Why people change? The role of cognitive-control processes in the onset and cessation of substance abuse disorders

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Abstract

Background: The current effectiveness of preventative and therapeutic interventions for substance use disorders (SUDs) is less than satisfying. Progress in understanding the processes related to the onset, course, and cessation of SUDs merits top priority, given the high prevalence and the severe negative consequences. Aims: This position paper, after summarizing major factors related to onset and cessation processes, discusses the possible role of cognitive-control dysfunctions in the complex interaction between mechanisms of change (mediators) and risk factors (moderators). Findings: In past decades, research has expanded our knowledge about the impact of SUDs on human behaviour. Formal interventions are effective, but there is limited information about the mechanisms responsible for change during the onset and cessation of SUDs and for individual differences related to them. Preliminary results suggest that impairments in higher order control functions play a role in SUDs. Conclusions: Deficiencies in our understanding of behaviour-change processes during the onset and cessation of SUDs require that research have a different focus. A better understanding of the relevance of impairments in executive-control functions might help to improve formal preventative and therapeutic interventions and social conditions. Such interventions might reduce the chances that a SUD will develop or increase the likelihood of recovery from it. Copyright © 2008 John Wiley & Sons, Ltd.

Key words: addiction, change processes, executive control, functions

Introduction

Substance use disorders (SUDs), including harmful or dependent use of alcohol, tobacco, illicit drugs, and psychotropic medications, are prevalent in Western societies (Rehm et al., 2005; Wittchen and Jacobi, 2005); they are associated with severe individual impairments and high social costs (Andlin-Sobocki et al., 2005; Andlin-Sobocki and Rehm, 2005). The overall effectiveness of measures to reduce the likelihood that a SUD will occur, to prevent its progression into a harmful pattern, and to improve treatment outcomes is less than satisfactory. Further progress in

understanding the processes and factors related to the onset, course, and cessation of substance use has top priority.

Previous research has concentrated primarily on factors that increase or decrease the occurrence and progression of a SUD and on factors that are related to outcome following an intervention. The identification of such factors constitutes important progress in research. However, to date we have acquired limited information about how to utilize these factors to improve prevention and treatment outcomes. In particular, we have little evidence about the relative

contribution of different variables and how they interact with one another; more importantly, we know little about the mechanisms underlying substance dependence and recovery. For example, through what mechanism(s) do(es) early childhood trauma increase the risk of later drug use (Mulvihill, 2005)? Is it low self-esteem, a deficit in positive rewards, or anxiety that is relieved by taking anxiolytic drugs? Why do only about 50% of the adolescents who experiment with cigarette smoking progress to patterns of regular or dependent smoking, whereas the other half stop smoking without any formal intervention (e.g., Bundeszentrale für gesundheitliche Aufklärung (BZgA) [Federal Centre for Health Education], 2004), despite similar availability of cigarettes that the two groups have? A better understanding of the processes underlying these marked individual differences should have an strong impact on prevention and early intervention.

There are similar deficits in our knowledge about the cessation of SUDs. People change both with and without the help of formal treatment, but what is the

effective agent in change? Is it a therapy-induced increase in drug-refusal competence; general, treatment-engendered change mechanisms, such as motivational enhancement or the activation of resources; positive therapist–patient interactions; or contextual factors, such as social pressure or support for change (Bühringer, 2006)? We need to know more about these processes and mechanisms of change in order to improve the effectiveness of preventative and therapeutic interventions and their effects on society, both in formal treatment settings and in the social environment.

In this position paper, after summarizing the major factors related to the onset and cessation of SUDs, we discuss the possible role of cognitive-control dysfunctions in the complex interaction between mechanisms of change (mediators) and risk factors (moderators). Within this context, the terms onset and cessation represent the two extremes of substance use patterns that can last almost a lifetime. With respect to the onset of drug use, initial experimental use might progress to regular, harmful, and dependent use, or

Figure 1. Onset and course of substance use and substance-use disorders (SUD) and transition-related factors (modified from Wittchen et al., 1999).

alternatively end with early natural remission. Likewise, cessation of drug use might either lead to persistent abstinence or relapse to harmful use. In this paper, we use the terms drugs and substances interchangeably to refer to all licit and illicit psychotropic substances.

