Cognitive biases toward alcohol-related words and executive deficits in polysubstance abusers with alcoholism

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ABSTRACT

Aim To study cognitive biases for alcohol-related cues on executive function tasks involving mental flexibility and response inhibition in polysubstance abusers with alcoholism.

Design The responses to alcohol-related cues of detoxified polysubstance abusers with alcoholism and of non-addicts were compared.

Setting The University of Iowa City, Iowa, USA.

Participants Thirty detoxified polysubstance abusers with alcoholism (PSA) and 30 healthy non-substance abusers (CONT).

Measurements Using the ‘Alcohol Shifting Task’, a variant of the go/no-go paradigm, we measured the response times and the accuracy of responses to targets and distracters. Sometimes the alcohol-related words were the targets for the ‘go’ response, with neutral words as distracters, sometimes the reverse. Several shifts in the type of the target occurred during the task.

Findings Relative to CONT, PSA were generally slower to respond to targets, but the group difference was smaller when alcohol-related words were the targets. A signal detection analysis also indicated that relative to CONT, the PSA had more difficulties discriminating between targets and distracters (low d’), and they showed more signs of decision bias (low C), reflecting increased readiness to respond to both targets and distracters. However, these discrimination and inhibition deficits were more pronounced when alcohol-related words were the targets. Furthermore, the weaknesses in RT and C were more pronounced in PSA after shifting the targets from alcohol-related to neutral words, or vice versa.

Conclusions These results suggest that PSA have cognitive biases towards information related to alcohol, and that these biases, as well as the poor executive functions (lower mental flexibility and response inhibition) revealed in PSA might be responsible for their failure to maintain abstinence.

KEYWORDS Alcoholism, cognitive biases, drugs, inhibition, relapse, shifting.

INTRODUCTION

Alcoholism is characterized by intense preoccupation with drinking alcohol despite the devastating consequences of this behaviour in terms of social and occupational status (APA 1994). Like other addictive disorders, alcoholism is characterized by chronic vulnerability to relapse after detoxification. More specifically, once detoxified from alcohol, 50% of patients resume drinking within 3 months and lose control over their consumption (e.g. Guardia et al. 2002; Noël et al. 2002). From an information-processing perspective, cognitive factors are seen as mediators of the relapse into alcoholism. Indeed, numerous studies have shown that non-amnesic...
alcoholic patients, detoxified and abstinent for 2–3 weeks, suffer from a variety of cognitive impairments which may persist after several weeks of abstinence (e.g. Parsons 1998; Noël et al. 2002), and may last for years (e.g. Parsons 1998). Specifically, these patients exhibit severe deficits in executive functions, including response inhibition, abstract reasoning, shifting attention, rule detection, decision-making and problem solving (e.g. Mazas, Finn & Steinmetz 2000; Noël et al. 2001). These deficits have been linked to structural and functional abnormalities in the frontal lobes (for a review, see Moselhy, Georgiou & Kahn 2001). Moreover, it has been found that deficits in working memory and inhibitory control could be critical factors responsible for the alcoholic’s weak capacity to remain abstinent after detoxification (Noël et al. 2002). In addition, some studies have demonstrated the presence, in recently detoxified alcoholics, of attentional biases for processing alcohol-related stimuli (for a review of the question, see Franken 2003). For example, on a modified version of the Stroop task, detoxified alcoholics were slower in naming the colour of alcohol-related than neutral words, a difference that was absent in non-alcoholic control participants (e.g. Johnsen et al. 1994; Sharma, Albery & Cook 2001). Furthermore, a recent study indicates that attentional bias for alcohol-related stimuli is also a reliable predictor of relapse (Cox et al. 2002). More specifically, unlike control participants and alcohol abusers detoxified successfully, alcohol abusers whose treatment was unsuccessful (i.e. those who relapsed or did not maintain out-patient contact after discharge) had a significant increase in attentional distraction for alcohol stimuli during the 4 weeks of in-patient treatment. These data suggest that cognitive deficits and biases are critical factors responsible for the maintenance of alcoholism or for relapse. However, few studies have explored the cognitive deficits and biases in recently detoxified alcoholics, and the interpretation of some of these results remains open to questions. For instance, if the Stroop task has generally been considered as examining resistance to interference (Nigg 2000), it might also be viewed as taxing mechanisms of inhibitory control, i.e. the suppression of pre-potent responses (i.e. to read the alcohol-related words rather than the colour).

