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Albertella, L.; Copeland, J.; Pearson, D.; Watson, P.; Wiers, R.W.; Le Pelley, M.E.

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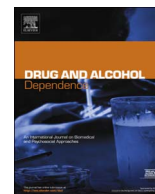
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Full length article

Selective attention moderates the relationship between attentional capture by signals of nondrug reward and illicit drug use



Lucy Albertella^{a,b,*}, Jan Copeland^b, Daniel Pearson^a, Poppy Watson^{a,c,d}, Reinout W. Wiers^{c,d}, Mike E. Le Pelley^a

^a School of Psychology, UNSW Sydney, Sydney, Australia

^b National Cannabis Prevention and Information Centre, UNSW Sydney, Sydney, Australia

^c Addiction Development and Psychopathology (ADAPT) lab, Department of Developmental Psychology, University of Amsterdam, Amsterdam, The Netherlands

^d Amsterdam Brain and Cognition, University of Amsterdam, Amsterdam, The Netherlands

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ABSTRACT

Background: The current study examined whether cognitive control moderates the association between (non-drug) reward-modulated attentional capture and use of alcohol and other drugs (AOD).

Methods: Participants were 66 university students who completed an assessment including questions about AOD use, a visual search task to measure value-modulated attentional capture, and a goal-directed selective attention task as a measure of cognitive control.

Results: The association between the effect of value-modulated attentional capture and illicit drug use was moderated by level of cognitive control. Among participants with lower levels of cognitive control, value-modulated attentional capture was associated with illicit drug use. This was not the case among participants with higher levels of cognitive control, who instead showed a significant association between illicit drug use and self-reported impulsivity, as well as alcohol use.

Conclusions: These results provide support for models that view addictive behaviours as resulting from interaction and competition between automatic and more reflective processes. That is, the mechanisms that ultimately drive addictive behaviour may differ between people low or high in cognitive control. This has important implications for understanding the development and maintenance of substance use disorders and potentially their treatment and prevention.

1. Introduction

According to dual-process models of addictive behaviours (for a review, see Stacy and Wiers, 2010), problematic substance use arises when relatively automatic, impulsive processes begin to dominate reflective processes in addiction-related decision making. This imbalance is considered to arise primarily from repeated exposure to alcohol and/or other drugs (AOD), which (through various proposed mechanisms) acts to strengthen the influence exerted by automatic appetitive processes over behaviour, relative to that exerted by reflective processes. For instance, a number of models propose that repeated and heavy exposure to AOD can sensitise the automatic system via the operation of learning processes, rendering an individual especially susceptible to maladaptive control by drug-related cues (Robinson and Berridge, 2000; Wiers et al., 2007). Specifically, it has been argued that through repeated pairing of certain stimuli with the rewarding consequences of taking a drug, those previously neutral stimuli come to

acquire *incentive salience*, subsequently attracting attention and evoking powerful approach responses in their own right (Berridge et al., 2009; Robinson and Berridge, 2000). Conversely, repeated exposures to AOD are proposed to weaken the reflective system, rendering it less able to oppose the influence exerted by the progressively stronger automatic system.

Aside from the long-term effects of substance use on both automatic and reflective processes, most models agree that premorbid individual differences in both type of processes can also influence the development and maintenance of addictive behaviours. Indeed, animal studies have shown individual differences in incentive salience attribution to play a role in predisposing individuals to addictive behaviours (Flagel et al., 2009). Likewise, individual differences in cognitive control are associated with future drug use (Squeglia et al., 2014). Especially strong support for dual-process theories comes from studies showing that individual differences in cognitive control moderate the relationship between automatic responding to AOD cues and actual AOD use. For

* Corresponding author at: School of Psychology, UNSW Sydney, NSW 2052, Australia.
E-mail address: l.albertella@unsw.edu.au (L. Albertella).

instance, Houben and Wiers (2009) found that positive implicit alcohol associations predicted alcohol use only in participants with poor response inhibition (i.e., weak cognitive control), whereas implicit alcohol associations were not related to future alcohol use among participants with good inhibitory control. Other findings have found a similar moderating effect of working memory on the ability of automatic AOD-related associations to predict later AOD use (Grenard et al., 2008; Thush et al., 2008), although some inconsistencies have been observed (for a review, see Wiers et al., 2015).

