2004). At times, it may be difficult to differentiate between trait and state impulsivity in that manifestations of underlying trait impulsivity may reflect a biological or environmental stimulus, not unlike state impulsivity (Mann, Waternaux, Hass, & Malone, 1999). The definition for impulsivity described earlier in this article (Moeller et al., 2001; Potenza, 2007) contains multiple factors, highlighting the heterogeneous nature of impulsivity. Much recent animal and human evidence points to the conclusion that impulsivity is not a singular construct, but rather a collection of theoretically related elements with differing neurobiological bases (Evenden, 1999; Winstanley, Dalley, Theobald, & Robbins, 2004). To take an example from the animal literature, Winstanley et al. found that depletion of forebrain serotonin in rats was associated with premature responding on a visual attention task, but had no effect on performance in a task assessing delay discounting (i.e., a tendency to favor small, immediate rewards over larger, but more distal rewards; Bickel, Miller, Yi, Kowal, Linquist, & Pitcock, 2007). Human research has also yielded findings indicative of heterogeneity in the construct of impulsivity. Swann and colleagues (2002) found that errors of commission on a continuous performance task were highly correlated with scores on the Barratt Impulsiveness Scale, Version 11 (BIS-11; Patton, Stanford, & Barratt, 1995)—a widely used self-report measure of impulsivity—but delay-discounting performance was only moderately correlated with BIS scores. Other studies have found either no correlations, modest correlations or even inverse correlations between measures of impulsivity across domains (e.g. rapid response vs. risky decision-making; Tucker et al., 2004), and self-report

and behavioral measures within domains (e.g., delay discounting) may not correlate (Krishnan-Sarin et al., 2007).

Impulsivity has been found to be associated with negative outcomes (Krishnan-Sarin et al., 2007) and relevant to multiple forms of psychopathology (Moeller et al., 2001). Impulsivity is an important characteristic of ADHD, borderline personality disorder and conduct disorder (Moeller et al.). The link between impulsivity and bipolar disorder seems particularly strong in that manic patients typically engage in impulsive acts (Harmon-Jones, Barratt, & Wigg, 1997). Other data link impulsivity to substance use, abuse and addiction (Moeller et al.; Tucker et al., 2004; Krishnan-Sarin et al., 2007).

Brain lesion studies provide insight into impulsivity. Damage to the orbitofrontal area may lead to disinhibited behavior, including perseveration and failure to assess consequences (Lyvers, 2000). Patients with stroke-inflicted damage to the ventromedial prefrontal cortex (vmPFC) have deficits in risk-reward decision-making (Bechara, 2005). Accordingly, these patients have been found to perform disadvantageously on the Iowa Gambling Task (IGT; Bechara et al., 1997). In this task, participants have the opportunity to draw cards from four different decks. Over time, selecting from two of the decks will lead to large monetary rewards that are paired intermittently with even larger monetary losses, such that one performs disadvantageously in the long run by repeatedly drawing from these two decks. In contrast, the other two decks are associated with consistent, moderate winnings that, in the long run, will lead to financial gain. Healthy participants often begin by preferentially drawing from the disadvantageous decks because of the large associated gains, but after some large losses, eventually transition to selecting from the advantageous decks. In her review of the literature on orbitofrontal cortex damage and decision-making, Fellows (2007) argues that the specific impairments at work likely include difficulty in rapidly learning from negative feedback, and problems with preference judgments in general. Injuries to regions of the prefrontal cortex (particularly the ventromedial component) have been associated with impulsive and risky decision making outside of the laboratory. Such was the case with Phineas Gage, a railroad worker who, at age 25, suffered an accident in which a steel tamping rod exploded through his face, exiting through the top of his skull. While he survived, his personality changed from mild-mannered and hard working before the accident to profane and ill-tempered afterwards. As with the stroke victims described above, frontal cortical regions, including the vmPFC are hypothesized to have been severely damaged in Gage's accident and have led to the drastic changes in his behavior (Damasio, 1994).