With the present study, we aimed to examine the cognitive biases and executive control deficits that may be present in polysubstance abusers with alcoholism (PSA), using a go/no-go paradigm. The main reason for selecting PSA was essentially epidemiological: patients who abuse both drugs and alcohol accounted for more than 42% of admissions to substance abuse treatment facilities reported by State agencies in 2000 (TEDS 2002). We sought to examine two mechanisms of executive control: (1) inhibitory control (‘stopping’) and (2) shifting (‘mental flexibility’), which are keys to control habits and to adapt to unfamiliar situations (Shallice & Burgess 1998). We hypothesized that PSA would exhibit impairments in tasks requiring inhibitory control, as well as shifting. Furthermore, we hypothesized that although PSA may have poor mechanisms of inhibitory control and shifting in general, these deficits are especially pronounced when processing and controlling alcohol-related information, thus explaining the difficulty of PSA to ‘stop’ their current course of thought and action (i.e. drinking) and ‘shift’ to a non-drinking activity.

**METHOD**

**Participants**

All subjects were adults (>18 years old) and provided informed consent that was approved by the appropriate human subject committees at the University of Iowa. The demographic data on the two groups are presented in Table 1.

**Healthy participants (CONT)**

All normal control subjects or CONT were recruited from the Iowa City area through local advertisement. The selection criteria of healthy subjects include the absence of a history of mental retardation, learning disability, psychiatric disorder, substance abuse, neurological disorder or systemic disease that might affect the central nervous system, based on clinical interviews conducted with these subjects before their inclusion in the study. All CONT were paid for their participation.

### Table 1 Demographic and clinical data of subjects who participated in the study.

<table>
<thead>
<tr>
<th></th>
<th>PSA</th>
<th>Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total n</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>Age (years): mean ± SD</td>
<td>33.6 (8.9)</td>
<td>31.2 (10.9)</td>
</tr>
<tr>
<td>Gender (M/F)</td>
<td>13/17</td>
<td>15/15</td>
</tr>
<tr>
<td>Education (years): mean ± SD</td>
<td>12.2 (3.42)</td>
<td>12.7 (2.92)</td>
</tr>
<tr>
<td>Drug of choice</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol</td>
<td>30</td>
<td>–</td>
</tr>
<tr>
<td>Metamphetamine</td>
<td>8</td>
<td>–</td>
</tr>
<tr>
<td>Cocaine/crack</td>
<td>16</td>
<td>–</td>
</tr>
<tr>
<td>Cannabis</td>
<td>13</td>
<td>–</td>
</tr>
<tr>
<td>Prior detoxification</td>
<td>2 (1.2)</td>
<td>–</td>
</tr>
<tr>
<td>treatments: mean ± SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Years of abuse: mean ± SD</td>
<td>11 (3.7)</td>
<td>–</td>
</tr>
<tr>
<td>Duration of abstinence</td>
<td>180 (11.9)</td>
<td>–</td>
</tr>
<tr>
<td>(in days): mean ± SD</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI</td>
<td>10 (1.3)</td>
<td>4 (0.6)</td>
</tr>
<tr>
<td>BAI</td>
<td>8.3 (1.1)</td>
<td>2 (0.3)</td>
</tr>
</tbody>
</table>

Numbers in parentheses are standard deviations.
Detoxified polysubstance abusers with alcoholism (PSA)

PSA were selected and tested shortly before completing a drug rehabilitation treatment at the Mid-Eastern Center for Chemical Abuse (MECCA). All PSA were paid for their participation in gift certificates at an hourly rate that was identical to that of CONT. The selection criteria for PSA were (1) meeting the Diagnostic and Statistical Manual version IV (DSM-IV) criteria for substance dependence; (2) absence of psychosis; and (3) no documented head injury or seizure disorder.

**Procedure**

All PSA were in-patients admitted to MECCA for detoxification and treatment. They had serious substance abuse problems requiring professional intervention, which was the reason for their admission.