Factors related to the onset and cessation of a SUD

Onset

Most conceptualizations of the onset of SUD assume that there is a progressive development from experimental and regular use to hazardous, harmful, and perhaps dependent use (Le Moal and Koob, 2007). Wittchen et al. (1999) integrate into a vulnerability-risk model (see Figure 1) a broad range of early (innate or acquired) vulnerability factors, such as genetic and family-history variables (Clark et al., 2005; Lieb et al., 2002; Schuckit, 2000); childhood disorders, such as conduct disorders, depression, and anxiety (Costello et al., 2003; McGue and Iacono, 2005); childhood stress and trauma (Goeders, 2004; Mulvihill, 2005); psychological dispositions and traits (e.g. impulsivity and affect liability; Dawe and Loxton, 2004); negative social/environmental factors (e.g. poverty); and more acute, proximal risk factors that occur shortly before and during the critical age-range for the development of a SUD. Examples include the availability of substances (von Sydow et al., 2002); early peer substance use and peer pressure (Bonomo et al., 2004); social networks and social support (Kobus, 2003); coping styles (Shirachi and Spirrison, 2006); and a wide range of impaired cognitive behavioural and emotional competences, such as in self-esteem, self-control, and coping skills. It is further assumed that relevant factors for initial and experimental use are related more to social and environmental conditions, whereas the progression to problematic use is influenced more by individual neurobiological and psychological impairments (Le Moal and Koob, 2007).

Cessation

Even though a large number of randomized-controlled studies (both single and multi-site) and clinical field studies have been conducted, we still have limited information about factors that lead to improvement, recovery, or relapse, whether such changes occur naturally or are due to either semi-professional (e.g. self-help groups) or professional interventions. People change both with and without the help of a formal intervention

(Perkonig et al., 1999, 2008; von Sydow et al., 2001), and therapeutic interventions are effective, regardless of the type of treatment and the disorder for which it is used (e.g. Anton et al., 2006; Project Match Research Group, 1997). Nevertheless, the evidence is still unclear or controversial with regard to change-dependent variables, such as patient and disorder heterogeneity, the specific type of intervention, psychotherapeutic processes (e.g. "problem activation"; Grawe, 2004), content-related factors (e.g. therapist-patient interaction; Horvath and Symonds, 1991), the social context in which the treatment occurs (family conditions, social support, social risks; Broome et al., 2002; Tracy et al., 2005), and the factors responsible for naturally occurring changes (Klingemann, 1991) (see Figure 2).

Challenges for future research

An impressive list of vulnerability and risk factors related to the development of SUDs and to reduction, cessation, and relapse have been identified. Nevertheless, for many years there was astonishingly little interest in understanding why individuals and groups differ in their risk for onset, continuation of, and early cessation of substance use or in the type and extent of negative consequences related to the substance use. The following major deficits in our knowledge must be taken into account, especially when new, empirically based preventative and therapeutic interventions are being developed:

- The relevance of individual differences in the risk for developing a SUD.
After initial, experimental use, most people do not develop a problematic pattern of substance use. More research is needed to identify protective factors.
- Relevance of specific vulnerability and risk factors.
For preventative purposes, more information is needed about the relative importance of innate and acquired individual, social, and environmental risk constellations.
- Processes of change.
Vulnerability, risk, and protective factors alter individuals' probability of developing and resolving a SUD (moderators), but they do not explain the underlying processes of change (mediators; Kraemer et al., 2001). How do risk factors such as early childhood trauma increase the risk of a person's developing a SUD? How does cognitive-behaviour

Figure 2. Factors related to reduction of use, remission, and relapse in substance-use disorders (SUD).

therapy change patterns of use (Morgenstern and Longabaugh, 2000)? More information is needed to help specify new prevention and treatment techniques.

- **Relevance of cognitive-control impairments.** Among the broad spectrum of factors related to SUD, the relevance of impairments in executive functions has received increased attention in recent years (Verdejo-Garcia et al., 2006). Such impairments might place a person at increased risk, but they might also be caused by early phases of a SUD and increase the person's risk of incurring further negative consequences, including the risk of treatment failure and relapse.