As suggested above, many studies have shown that AOD use and disorders are associated with abnormal attentional biases towards drug-related stimuli (see Field and Cox, 2008; for a review). Notably, recent studies have also linked AOD use and disorders with abnormal attentional biases for non-drug reward-related stimuli. One such study found that adolescents who reported higher levels of alcohol, tobacco, and cannabis use showed greater attentional engagement with cues that predicted non-drug reward (van Hemel-Ruiter et al., 2013). In another study, people in methadone treatment for opiate dependence showed significantly greater attentional capture stimuli related to non-drug reward in a visual search task, compared to healthy controls (Anderson et al., 2013). On the basis of this finding, Anderson et al. (2013) suggested that previous findings of attentional biases toward AOD-related cues in people with substance use disorders may in fact have arisen from a pre-existing attentional bias toward cues associated with reward in general. In other words, those individuals with a general predisposition towards automatic attentional capture by reward-related stimuli may be more susceptible to developing AOD-use disorders.

Individual differences in attentional capture by stimuli associated with non-drug reward may be considered to reflect a vulnerability within automatic processes, somewhat parallel to individual differences in attribution of incentive salience to reward-associated cues in the animal literature. Importantly, as mentioned above, animal studies suggest that such individual differences in attribution of incentive salience may indicate individual vulnerability to developing AOD-use problems. According to dual-process theories, the degree to which such individual differences in attentional capture by stimuli associated with reward actually influence behaviour would depend on the degree of cognitive control available to the individual. To date, however, studies have focused on automatic/implicit behaviour and attitudes toward AOD-related cues only; no existing study has explored whether cognitive control capacity might also moderate the relationship between attentional capture (by cues associated with non-drug reward) and AOD use. Such a moderating effect would strongly support existing dual-process accounts and extend them by highlighting the role of individual differences in attribution of incentive salience to cues associated with rewarding outcomes in general, and not just drug-related rewards.

Furthermore, existing studies exploring individual differences in attentional capture by stimuli associated with non-drug reward have used procedures in which participants are initially trained that orienting attention to the critical stimuli yields reward. The resulting attentional biases could therefore reflect instrumental conditioning of ‘attentional habits’, where reward reinforces the instrumental response of attending to a particular stimulus (Anderson, 2016; Le Pelley et al., 2016). As such, these studies cannot be considered to parallel animal studies of incentive salience attribution, which have typically used Pavlovian rather than instrumental conditioning procedures (e.g., Flagel et al., 2008). A visual search procedure recently developed by Le Pelley et al. (2015) allows us to overcome this issue. This study used a gaze-contingent procedure in which eye-movements were the means by which participants made their responses, and also provided the measure of attention. Eye-movements are tightly coupled with shifts of attention—an eye-movement to a given location is always preceded by a spatial shift of attention that location (Deubel and Schneider, 1996)—and so provide an excellent, online index of attention. On each trial of Le Pelley et al.’s (2015) procedure, participants have to make an