The duration of their abstinence from substance use was known from the length of their stay at MECCA. The duration varied from one individual to another, but the minimum period was 15 days. Each PSA was tested at the end of treatment, i.e. shortly before discharge. Thus, at the time of testing, the PSA were no longer in acute withdrawal or taking any medication to control withdrawal.

PSA were checked routinely for substance abuse at MECCA during their treatment. They were also breathalyzed and subject to urine toxicology screening for opiates, stimulants and marijuana immediately before testing. Therefore, we can be reasonably sure that there was no use of substances during the entire period of abstinence.

The Structured Clinical Interview for DSM-IV (SCID-IV) was used to assign Axis I diagnoses (including alcohol and other drug abuse and/or dependence). Drugs of abuse, the duration of abstinence, the number of times in treatment and the total number of years of abuse were obtained from interviews. The complete screening and psychological testing procedures, including inclusion/exclusion criteria and the assessment of comorbid psychopathologies, are described elsewhere in more details (Bechara et al. 2001).

**Beck Depression Inventory (BDI)** (Beck 1987)

The level of depression was assessed by BDI, a 21-item self-report survey asking about depressive symptoms. Each item is scored from 0 to 3, with higher scores indicating greater symptom severity.

**Beck Anxiety Inventory (BAI)** (Beck & Steer 1993)

The level of anxiety was assessed using the BAI, also a 21-item self-report survey with the same ratings.

**Statistical analyses**

We used the time taken to respond to a target (RT) as the primary dependent measure. RTs less than 100 ms (anticipation) were excluded from our analyses. A high number of false alarms (response to a distracter) combined with a low number of hits (response to a target) indicate an inability to discriminate targets from distracters. False alarms alone cannot be interpreted as an indication of disinhibition. Therefore, we performed a signal detection analysis to distinguish discrimination (d') from decision bias (C) (Snodgrass & Corwin 1988; formulae are given in Appendix I). A d’ value of 0 or less indicates...
that subjects were either unable to discriminate targets from distracters, or they were not performing the task as instructed. A high d’ indicates good discrimination ability (e.g. more hits and less false alarms). When C is less than 0, this reflects a higher readiness to respond to any stimulus, i.e. more hits and less false alarms. Because decision bias (C) takes into account both hits and false alarms, it is a better indicator of disinhibition than false alarms alone. Thus a low C was considered a sign of disinhibition.

A group (PSA versus CONT, between) × target (alcohol versus neutral, within) × condition (shift versus no-shift, within) ANOVA was performed on RT, d’ and C. Cognitive biases are inferred from the group–target interactions. Shifting abilities are inferred from the group–condition interactions.

The acceptance or rejection of a hypothesis based on P-values alone has been shown to be problematic, because the P-values do not distinguish effect from sample size (Cohen 1994; Schmidt 1996). We followed the recommendation of the Task Force on Statistical Inference (Wilkinson 1999) and the APA (2001) and calculated effect sizes within 95% confidence intervals, and used these for our interpretation. The magnitude of the ANOVA effects was measured with a correlation (r_{effect size}) following the method developed by Rosnow & Rosenthal (2003). A r_{effect size} > 0.10 is generally considered as a small effect, > 0.30 as a moderate effect and > 0.50 as a large effect (Cohen 1988). A r_{effect size} < 0.10 corresponds to a negligible effect and is not reported in the results.

**RESULTS**

The composition of PSA and CONT groups were similar in age, education and gender. However, PSA were more depressed and anxious than CONT \[ t(1.58) = 11.9, P < 0.001, t(1.58) = 14.5, P < 0.001, \text{respectively}. \]

**Alcohol shifting task**

**Reaction times (see Table 2)**

Using reaction time (RT) as the dependent measure, a three-way ANOVA (group × target × condition) revealed a moderate effect of group [F\(_{1,58} = 7.14, P < 0.01, r_{effect size} = 0.33, CI = (0.08, 0.54)\], with PSA being slower than CONT in their processing speed when detecting targets. A large effect of ‘type of target’ also emerged from this analysis [F\(_{1,58} = 76.22, P < 0.001, r_{effect size} = 0.75, CI = (0.62, 0.85)\], with participants being slower to detect neutral than alcohol-related targets. Furthermore, the group–target interaction effect was moderate to large [F\(_{1,58} = 18.35, P < 0.001, r_{effect size} = 0.49, CI = (0.27, 0.66)\], Comparisons of the means revealed that the slowing down of PSA, relative to CONT, was more pronounced when the target stimuli were neutral as opposed to alcohol-related words (Table 2). In addition, the group–condition interaction effect was small to moderate [F\(_{1,58} = 3.56, P = 0.02, r_{effect size} = 0.29, CI = (0.04, 0.51)\]. Comparisons of these means indicated that the slowing down of PSA, relative to CONT, was more pronounced in the shift than in the non-shift conditions (Table 2).