The role of cognitive-control processes in SUDs

As the foregoing overview shows, our knowledge about distal vulnerability factors in addictive behaviours is substantial. At the same time, there is a paucity of theories that specify the proximal, cognitive and neurobiological mechanisms that mediate the transition from regular use to substance abuse or from successful cessation to relapse. Research on the neurobiological

bases of substance use has so far focused mostly on dysfunctional alterations in neural circuits related to learning, reward, and incentive motivation (for reviews see Hyman et al., 2006; Robinson and Berridge, 2003). However, from recent cognitive and neuroimaging studies, it is becoming increasingly clear that a comprehensive understanding of the mechanisms mediating the onset and cessation of an addictive behaviour requires us to take cognitive and executive-control processes into account. A central feature of an addictive behaviour is the person's apparent lack of volitional control over drug self-administrative, despite his or her awareness of the severe, adverse consequences of continued use. Addictive behaviours, that is, are characterized by impaired self-control and a marked reduction in the degrees of freedom in the addicted person's behavioural choices (Lubman et al., 2004).

Impaired cognitive and volitional control functions in SUDs

During the past decade, the cognitive mechanisms and neural systems underlying executive functions and volitional action have become a central research focus in

experimental psychology and cognitive neuroscience. In the context of this research, the term volition is not viewed as a mental capacity detached from the physical world that enables people to make free choices independently of causal determination. Instead, volition is used as a summary term for a set of cognitive-control functions that underlie the capacity of humans to select and implement actions on the basis of anticipated future outcomes and to suppress competing habitual or impulsive responses in favour of pursuing long-term goals and intentions (Goschke, 2003; Kuhl, 1996; Metcalfe and Mischel, 1999; Miller and Cohen, 2001; Monsell and Driver, 2000). Thus, whether or not an individual is able to resist an immediate temptation in order to pursue a long-term goal (for instance, to maintain a healthy diet or to refrain from smoking) is assumed to depend on whether or not the individual is able to recruit cognitive-control processes that help to suppress competing motivational tendencies.

Cognitive-control processes are defined as mechanisms that configure and modulate lower-level sensory, affective, mnemonic, and motor processes according to a currently active goal and which enable individuals to execute goal-congruent responses despite competition from strong habits or competing emotional impulses (Goschke, 2003, 2006a; Kuhl, 1996; Miller and Cohen, 2001). Examples of cognitive-control functions are the ability to: (1) maintain long-term goals in the face of distracting stimuli (maintenance and shielding); (2) inhibit strong, but unwanted habitual responses or emotional impulses (impulse suppression); (3) flexibly adapt behavioural dispositions to changing task demands (switching); (4) modify one's behaviour in response to errors, negative feedback, or unexpected action outcomes (error and conflict monitoring).

There is increasing evidence that cognitive-control functions are compromised in drug addicts, as indicated by their impairments on tasks requiring decision-making, guidance of action by anticipated long-term consequences, inhibitory impulse control, and feedback processing (e.g. Bechara et al., 2006; Bechara et al., 2002; Bolla et al., 2002; Grant et al., 2000; Finn et al., 2002; Lyvers and Yakimoff, 2003; Mazas et al., 2000; Monterosso et al., 2001; Petry et al., 1998; Stout et al., 2005; Whitlow et al., 2004). Moreover, neuroimaging studies have revealed metabolic, morphological, and functional differences between drug users and control participants in a distributed network of frontal brain areas involved in cognitive-control functions,

including the dorsolateral prefrontal cortex (PFC), orbitofrontal and ventromedial PFC, and anterior cingulate cortex (ACC) (e.g. Bolla et al., 2004; Eldreth et al., 2004; Hester and Garavan, 2004; Kaufman et al., 2003; Liu et al., 1998; Matochik et al., 2003; Stapleton et al., 1995; Volkow et al., 1992; for reviews see Bechara et al., 2006; Garavan and Stout, 2005; Kalivas and Volkow, 2005; Lubman et al., 2004).