Other brain regions, including the amygdala, have been implicated in IGT performance (Bechara et al., 1999). The amygdala is involved in emotional processing, attaching affective meaning to stimuli in the environment (Tancredi, 2005). Findings from studies of decision-making in individuals with lesions in vmPFC, amygdala and other brain regions led to the somatic marker hypothesis of decision making, which posits that emotional processes influence rational decisions (Damasio, 1994). According to this hypothesis, individuals with vmPFC lesions draw from disadvantageous decks because their injury precludes receipt and processing of relevant somatic information. In contrast, those with damage to the amygdala tend to be aware of which decks are the best ones, yet they nevertheless choose repeatedly from disadvantageous decks, ostensibly because they do not experience the same feelings of discomfort after drawing from these decks in the manner that non-lesioned individuals do (Bechara et al., 1997; Tancredi).

Trait disinhibited individuals without brain injuries have been characterized by seemingly analogous dysfunction in regional brain activation, compared to control subjects. Individuals with pathological gambling often score high on measures of impulsivity and disadvantageous risk/reward decision-making (Pozena, 2006). In a simulated gambling task, individuals with

pathological gambling as compared to those without were characterized by decreased activation in the vmPFC and ventral striatum, regions implicated in drug addiction and reward processing (Chambers et al., 2003; McClure, Laibson, Lowenstein, & Cohen, 2004; Reuter et al, 2005). Individuals with pathological gambling also show differences in neurotransmitter levels (e.g., levels of serotonin metabolites) that have been associated with impulsivity (Brewer & Potenza, 2008).

Trait disinhibition appears to have important determinants over the lifespan. As with substance addictions, trait disinhibition appears heritable, and data suggest the involvement of multiple genes contributing to both impulsivity and addictions (Kreek et al., 2005). Development of the prefrontal cortex occurs over the lifespan, with substantial volumetric changes occurring from infancy through the third decade of life (Giedd, 2004). As such, one might anticipate notable consequences if certain aspects of development do not occur normatively in the proper timeframe. Two case studies reported by Anderson and colleagues (1999) suggest that there is a critical period for the development certain aspects of prosocial functioning. They detail the cases of two young adults who had received injuries to the prefrontal cortex at a very young age (15 months for one and three months for the other). As with patients who have experienced similar injuries during adulthood, these patients frequently engaged in disinhibited and antisocial behavior. But unlike those who were injured in adulthood, these patients were not able to develop and maintain factual knowledge of social conventions and principles of morality. These characteristics suggest that the early occurrence of their injuries may have resulted in their missing a critical period in their cognitive development.

In summary, as with addictions, data support the heritability of trait disinhibition and the existence of developmental stages relating to the propensity to engage in impulsive acts, risky decision-making and antisocial behavior. The onset of trait disinhibition and its various manifestations appear influenced by biological processes, including altered function in brain activation patterns and neurotransmitter levels.

Links between trait disinhibition and addictive behaviors

In addition to ADHD, borderline personality disorder and bipolar disorder, addiction represents another form of psychopathology closely related to impulsivity (Moeller et al., 2001). While addiction may not be inherently impulsive in nature (Moeller et al.), addiction and impulsivity frequently co-occur (Potenza, 2006). In adults, impulsivity has been linked with self-reports of heavy substance use and addiction. In his review of individual variation in college drinking, Baer (2002) reports that impulsivity has strong associations with heavy drinking. He concluded that possible mechanisms for this association include positive social motives, positively-valenced alcohol-related expectancies and frequent participation in drinking games. Regarding links with addiction, a sample of substance abusing patients had significantly higher total scores than a sample of undergraduates on the Barratt Impulsiveness Scale (Patton et al., 1995).

Links between impulsivity and addiction have also been found using behavioral tasks. Delay discounting paradigms appear relevant in that a tendency to prefer immediate over delayed rewards is considered to be an aspect of impulsivity, and addiction is typified by a choice of the use of a substance in the present, often at the expense of concern about future outcomes (Bickel et al., 2007). Accordingly, addicted individuals tend to discount future rewards more steeply than do non-addicted individuals. This finding has been replicated for drug addictions and impulse control disorders, including opiate, cocaine, and alcohol abuse/dependence and pathological gambling (see Bickel et al., for a review).

Associations have also been found between substance use and risky decision-making on a behavioral task. Goudriaan, Grekin and Sher (2007) classified college students according to self-reported heavy episodic drinking over a course of two years. They found that the subgroup

of stable heavy episodic drinkers tended to make fewer advantageous choices on the IGT compared with the low heavy drinking subgroup. The Goudriaan et al. findings are consistent with others linking suboptimal performance on the IGT with substance dependence, including dependence on cocaine (Grant, Contoreggi, & London, 2000) and alcohol (Farrell et al., 2001).