**Discrimination (d’)** (see Table 2)

Comparing the means of hits and false alarms (not reported) revealed that PSA made more false alarms than CONT. They also made fewer hits, but the group difference was smaller. This preliminary analysis suggested that PSA had difficulties discriminating targets from distracters (low d’), and also they were more disinhibited (low C). This was supported by the three-way ANOVA (group × target × condition) on d’ and C. When considering d’, the ANOVA revealed a moderate effect of group [F\(_{1,58} = 6.35, P = 0.01, r_{effect size} = 0.31, CI = (0.07, 0.53)\], with PSA having lower discrimination ability than CONT. A moderate effect of type of target was found [F\(_{1,58} = 6.26, P = 0.02, r_{effect size} = 0.31, CI = (0.06, 0.52)\],

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Mean RTs, d’ and C for PSA and CONT on the Alcohol Shifting Task.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PSA</td>
</tr>
<tr>
<td></td>
<td>(n = 30)</td>
</tr>
<tr>
<td><strong>RTs</strong></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td></td>
</tr>
<tr>
<td>Non-shift</td>
<td>552.40 (86.90)</td>
</tr>
<tr>
<td>Shift</td>
<td>564.08 (91.25)</td>
</tr>
<tr>
<td>Type of target</td>
<td></td>
</tr>
<tr>
<td>Neutral</td>
<td>592.61 (91.02)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>523.87 (72.53)</td>
</tr>
<tr>
<td><strong>d’</strong></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td></td>
</tr>
<tr>
<td>Non-shift</td>
<td>2.10 (0.93)</td>
</tr>
<tr>
<td>Shift</td>
<td>1.92 (0.89)</td>
</tr>
<tr>
<td>Type of target</td>
<td></td>
</tr>
<tr>
<td>Neutral</td>
<td>1.93 (0.87)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>2.09 (0.95)</td>
</tr>
<tr>
<td><strong>C</strong></td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td></td>
</tr>
<tr>
<td>Non-shift</td>
<td>−0.22 (0.36)</td>
</tr>
<tr>
<td>Shift</td>
<td>−0.29 (0.41)</td>
</tr>
<tr>
<td>Type of target</td>
<td></td>
</tr>
<tr>
<td>Neutral</td>
<td>−0.28 (0.39)</td>
</tr>
<tr>
<td>Alcohol</td>
<td>−0.23 (0.38)</td>
</tr>
</tbody>
</table>

Numbers in parentheses are standard deviations.
with the discrimination being better when the targets were alcohol-related as opposed to neutral words. A small to moderate effect of condition was also found \( F_{1,SS} = 5.24, P = 0.03, r_{\text{effect size}} = 0.29, CI = (0.04, 0.50) \) with discrimination being better in the non-shift condition. The group–target interaction effect was small \( F_{1,SS} = 0.77, P = 0.38, r_{\text{effect size}} = 0.11, CI = (-0.14, 0.36) \). Comparisons of the means revealed that the impaired discrimination of PSA was more pronounced when the targets were alcohol-related as opposed to neutral words (Table 2).