We now briefly highlight three exemplary domains of cognitive control that appear particularly important for explaining the breakdown of self-control in addicted individuals. Figure 3 gives a schematic outline of these control functions and associated brain systems:

- (1) *Goal maintenance, top-down modulation, and impulse inhibition.* A brain structure of critical importance for cognitive control is the lateral PFC, which has been activated in neuroimaging studies in a wide variety of tasks involving cognitive control, including working memory and goal maintenance (e.g. Braver et al., 1997), reasoning and planning (e.g. van den Heuvel et al., 2003), flexible switching between tasks (Gruber and Goschke, 2004), inhibition of prepotent responses (Aron et al., 2004), and the voluntary suppression of emotional reactions (Beauregard et al., 2001; Walter et al., 2005). A central function of the lateral PFC appears to be the active maintenance of goal representations and the top-down modulation or "biasing" of lower-level systems in accordance with the active goal (Goschke, 2003; Miller and Cohen, 2001). Impairments in these functions lead to a reduced modulation of response selection by long-term goals, which manifests itself as an insufficient ability to inhibit habitual or impulsive responses that are triggered by immediate incentives (see the top-down pathway from PFC to the Affective-Motivation Network in Figure 3). There is evidence that drug addicts compared to control participants show aberrant patterns of brain activation in regions of the PFC presumably involved in top-down control and inhibition (e.g. Bolla et al., 2003; Ersche et al., 2005; Goldstein and Volkow, 2002; Hester and Garavan, 2004; Kaufman et al., 2003).
- (2) *Inter-temporal choice and impulse control.* The orbitofrontal cortex (OFC), which has extensive connections to "limbic" brain areas, such as the amygdala and striatum, is involved in the representation of expected action outcomes and associated

Figure 3. Schematic outline of some of the brain systems and cognitive-affective processes involved in impaired cognitive control in addictive behaviour.

reward values (for reviews, see Kringelbach, 2005; Schoenbaum and Roesch, 2005; Walter et al., 2005). Patients with damage to the OFC often make maladaptive decisions, which appear partly due to an overly strong reliance on immediate reward and a neglect of delayed negative outcomes (Bechara et al., 2006). Impairments in decision-making have been investigated with the Iowa Gambling Task (Bechara et al., 1997), in which participants choose between four decks of cards yielding different (facsimile) monetary gains or losses. Patients with lesions in orbitofrontal and ventromedial regions of the PFC continue to make disadvantageous choices even after repeated negative feedback, indicating that these patients neglect delayed future rewards or punishments (Bechara et al., 1997). There is some evidence that substance abusers also are hypersensitive to immediate rewards but neglectful of losses on the Iowa Gambling Task (Bechara et al., 1997; Bechara et al., 2006) and that they show impairments in decision-making tasks requiring the ability to suppress impulsive responses to immediate rewards and to choose actions on the basis of anti-

cipated long-term outcomes (e.g. Dom, Sabbe, Hulstijn, and van den Brink, 2005; Rogers et al., 1999).

- (3) *Error- and conflict-monitoring.* There is accumulating evidence that regions in the medial PFC, notably the ACC, form part of an alarm system that is involved in monitoring error and response-conflicts during task performance (Carter et al., 2000; Kerns et al., 2004; Ridderinkhof et al., 2004; Yeung et al., 2004). According to an influential hypothesis (Botvinick et al., 2001; Botvinick et al., 2004), the ACC evaluates whether goal attainment is endangered and enhanced cognitive control is needed in order to shield a current goal from distraction by enhanced inhibition of distracting sources of information. Drug users have been shown to exhibit impairments in the monitoring of response conflicts and action errors, as well as in the context-sensitive adaptation of cognitive control in response to errors or conflicts, and these impairments have been accompanied by reduced activity in neural systems such as the ACC, which are critically involved in error detection and conflict monitoring [see Garavan

and Stout (2005) for a review]. Substance abusers may thus be less able than others to adjust their responses and cognitive-processing strategies in response to negative feedback (see the pathway from the monitoring network to the cognitive-control network in Figure 3).