These and other findings suggest an association between trait disinhibition and addictive behaviors. The precise nature of the association, however, is not certain. Three possible explanations are that disinhibition increases the likelihood that one will engage in addictive behaviors and perhaps eventually develop an addiction (Belin, Mar, Dalley, Robbins, & Everitt, 2008); that aspects of trait disinhibition have common underlying causes with addictive behaviors (Chambers et al., 2003; Kreek et al., 2005) and that disinhibitory behavior and characteristics are a consequence of prolonged involvement engagement in addictive behaviors (Beveridge et al., 2008). These three possibilities need not necessarily be mutually exclusive.

Trait disinhibition may place individuals at risk for addictive behaviors. Tarter and colleagues developed an index of a latent trait they refer to as neurobehavior disinhibition, which includes measures of affective, behavioral and cognitive indicators of difficulty in self-regulation (Tarter et al., 2003). Recently, this group reported results from a longitudinal study in which a “high-risk” sample of boys was first assessed between the ages of 10-12, again at age 16 and then at age 19 (Mezzich, Tarter, Feske, Kirisci, McNamee, & Day, 2007). They reported that neurobehavior disinhibition trait score at the age 10-12 and at the age 16 assessment predicted substance use disorder diagnosis at age 19, along with a number of other adverse outcomes including arrest and violent behavior.

While these developmental findings in humans may suggest a causal role for trait disinhibition leading to addictive behaviors, several potential confounds, including co-morbid psychiatric conditions and socio-economic status, cannot be excluded (Beveridge et al., 2008). Animal studies have helped to clarify links between trait disinhibition and addictive behaviors. Belin, Mar, Dalley, Robbins and Everitt (2008) investigated links between two traits associated with disinhibition (i.e., impulsivity and novelty seeking) and the shift to compulsive cocaine use that characterizes human addiction (O'Brien et al., 2006). Belin et al. characterized rats as high or low responders based on their level of locomotor reactivity in a novel environment (thought to model sensation seeking) and as high or low in impulsivity based on their observed ability to delay performance in a five-choice serial reaction-time task. They compared high and low responding rats and high and low impulsivity rats in their likelihood of initiating cocaine self-administration and in the transition to compulsive use (e.g., continued cocaine use despite negative consequences). They found that while high responders were more likely to initiate cocaine self-administration, high impulsivity predicted propensity to transition from use to a compulsive pattern of use. These findings suggest that sensation seeking predicts cocaine consumption, while impulsivity predicts risk of developing an addiction.

A second possible explanation for associations between trait disinhibition and addictive behaviors is that the two share a common etiology. Such commonalities may present at genetic levels (Kreek et al. 2005), represent common ways in which motivational behavioral repertoires develop (Chambers, Bickel and Potenza, 2007), or be related to other mechanisms.

A third potential explanation is that addictive behaviors lead to disinhibition. Bechara (2005) summarized neuroimaging work linking impulsivity with addictive behaviors in his hypothesis outlining two separate but interacting neural systems responsible for controlling decision-making. One system is characterized as impulsive and is heavily influenced by the amygdala, and the other, reflective system is dominated by the prefrontal cortex. Bechara posits that addiction results from an imbalance that occurs in the course of drug use in which the impulsive

system comes to dominate the reflective system, leading to a decrement in the user's willpower to resist using drugs. In their review delineating associations between addictive behaviors and decision making, Redish, Jensen & Johnson (2008) posit that addictive behaviors drive maladaptive decision-making through pharmacologic interactions with neurophysiological mechanisms that evolved for the process of normal learning. Such decisions may at times be considered impulsive, risky and/or immoral. Like the process of learning many other behaviors (e.g., driving a car), thinking about drug use may initially be deliberate and involve significant planning, but with frequent repetition, these behaviors may transition to involving more rapid, unplanned, habitual actions (Belin & Everitt, 2008; Chambers et al., 2007; Yin & Knowlton, 2006). These habitual actions may be less sensitive to consequences. For addicted individuals, the decision to continue to use substances may reflect the habit system dominating the decision-making process (Bechara, 2005; Robinson & Berridge, 1993; Tiffany, 1990). Both Bechara and Redish et al. describe scenarios in which substance use may lead to increased disinhibition. Alternatively, substance use may occur as a result of domination of decision-making by controlled, reflective processes (see Redish et al., for examples of such scenarios).