**Decision bias (C) (see Table 2)**

The three-way ANOVA revealed a small effect of group \( F_{1,SS} = 2.51, P = 0.12, r_{\text{effect size}} = 0.20, CI = (-0.05, 0.43) \) with PSA expressing more decision bias than CONT (low C). It also revealed a moderate effect of type of target \( F_{1,SS} = 6.89, P = 0.01, r_{\text{effect size}} = 0.33, CI = (0.08, 0.54) \) with all participants showing a weaker decision bias when the targets were alcohol-related words (high C), and a small effect of condition \( F_{1,SS} = 1.30, P = 0.26, r_{\text{effect size}} = 0.15, CI = (-0.11, 0.39) \) with a stronger decision bias in the shift condition (low C). The group–target interaction effect was small \( F_{1,SS} = 2.59, P = 0.11, r_{\text{effect size}} = 0.21, CI = (-0.05, 0.44) \). Comparisons of the means revealed that PSA had a stronger decision bias than CONT (low C), especially when the targets were alcohol-related words (Table 2). The group–condition interaction effect was small \( F_{1,SS} = 1.26, P = 0.27, r_{\text{effect size}} = 0.15, CI = (-0.11, 0.39) \). Comparisons of the means revealed that the decision bias of PSA was more pronounced in the shift condition (low C, Table 2). At the beginning of the signal analysis, we noted that PSA had fewer hits and more false alarms than CONT. Thus, the decision bias of PSA can be due only to a higher number of false alarms. Taking decision bias as a sign of disinhibition, the signal detection analysis indicated that PSA were more disinhibited than CONT, especially when alcohol-related words were the targets, or when there was a shift in the target from one type of words to another.

**Relationship between clinical variables and cognitive measures**

When considering the correlation between the number of prior detoxification treatments, the duration of alcoholism, the number of days of abstinence, depression and anxiety scores and performance on the Alcohol Shifting task, we found a positive correlation between the duration of alcoholism and the measure of cognitive bias, assessed as the RT to neutral words minus the RT to alcohol-related words \( r = 0.45, P < 0.01, CI = (0.21, 0.65) \). This relationship corresponds to a moderate effect size. No other correlations reached significance.

**DISCUSSION**

The primary aim of the present study was to examine the presence of cognitive deficits and biases in alcohol dependent polysubstance abusers (PSA). We studied different mechanisms of executive control (i.e. response inhibition and shifting) and cognitive bias for alcohol-related words. The results indicate that PSA exhibit response inhibition and shifting deficits together with cognitive biases towards alcohol-related stimuli.

On a modified version of a go/no-go paradigm (the Alcohol Shifting task), in comparison with matched healthy participants (CONT), PSA took longer to respond to targets, especially when the targets were neutral words. This effect was greater when there was a shift in targets from one type of stimulus to another (e.g. from alcohol-related to neutral words, or vice versa). A signal detection analysis showed that the PSA were less able to discriminate targets from distracters, particularly when the targets were alcohol-related words. Furthermore, this analysis showed that the PSA had a lower C-value than CONT, especially when alcohol-related words were targets. This indicator of disinhibition (low C-value) observed in PSA was more pronounced when the targets shift from one type of stimulus to another. These findings reveal slow processing speed, poor target discrimination and deficits of response inhibition and mental flexibility in PSA. The effect size analyses indicated that these impairments were small to moderate. Reaction time analyses also suggested that PSA allocated more attentional resources to alcohol than to non-alcohol-related information. However, their discrimination and inhibition deficits were exacerbated when they had to detect alcohol-related words. The magnitude of these cognitive biases was also small to moderate.

The differences in reaction time on the Alcohol Shifting task indicate that PSA were slower than CONT in their ability to discriminate between targets and distracters, but this impairment was less pronounced when the targets were alcohol-related as opposed to neutral words. This effect could be attributed to an attentional bias towards alcohol-related cues (i.e. paying more attention to alcohol-related information). This is consistent with previous studies showing that individuals who abuse alcohol demonstrate heightened attention to alcohol cues (e.g. Johnsen et al 1994; Stetter et al 1995; Sharma et al 2001; Lusher, Chandler & Ball 2004). In these studies, alcoholics were slower than non-alcoholic controls in naming the colour of alcohol-related words on the Alcohol Stroop task. Another method used for determining whether attentional resources are captured by alcohol-related cues is the visual probe task, in which participants respond as quickly as possible to probe stimuli that are presented immediately after the display of a pair of words.
or pictures. In this task, heavy social drinkers respond faster to probes that appear in the location of alcohol-related than neutral pictures (Townshend & Duka 2001), thus suggesting that their attention is allocated preferentially to the spatial location of drug cues. Consistent with this idea, using a task in which individuals were asked to identify transient changes in visual scenes, people drinking ‘socially’ detected substance-related changes quicker than light and non-alcohol drinkers (Jones et al. 2003).