A multiple-control systems perspective on addictive behaviours: interaction of higher cognitive-control and basic motivational systems

The importance of impaired cognitive-control processes for addictive behaviours becomes obvious when considered together with the evidence for dysfunctional alterations in subcortical systems involved in motivation, emotion, and reward. Like other recent proposals, we assume that behavioural indicators such as insufficient goal maintenance, top-down control and impulse inhibition, exaggerated responses to short-term rewards and impaired error- and conflict-monitoring in drug addicts reflect an imbalance between two distinct neural control systems: (1) an automatic, implicit, or "impulsive" system that processes cues signalling immediate threats and rewards and that evolved to enable organisms to respond quickly and automatically to current needs (and which is presumably hyperactive in drug abusers; cf. Robinson and Berridge, 2003); (2) a higher order ("reflective") cognitive-control system that underlies the cognitive representation of delayed action outcomes and the guidance of behaviour by long-term goals (and which is presumably hypoactive in drug addicts) (cf. Bechara et al., 2006; Deutsch and Strack, 2006; Goschke, 2006b; Jentsch and Taylor, 1999; LeDoux, 2003; Lieberman, 2000; Volkow et al., 2004; Volkow and Li, 2004; Yin and Knowlton, 2006).

The assumption of an imbalance between a hyperactive impulsive system and a hypoactive cognitive-control system may explain why addicts continue to use drugs despite being aware of the adverse, long-term physical and psychological consequences. As we noted earlier, drug use can be considered as a prototypical example of an inter-temporal choice conflict between an immediate reward (the short-term effects of the drug) and delayed consequences (e.g. the long-term costs of drug abuse). Traditional behavioural-choice models attribute impulsive behaviour in inter-temporal choice conflicts (i.e. choosing a smaller, immediate reward despite larger, delayed costs of doing so) to the fact that individuals discount the value of delayed future rewards and costs according to a hyperbolic temporal discounting func-

tion, which leads to an overweighting of immediate short-term rewards relative to long-term outcomes (e.g. Ainslie, 2001). Recent evidence indicates that the degree to which delayed outcomes are discounted results from the interaction between two distinct neural networks, one of which is tuned to immediate rewards and steeply discounts delayed rewards, and the other mediates the anticipation of future rewards and discounts delayed rewards only minimally (McClure et al., 2004; Tanaka et al., 2004). In particular, in a functional magnetic resonance imaging (fMRI)-study (McClure et al., 2004) in which participants made choices between monetary reward options differing in magnitude and delay of delivery, it was found that parts of the limbic system connected to the midbrain dopamine system (including the ventral striatum and medial OFC) were preferentially activated by decisions involving immediately available rewards, whereas regions of the lateral prefrontal and posterior parietal cortex were activated irrespective of delay. Importantly, fronto-parietal areas were significantly more active than limbic areas when participants chose the later option, whereas activity in the two regions was comparable (with a trend toward greater limbic-system activity) when participants chose the earlier option.

These results suggest that an addictive behaviour – especially the lack of control over strong motivational impulses triggered by drug-related cues and the reduced ability to choose actions in accordance with anticipated long-term consequences – reflects, on the one hand, a dysfunctional alteration in the interaction and relative balance between prefrontal and parietal brain areas involved in anticipation of long-term rewards and executive control, and, on the other hand, subcortical structures that mediate affective responses to immediate rewards.

Implications for future research

In conclusion, our selective review of recent findings from experimental psychology and neuroimaging studies shows that progress in explaining the neurocognitive mechanisms underlying addictive behaviours requires that we gain a better understanding, on the one hand, of the dynamic interaction between brain systems involved in volition, cognitive control, and executive functions and, on the other hand, of basic motivational-affective systems involved in the implicit processing of rewards and incentives. This perspective raises a number of basic questions that might serve to define the core aims for future research:

- (1) We have limited knowledge about whether impairments in brain systems involved in error-monitoring, inhibition, or impulse control are consequences of prolonged substance abuse, or whether premorbid dysfunctions in these neural circuits place people at risk for developing SUDs or for being prone to relapse after withdrawal, especially under elevated stress.
- (2) Little is known about which components of cognitive control are relevant for predicting different aspects of addictive behaviours. It is thus necessary to investigate the relative importance of different factors in the development of addictive behaviours, such as (a) hypersensitivity to immediate rewards, (b) hyposensitivity to negative consequences, (c) reduced sensitivity to delayed action outcomes, (d) impaired conflict- and error-monitoring, and (e) impaired impulse control.
- (3) From the perspective of our multiple control-systems hypothesis, the behaviour of addicted individuals cannot be accounted for solely in terms of their explicit, verbally expressed beliefs, attitudes, and goals; instead, implicit motivational and emotional processes must be taken into account (cf. Wiers and Stacy, 2006). Whereas research on the determinants of drug abuse has been dominated by reliance on explicit measures (questionnaires, self-reports) assessing drug abusers' beliefs about their feelings, thoughts, goals, and the causes of their behaviour, it is now important to place greater weight on "indirect" tasks that allow implicit cognitive and affective processes to be measured. There is, in fact, preliminary evidence that implicit measures of responsiveness to drug-related cues account for large portions of the variance in drug use and relapse (e.g. Waters et al., 2003). Moreover, it has been shown that subliminal reward cues or small doses of drugs can trigger implicit "wanting" and instigate drug-seeking behaviour in addicted people, even if the person does not experience a conscious emotion or a desire or goal to use (Berridge and Winkielman, 2002; Fischman and Foltin, 1992; Lamb et al., 1991).
- (4) How can we account for the obvious paradox that, on the one hand, behavioural and neuromaging studies have revealed impairments in cognitive-control functions in drug addicts, whereas, on the other hand, drug addicts often exhibit considerable long-term planning and self-control in their desire to procure and use drugs?
- (5) Are impairments in cognitive control general or disorder-specific? Improved research on impairments in cognitive-control functions among people with a SUD would help us better understand how individuals develop and recover from a SUD or a related addictive behaviour (i.e. impulse-control disorder).
- (6) What about "addiction-like" behaviours that do not involve substance use (e.g. pathological gambling)? There is growing evidence that impairments in cognitive-control functions and impulsivity are also relevant for "behavioural" addictions (impulse-control disorders), such as pathological gambling (e.g. O'Connor and Dickerson, 2003; Potenza et al., 2003). However, similarities and differences between these impairments and the processes involved in SUDs and their relevance for prevention and treatment are not yet clear.
- (7) How are brain circuits mediating cognitive-control processes and implicit incentive motivation affected by therapeutic interventions? Can impaired cognitive control-processes be remedied by therapeutic interventions and will the effects be reflected in changes in neural-activation patterns? Can volitional-control competencies and efficiency in using them be specifically trained in high-risk groups and thus serve to protect susceptible individuals?

Discussion

In this paper on the onset and cessation of substance use and SUDs, we ask why people change and what factors cause them to do so. An even more challenging question is why various people show different patterns of onset, course, and cessation of substance use, even though they have similar exposure to substances and to formal interventions and have experienced similar social contexts. Better understanding of these issues would be theoretically important, and would also help us to develop better preventative and therapeutic interventions. It would probably also help to shape social-context factors that would guide informal interventions (e.g. self-help groups) and enhance natural change.

Recent research has identified a multitude of factors that affect onset, progression, and cessation and the success or failure of treatment and prevention interventions. Correlations among variables have been identified, but information about cause-and-effect relationships is limited. Improved research on impairments in cogni-

tive-control among people with a SUD seems to be a promising way for understanding individual differences in pathways into and out of SUDs and related addictive behaviours (i.e. impulse-control disorders).

In spite of initial, promising results on the relevance of cognitive-control dysfunctions and the urgent need for additional research on this topic, we should avoid the shortcoming of merely selecting another variable to investigate for its potential role in substance abusers' ability to change. Research has indicated that underlying processes are influenced by participant- and substance-related variables and the social context in which substance use occurs. In order for better preventative and therapeutic interventions to be developed, this range of factors must be taken into account.

In view of our present state of knowledge and the broad range of factors that are related to SUDs, we suggest three goals for future research. First, research should focus more on processes of change and the variables that affect them. Second, research knowledge from various disciplines, ranging from neurobiology to social psychology, should be integrated. Third, research methodology should be extended beyond randomized control trials, and closer cooperation between disciplines is needed. This would include neurobiology, neuropsychology, and mental-health disciplines; psychological research on basic human functions, such as cognition and emotion; the integration of research technology, such as fMRT; and longitudinal analyses of substance use during adolescence.

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