To summarize, trait disinhibition and addictive behaviors are associated, although the nature of the association warrants clarification. Associations between trait disinhibition and addiction may be demonstrated using self-report and behavioral measures. Trait disinhibition may act as a precursor to the onset of addiction in a considerable subset of addicted individuals, consistent with findings in animal studies (Belin, Mar, Dalley, Robbins, & Everitt, 2008; Dalley et al., 2007). The association between disinhibition and addiction may also be explained by common etiologies (Chambers et al., 2003; Kreek et al., 2005; Chambers et al., 2007). Addictive behaviors may also lead to disinhibition (Bechara, 2005; Beveridge et al., 2008; Redish et al., 2008).

Intoxication and substance-induced disinhibition

To this point, we have discussed mainly trait disinhibition. Temporary disinhibited states may be induced by external or internal/biological stimuli, even in individuals low in trait disinhibition (Dougherty et al., 2004), as may be seen with suicide. The “escape theory” of suicide (Baumeister, 1990) posits that individuals engaging in suicidal behavior are not necessarily impulsive at a trait level. Environmental factors, however, may induce an impulsive state, leading to a reduced capacity to inhibit behavior (Dougherty et al.). Particularly relevant to the present review is addictive substances as factors leading to increased impulsivity, with alcohol representing arguably the most commonly used substance involved in behavioral disinhibition (Bond, 1998). A limitation to substance-induced disinhibition that may manifest itself in some circumstances is tolerance to the subjective effects of substances. In alcohol, acute tolerance is defined as “reduced impairment at a given blood alcohol concentration” (Fillmore, Marczinski, & Bowman, 2005, p. 663). In a sample of social drinkers, Fillmore et al. found evidence of acute tolerance to the impaired activation of behaviors but not to the inhibition of behaviors.

Addictive substances may evoke disinhibitory states, particularly during intoxication (Bond, 1998; Lyvers, 2000). The neural mechanisms for substance-induced disinhibition that Bond offers share similarities with Bechara's (2005) dual neural system account. Bond cites Patterson and Newman's (1993) theory, which posits that disinhibition interferes with a switch from a dominant response set typified by appetitive, goal-directed approach behaviors, to a more passive, information-gathering state following the experience of an unexpected adverse event. While Patterson and Newman's theory was designed to explain trait disinhibition, Bond argued that it offers a useful basis for understanding substance-induced disinhibition. Lyvers concluded that both acute and chronic substance administration may lead to frontal lobe impairment. Specifically, alcohol, cocaine and opioid use may acutely depress prefrontal

cortical activity. Similarities between the effect of intoxication on the brain and the effects of injuries to the frontal lobes have been described. Both have been associated with a lack of awareness of errors and of the impact of one's own actions on others, along with failures to self-regulate, including a lack of recognition of inappropriate behaviors (Lyvers).

According to Bond (1998), the difficulty in shifting from a dominant to a passive response set that typifies disinhibition is mediated by declining serotonin levels. A contributing role for dopamine, as well as other neurotransmitters, in the effects of acute drug administration also has support. Acute administration of all known addictive substances is associated with an increase in extracellular dopamine concentrations in limbic areas of the brain, especially in the nucleus accumbens (Ritz, Lamb, Goldberg, & Kuhar, 1987). Dopamine increase may also be observed in frontal cortical regions (Goeders & Smith, 1986). High concentrations of dopamine in subcortical areas are associated with rapid, dominant, approach responses, while high concentrations in the cortex may serve to inhibit control processes. As Jentsch & Taylor (1999) and others (e.g., Arnsten, 2007) have argued, there is a homeostatic level of dopamine in the brain such that levels that are too high or too low are suboptimal. Cocaine, opioid and alcohol intoxication have been associated with reduced glucose metabolism throughout the brain (London et al., 1990a; London et al., 1990b; Volkow et al., 1990). Changes in the brain during intoxication may result in individuals remaining in a dominant, appetitive, approach state when a shift to a passive, information-gathering state may be optimal (Bond; Patterson & Newman, 1993), thus increasing the likelihood that impulsive and risky actions may ensue. Both individual and environmental factors, however, may contribute to determinations of optimal neurotransmitter levels and behavioral decisions.