The presence of a cognitive attentional bias for alcohol cues in PSA is congruent with both the psychopharmacological and neuroanatomical perspectives. In a review, Franken (2003) emphasized that when conditioned drug stimuli are present they increase dopamine levels in the corticostriatal circuit, particularly in the anterior cingulate, amygdala and nucleus accumbens, which in turn serves to draw the subject’s attention towards the drug-related stimulus.

An alternative, but related, explanation for why PSA are especially slow in responding to neutral words might be because alcohol cues represent a source of interference for processing these stimuli. In other words, PSA may have difficulty disengaging their attention from such cues. In support, recent studies have emphasized the abnormal maintenance of attention to cigarette cues in smokers who craved cigarettes (Field, Mogg & Bradley 2004) and to alcohol cues in heavy social drinkers (Field et al. 2004). Consequently, it might take longer to attend to neutral words, because of the time needed to re-orientate attentional resources focused heavily on alcohol cues.

Another main finding from the signal detection analysis was that, on the Alcohol Shifting task, PSA were especially disinhibited when alcohol-related words had to be detected. Disinhibition of PSA was evidenced by a lower C-value, due to a greater number of false alarms (i.e. they responded more often to distracters). The PSA’s difficulty in inhibiting a response is in accordance with those we obtained recently using the Hayling task (Noël et al. 2001). In this task (Burgess & Shallice 1996), subjects were asked to give a word that made no sense at all in the context of a sentence in which the last very predictable word was missing. Recently detoxified alcoholics were slower than CONT, and they also gave more words related to the predictable one, thus indicating a response inhibition deficit (Noël et al. 2001). Our present results are also consistent with studies showing that detoxified polysubstance abusers made more perseverative errors in the Wisconsin Card Sorting task (Bechara et al. 2001).

In addition, we found that PSA took longer than CONT to respond, especially when they had to reverse a stimulus–reward association (i.e. shift condition) by responding when the target had just changed. This finding is in agreement with other studies showing that recently detoxified alcoholics exhibit a deficit of shifting in a variety of cognitive tasks (Noël et al. 2001; Hildebrandt et al. 2004). For instance, on the flexibility condition of the Stroop task, which requires subjects to alternate between two rules (i.e. to read the words rather than name its color when it is underlined), abstinent alcoholics took longer than healthy controls to respond during the interference condition (i.e. to name the colour of the print of a word printed in an incongruent colour) (Noël et al. 2001). Studies of patients with focal brain lesions, as well as functional neuroimaging studies, suggest that the anatomical substrate of reversal learning in humans involves the prefrontal cortex. Indeed, these studies suggest that the ventromedial prefrontal cortex, especially its more posterior sector, is critical for adjusting responses when the reinforcement value of stimuli changes (e.g. Fellows & Farah 2003; for a review, see Collette & Van der Linden 2002). In people dependent on alcohol, cocaine or heroin there are structural as well as functional abnormalities in ventral and orbital regions of the prefrontal cortex that might be responsible for deficits in shifting from one contingency to another (for a review, see Goldstein & Volkow 2002).

Regarding the potential influence of the high levels of depression and anxiety observed in PSA on their cognitive functioning, we did not find a significant correlation between cognitive performance and measures of BDI and BAI, which suggests that the observed deficits and biases in PSA are not linked to their symptoms of depression or anxiety. On the other hand, we found a positive correlation between the duration of alcoholism and attentional bias for alcohol cues, thus indicating that this cognitive bias may play a role in the severity of alcoholism.

In summary, our data indicate that PSA exhibit two kinds of cognitive impairment: that is, one relating to deficits in executive control (response inhibition and shifting), and the other relating to an increase in cognitive biases (alcohol-related stimuli capturing the attention).