Laboratory behavioral tasks performed following an alcohol challenge suggest similarities between alcohol-induced disinhibition and trait disinhibition. One study compared the effects of three doses of alcohol (0.00, 0.45, and 0.65 kg/g) on adults' performance on a cued go/no-go task (Marczinski, Abrams, Van Selst, & Fillmore, 2005). The researchers found dose-dependent increases in commission errors on a version of the task in which participants were required to make key press responses to go targets and suppress key presses to no-go targets.

Bond (1998) explains that, "Although behavioral disinhibition is frequently associated with aggression, it can also lead to effects on other social behaviour resulting in inappropriate sexual advances or the hyperactivity associated with mania" (p. 42). The latter experience, which may have elements of euphoria, appears associated with activation of the right prefrontal cortex during alcohol intoxication (Wallace et al., 1996). The Drinking-Induced Disinhibition Scale (DIDS), a self-report measure, assesses three types of disinhibitory effects of alcohol, defined as thoughts, feelings and experiences that are typically inhibited but more likely to be expressed while under the influence of alcohol (Leeman, Toll, & Volpicelli, 2007). The measure assesses euphoric/social, sexual and dysphoric (i.e., capturing the experience of the "sad drunk," in which an individual expresses typically inhibited negative affect while under the influence) disinhibition. Supporting the notion of dysphoric disinhibition, findings from the social psychology literature have demonstrated that induction of negative affect may lead to reduced impulse control (Gray, 1999; Tice, Bratslavsky & Baumeister, 2001). Scores on the three subscales of the DIDS were found to have disparate associations with measures of problem drinking. In a sample of first-year university undergraduates, sexual disinhibition was significantly associated with heavy episodic drinking, while dysphoric disinhibition was significantly associated with alcohol-related consequences and euphoric/social disinhibition had modest but significant associations with both heavy episodic drinking and alcohol-related consequences (Leeman, Toll, & Volpicelli). These findings indicate that, like trait disinhibition, alcohol-induced disinhibition appears heterogeneous in nature.

The relationship among alcohol-induced disinhibition and risk-taking has been investigated in laboratory paradigms. In an alcohol challenge study, participants who were administered alcohol were more likely than those given placebo to lose their entire stake in a subsequent gambling task, suggesting that alcohol use may lead to impulsive or risky behavior resulting in gambling losses (Kyndon & Dickerson, 1999). During fMRI, Calhoun, Pekar and Pearlson (2004) tested participants' performance in a driving simulator following the administration of placebo, low and high dose alcohol. Following the high dose, participants spent more time driving over the speed limit and experienced more vehicle collisions (at a trend level) than during a baseline run conducted while they were sober. Associations were found between driving behavior and activations in the orbitofrontal and motor cortices and the cerebellum following alcohol administration.

Initial substance use may also impair control over further substance use. Impaired control—defined as “a breakdown of an intention to limit consumption in a particular situation” (Heather, Tebbutt, Mattick, & Zamir, 1993, p. 701)—has long been considered an important element of addiction (Levine, 1978). According to Lyvers (2000), impaired control may be related to long lasting frontal cortical changes resulting from chronic substance use. Impaired control could be conceptualized as a form of substance-induced disinhibition in cases when initial substance use disinhibits predetermined limits on consumption behavior, leading to heavy use. The centrality of impaired control is reflected in two of the criteria for substance dependence in the fourth edition of the Diagnostic and Statistical Manual (DSM-IV): “The substance is often taken in larger amounts or over a longer period than was intended,” and, “There is a persistent desire or unsuccessful efforts to cut down or control substance use” (APA, 1994, p. 181).

The Impaired Control Scale (ICS; Heather et al., 1993) is a reliable and valid self-report instrument that has been significantly and positively correlated with measures of alcohol-related problems and dependence among drinkers in treatment. ICS scores have correlated with treatment outcome, with greater impairment of control associated with lower likelihoods of successful outcome (Heather, Booth, & Luce, 1998; Heather & Dawe, 2005). Scores on the ICS have been associated with both heavy episodic drinking and alcohol-related consequences in a sample of first-year undergraduates, even when covarying for other predictors of problem drinking (Leeman, Fenton, & Volpicelli, 2007).

To summarize, addictive substances may elicit a disinhibitory state in the user. The brain and behavioral effects of substance-induced disinhibition resemble those of trait disinhibition in several ways. Substance-induced disinhibition is a heterogeneous phenomenon with different individuals reporting different types of disinhibiting influences.

Legal Implications of intoxication, addiction, and disinhibition

Neuroscience research has raised questions concerning what it means to be responsible for becoming addicted to drugs or alcohol, for the behavioral symptoms of addiction, and for relapse (Bonnie, 2002). Impaired control in addiction suggests that there are neurobiological processes (for example, the urge to drink) over which the addicted individual may have relatively diminished control. For an individual with an addiction, conditioned feelings of drug or alcohol urges may be involuntarily aroused by different cues. Do such findings suggest that individuals with addiction are not responsible for using drugs after they have become addicted? Is an impulse “irresistible” or simply not resisted? While a mental health perspective on behavior may suggest that conduct represents the product of multiple, possibly “causal” factors that determine choice, the legal perspective addresses these questions of alleged impaired volition by examining issues of responsibility, consciousness, mental states of intention and knowledge, and the capacity for rationality and compulsion (Morse, 2007).

The question for the legal system has been whether people who voluntarily use drugs are responsible for the consequences of their actions. There is a well-documented link between substance use and crime: approximately 70% of those arrested for violent crimes test positive for substances (Sinha & Easton, 1999), alcohol use frequently precedes or accompanies marital violence (Leonard & Quigley, 1999), and child abuse and neglect have been associated with substance use (Schuck & Widom, 2003). For an individual with an addiction, is an “inability” (or diminished ability) to obey the law the same as failure to obey the law?

A goal of the criminal justice system is to establish responsibility for criminal acts, and a major tenet of responsibility is a person's intention. Criminal behavior is punishable when the actor is morally culpable. The assumption of criminal law is that a person has the ability to distinguish between lawful and unlawful behavior and has the freedom to act on this knowledge. Certain individuals may lack some capacities to make rational and voluntary choices due to mental disease and this could eliminate or mitigate the moral authority to impose criminal sanctions. Certain legal defenses are therefore recognized that either excuse completely or reduce the person's responsibility or moral blameworthiness (Morse, 2007).

Mental disease alone does not exculpate a defendant from criminal responsibility. The mental disease must bear a relationship to the criminal behavior such that the defendant is morally not culpable. How to determine best that relationship has been a matter of debate for decades in the law. In response to this difficult question, the law has established tests of criminal responsibility. These are often referred to as “insanity defenses,” and, in the United States, individual states have adopted different tests with different names (Morse, 2007).

Although the insanity defense has existed since the 12th century, it was initially not considered an argument for the defendant to be found not guilty, but instead a way for a defendant to mitigate a sentence. The seminal case in sanity came from England and is now called the M'Naghten rule. In 1843, Daniel M'Naghten, an Englishman who apparently was under the delusion he was being persecuted (a presumed symptom of paranoid schizophrenia from which he was apparently suffering), shot and killed Edward Drummond, Secretary to British Prime Minister Sir Robert Peel. Mr. M'Naghten was under the delusion that Mr. Drummond was Sir Peel. Mr. M'Naghten was found not guilty on the grounds he was insane at the time of his act. The case in turn led to a standard regarding the defense of insanity. The M'Naghten rule states: “To establish a defense on the ground of insanity, it must be clearly proved that, at the time of the committing of the act, the party accused was laboring under such a defect of reason, from disease of mind, as not to know the nature and quality of the act he was doing; or if he did know it, that he did not know he was doing what was wrong” (8 Eng. Rep. 718, 1843). The test to determine if defendants can distinguish right from wrong is based on the idea that they must know the difference in order to be convicted of a crime.

The “irresistible impulse defense” is another test of criminal responsibility designed to address defendants who know their acts are against the law but who cannot control their impulses to commit them. This test is often combined with M'Naghten in many jurisdictions. It suggests that the defendant is not liable if, by reason of mental illness, he is unable to exert control over his actions (Weil, 1989). Generally this is construed to apply only to impulses that arise suddenly and are acted upon without reflection. How one can differentiate an uncontrolled act and an uncontrollable act is a matter of debate. Also, courts have typically been reticent to believe that most mental illnesses impair control to such an extent that the person is a mere “robot” giving in to impulses.