We propose that impairments in response inhibition and shifting impairments, together with cognitive biases towards alcohol-related stimuli, are, at least partially, responsible for the maintenance of alcoholism or for relapse after a period of abstinence. In particular, PSA’s cognitive biases could result in alcohol-related representations breaking through into awareness (i.e. being loaded into working memory), and thus being expressed as intrusive thoughts. Given the potential influence of intrusive thoughts and craving on alcohol use and relapse (May et al. 2004), the PSA’s abilities to inhibit dominant responses, to re-orient attention, to select an alternative response and to shift from one thought to another may influence the propensity of an individual to
relapse. Our approach is compatible with neurobiological and functional neuroimaging-based models proposing that drug-seeking behaviour may be due to two related processes: (1) an increased in the incentive motivational qualities of drug and associated stimuli (due to subcortical dysfunction) and (2) impaired inhibitory control (due to frontal cortical dysfunction) (for a review, see Lubman, Yücel & Panteles 2004).

Although the presence of an attention bias for alcohol cues is generally viewed as supporting the ‘incentive-sensitization theory’, which posits that, in drug addicts, these stimuli capture attention and are perceived as very ‘wanted’ (Robinson & Berridge et al. 1988): we argue that, in humans, executive-regulatory function deficits are also necessary to elicit compulsive drug use. Response inhibition and mental flexibility might also be required when automatized, stimulus-bound, stereotyped, effortless drug use behaviors leading to the continuance of alcoholism are impeded by a person attempting to quit drinking (see Tiffany, 1990).

There are three limitations in the present study that call for future investigations. First, the modified go/no go paradigm used does not allow the determination of the nature of the attention bias observed in PSA. In other words, is this bias pre-conscious, conscious or both? Secondly, we were unable to identify the effect of other psychoactive substances (i.e. cannabis, ecstasy, cocaine and heroin) on PSA's cognitive functioning. We did not compare their cognitive performance in relation to different drugs, and therefore we cannot isolate the effect of each drug on cognition. However, compared with many other drugs, alcohol has a more detrimental effect on executive functions (for a comparison between cocaine and alcohol addiction, see the recent paper by Goldstein et al. 2004).

Finally, we could not determine whether the cognitive deficits and biases found in PSA existed before they became alcoholics. These deficits and biases could have been a cause of their addiction rather than being a consequence of them. In support of the hypothesis that some inhibition/flexibility deficits could predate the onset of drug dependence, a recent fMRI study has compared youths with and without a family history of alcoholism, using a go/no-go procedure, and has demonstrated that youths with a family history of alcoholism showed less inhibitory frontal response than other youths, thus suggesting that inhibition deficits could be predisposing factors to alcohol abuse (Schweinsburg et al. 2004).

To summarize, the findings of the present study showed that response inhibition and shifting were impaired in PSA. In addition, these patients exhibit cognitive biases for alcohol-related cues. These cognitive impairments are conceived as factors involved in the poor ability of PSA to maintain abstinence and control their compulsive drug use.

Acknowledgements

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References


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**APPENDIX I**

Formula used for the signal detection analysis (Snodgrass & Corwin 1988).

**Hit and false alarm probability**

\[ P(\text{‘hit’}) = \left( \frac{\Sigma \text{answer}}{\Sigma \text{target}} \right) \]

\[ P(\text{‘false alarm’}) = \left( \frac{\Sigma \text{answer}}{\Sigma \text{non-target}} \right) \]

**Corrected probability**

\[ P_{\text{corrected}}(\text{‘hit’}) = \left( \frac{\Sigma \text{answer} + 0.5}{\Sigma \text{target} + 1} \right) \]

\[ P_{\text{corrected}}(\text{‘false alarm’}) = \left( \frac{\Sigma \text{answer} + 0.5}{\Sigma \text{non-target} + 1} \right) \]

**Discrimination and decision bias**

\[ \Delta = Z[P_{\text{corrected}}(\text{‘hit’})] - Z[P_{\text{corrected}}(\text{‘false alarm’})] \]

\[ C = -0.5 * \left[ Z[P_{\text{corrected}}(\text{‘hit’})] + Z[\{P_{\text{corrected}}(\text{‘false alarm’})]\} \right] \]

‘\Sigma answer’ is the total number of responses, ‘\Sigma target’ the total number of targets, and ‘\Sigma non-target’ the total number of distractors. \( P(\text{‘hit’}) \) is the probability to respond to a target. \( P(\text{‘false alarm’}) \) is the probability of respond to a distracter. \( Z(p) \) is the quantile function of the normal distribution. Z are calculated on \( P_{\text{corrected}} \) to avoid infinite value when \( P = 1 \).