A slightly different standard was devised by the American Law Institute and is currently used in 27 states: “a person is not responsible for criminal conduct if at the time of such conduct as a result of mental disease or defect he lacks substantial capacity either to appreciate the

wrongfulness of his conduct or to conform his conduct to the requirements of law.” This standard asks whether defendants have a substantial incapacity to appreciate the criminality of their conduct or to conform their conduct to the law (Bromberg, 1992).

The Federal Insanity Defense Reform Act holds: “It is an affirmative defense to a prosecution under any Federal statute that, at the time of the commission of the acts constituting the offense, the defendant, as a result of a severe mental disease or defect, was unable to appreciate the nature and quality or the wrongfulness of his acts. Mental disease or defect does not otherwise constitute a defense” (18 U.S.C. § 17(a)). The federal statute therefore does not use the irresistible impulse test, and it is the defendant who must prove his inability to appreciate the nature and quality of the crime.

Can a person who suffers from a mental disease caused by or related to the voluntary use of intoxicants rely upon an insanity defense? The mental disease or defect that is termed “insanity” is generally viewed as an involuntary condition that one would not willingly contract. The exercise of choice which underlies the voluntary use of substances is typically treated differently under the law. Generally it is agreed that a person who is intoxicated is not insane. An intoxicated person is presumed sane. Therefore, voluntary intoxication at the time of criminal activity will generally not relieve a person of criminal responsibility. Absolving individuals from criminal responsibility who have temporarily impaired their mental capacities by use of alcohol or drugs would encourage recklessness (Bonnie, 2002). A few states, however, recognize the doctrine of “settled insanity.” Where the continued use of drugs or alcohol has resulted in a “fixed” condition of insanity, which is distinct from and independent of the period of intoxication, the person can then be excused from criminal responsibility (Feix and Wolber, 2007). This insanity is not the same as a “compulsive need” for alcohol or drugs and is not part of voluntary intoxication. The insanity must be beyond the person's control [United v Knott, 894 F2d 1119 (9th Cir. 1990); United States v Garcia 94 F3d 57 (2nd Cir. 1996)].

Diminished capacity is a partial defense, allowed in only some jurisdictions, which may recognize intoxication when a specific intent for a crime is required under the law. The defense may therefore argue that intoxication prevented the person from having the specific intent to commit the crime. Guilt is often then found for a lesser crime that does not require the specific intent (Bonnie, 2002). Even when intoxication does not rise to the level of a defense, it may lead to at least partial mitigation in punishment (Keiter, 1997).

Given the increasing neuroscientific understanding of impulsivity, some have called for a revival of the “control test” for insanity (i.e., awareness that some people understand the rules of behavior but are unable to follow them) (Redding, 2006). One proposed idea is that when this defense is raised, there must be evidence, based on expert testimony, of a substantial dysfunction of the frontal lobes and/or the limbic system due to brain damage or illness (Redding). This would prevent insanity defenses that have either no evidence of brain damage or that are based on “low-grade diffuse frontal lobe dysfunction” that one might find, for example, in attention deficit hyperactivity disorder (Redding).

Moral implications of intoxication, addiction and disinhibition

To this point, we have discussed data supporting the underlying, biological bases to the onset and maintenance of addictive behaviors, state and trait disinhibition, and links between disinhibition and addictive behaviors. Two main tenets of Western morality—the positive association between volition and personal responsibility for one's own actions (Gert, 2008) and the proscription against harm toward others (Shweder et al., 1997)—are both relevant to disinhibition and addictive behaviors. The definitions of disinhibition (Anderson et al., 2003), impulsivity (Moeller et al., 2001; Potenza, 2006; 2007) and addiction (O'Brien et al.,

2006) cited earlier include a tendency to act with diminished regard for negative consequences. This type of pattern may result in interpersonal harm (e.g., drunk driving accidents, physical injuries), and thus choosing to engage in these behaviors has moral implications.

The ramifications of knowledge regarding the biological etiologies underlying disinhibition and addiction as they relate to volition may raise controversy. Trait-disinhibited or addicted individuals (particularly those with related psychopathology such as antisocial personality disorder or bipolar disorder) may, at times, act with altered or poorly controlled volition (Moeller et al., 2001). Relevant issues to consider may include the degree to which volition remains intact. It could be argued that if one acts with little to no volition, it may be difficult to hold the individual responsible either legally (Morse, 2007; Weil, 1989) or morally (Gert, 2008). If the decrement to volition is smaller, the questions become even more complex.

In most cases, impairment to volition that occurs with trait disinhibition and addiction will not be complete. As Satel (1998) reminds us, individuals typically decide on their own whether or not to consume substances. In considering what impact a decrement to volition should have on personal responsibility, it may be valuable to consider competing notions of free will, which may be defined as a non-substance-exposed state in which individuals are able to make and pursue their own choices. According to determinism, events occurring in the world are governed by natural law, such that all occurrences can be traced back to some preceding causal factor. This school of thought gives a limited role to free will. The incompatibilist school contends that if determinism were true, then this would nullify free will. In contrast, the compatibilist school believes that determinism and free will are not necessarily mutually exclusive. Metaphysical libertarians argue that humans have free will, the notion of free will is not compatible with determinism and thus, determinism is false (see Vihvelin [2008] for discussion of these philosophical schools of thought).

Absolute free will is typically not viewed as a prerequisite for personal choice. Thus, while trait disinhibited or addicted individuals may operate with decreased volition and without absolute free will, in most cases, they still have ability to decide and accordingly, should still be held morally and legally accountable for their actions (Morse, 2007). One could even argue more strongly on the side of personal responsibility. An enhanced knowledge about biological factors underlying addiction and trait disinhibition could make individuals *more* aware of the serious potential ramifications of these behaviors (e.g., awareness that the acute effects of intoxication on the brain resemble those observed in patients with brain lesions, Lyvers, 2000). Enhanced knowledge about the neurobiology of addiction serves to advance the development of novel treatments, the presence of which could also be viewed as increasing one's personal responsibility for obtaining effective treatment if needed.

As stated from the outset, the goal of the present paper was not to “excuse bad behavior”, but rather to explore the evidence for biological bases to disinhibition and addictive behaviors and in so doing, contribute to the discourse about the possible influences of these findings on notions of volition, free will, legality and moral responsibility. The study of disinhibition and addictive behaviors is rapidly developing and expanding. Thus, new data emerge on a regular basis. Given the moral implications of addictive behaviors and disinhibition, it is anticipated that these findings will continue to interface with the moral and legal realms. Accordingly, over time, it is expected that these new research findings may lead to further reconsideration of the moral and legal implications of disinhibition and addictive behaviors.

While issues of free will, rationality and individual responsibility may make for stimulating debate and have important legal implications, from a clinical perspective, the most productive and pragmatic course of action might involve improving our understanding of clinically relevant behavioral and biological phenomena in order to prevent illness and treat patients more

effectively. Multiple treatment approaches already utilize information regarding individual differences. Counseling approaches for reducing heavy alcohol use such as Brief Alcohol Screening and Intervention for College Students (BASICS; Dimeff, Baer, Kivlahan, & Marlatt, 1999) make extensive use of individualized feedback. Participants in a BASICS intervention receive a detailed feedback sheet based on results from a thorough biopsychosocial evaluation. This feedback includes information regarding their levels of alcohol consumption compared to age-matched peers and their potential risk stemming from their family history of alcohol problems, with the idea that this knowledge can be used as a tool to facilitate behavioral change. Chamberlain and colleagues (2006) reviewed neurotransmitter systems underlying cognitive impairments related to disinhibition and their relevance to ADHD and its pharmacological treatment. Analogously, Redish et al. (2008) outline multiple ways in which addiction may result from maladaptive decision-making and suggest that interventions should be targeted to match the patient's specific vulnerabilities with regard to decision-making. A current challenge to researchers and clinicians involves investigating constructs relevant to impaired impulse control and incorporating the knowledge gained into treatments that best match patients' needs.

Conclusions

Parallels exist between addictive behaviors and trait disinhibition. During substance-induced disinhibition, substance users demonstrate features resembling individuals who have chronically used substances and those who are trait disinhibited. The ways in which state and trait patterns of disinhibition relate to volition and free will have important clinical and legal implications. Continued discussion and debate is anticipated as an improved understanding is attained of the biological bases of these processes and their relevance to specific psychiatric conditions.

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