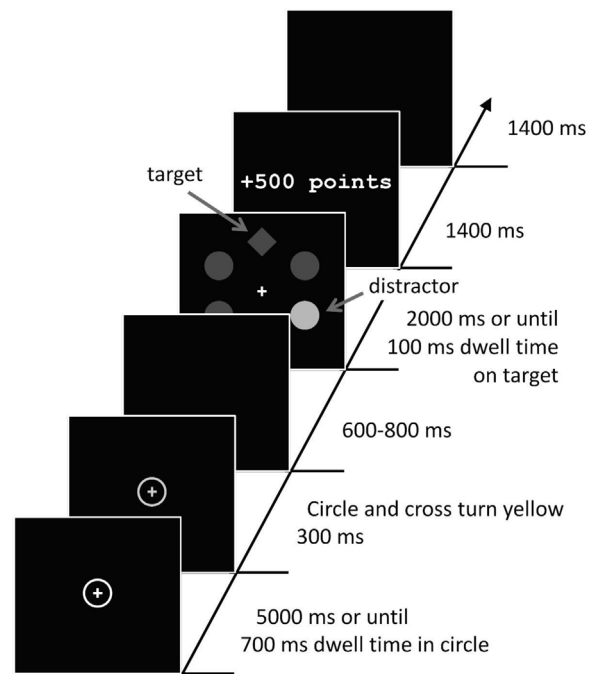


Fig. 1. Sequence of trial events in the value-modulated attentional capture (VMAC) task. On each trial, participants responded by moving their eyes to the diamond target in the search display. One of the non-target circles could be a colour singleton distractor. Fast, correct responses received monetary reward, depending on the distractor colour. A high-value distractor colour reliably predicted large reward (500 points); a low-value colour reliably predicted small reward (1 point); on distractor-absent trials, large and small rewards were equally likely. If any gaze fell within a small region of interest (ROI) surrounding the distractor (or, on distractor-absent trials, an equivalent ROI positioned around a randomly-chosen circle), the trial was deemed an omission trial and no reward was delivered.

eye-movement (a saccade) to a diamond-shaped target among circles, as quickly as possible. On most trials, one of the non-target circles is coloured, either red or blue (all other shapes are grey; see Fig. 1); hence this is an example of an additional singleton task (Theeuwes, 1991, 1992). The colour-singleton circle is referred to as the distractor. The colour of the distractor on a particular trial signals the magnitude of reward that is available. As such, these colours constitute Pavlovian signals of reward magnitude. Crucially, under these conditions the reward-predictive stimulus (the coloured distractor) is not the target to which participants must orient their gaze (or attention) in order to receive reward. In fact, the task is arranged such that, if participants look at or near the distractor prior to looking at the target, the reward on that trial is omitted. Nevertheless, participants are more likely to look at the distractor when it appears in the colour signalling high reward than the colour signalling low reward (Failing et al., 2015; Le Pelley et al., 2015; Pearson et al., 2015; Pearson et al., 2016), a finding referred to as *value-modulated attentional capture* (VMAC). Since participants are never rewarded for looking at the distractor in this task, there is no reinforcement for the instrumental response of looking at the distractor. Consequently, the VMAC procedure used by Le Pelley et al. (2015) provides a clearer demonstration of the modulation of attentional capture as a result of Pavlovian reward prediction.

The current study used the VMAC procedure to examine how (non-drug) reward-modulated attentional capture is related to AOD use in a sample of university students, and whether cognitive control (measured using a goal-directed selective attention task) moderates such a relationship. In this task, participants are required to select a target stimulus while ignoring a distractor stimulus. As the target and distractor stimuli are of similar salience in this task, performance may be considered a reflection of the extent to which participants are applying top-down, goal-directed attention to select the target and

ignore the distractor on each trial.

2. Material and methods

2.1. Ethical approval

Ethical approval was obtained from the UNSW Sydney Human Research Ethics Committee. All participants provided informed consent prior to participating.

2.2. Participants and apparatus

Participants were 66 undergraduate students, who received course credit for taking part, as well as payment (mean = \$11.13 AUD, SD = \$3.48) depending on their performance in the VMAC task. Participants were tested individually using a PC with a 23-in. monitor attached to a Tobii TX300 eye-tracker, with 300 Hz temporal resolution. No participant reported having used an illicit drug in the 24 h prior to participation.

2.3. Value-modulated attentional capture (VMAC) task: apparatus, stimuli, and procedure

The method for the VMAC task was very similar to that of [Le Pelley et al. \(2015, Experiment 3\)](#). During this task, participants were positioned in a chin-rest 60 cm from the screen. Throughout the task, the program controlling stimulus presentation requested data from the eyetracker every 10 ms, and participants' gaze location was defined as the average of the locations recorded in the most recent 10 ms sample.

Each trial included a fixation display, a search display, and a feedback display ([Fig. 1](#)). All stimuli were presented on a black background. The fixation display consisted of a white cross in a white circle (diameter 3.0° visual angle), presented in the centre of the screen. The search display comprised six filled shapes (2.3 × 2.3° visual angle), arranged at equal intervals around an imaginary circle of diameter 10.1°. Five of these shapes were circles and one was a diamond. Four of the circles were filled in grey, as was the diamond. The remaining circle (the distractor) was filled either in red, blue, or the same shade of grey as the other shapes. RGB values for the colours used were: red [255, 0, 0], blue [87, 87, 255], and grey [70, 70, 70]. The resulting red and blue values had similar luminance (~42.5 cd/m²), which was higher than that of the grey (~32 cd/m²).

Red and blue were assigned to act as high-value and low-value colours in a counterbalanced fashion across participants. The experiment consisted of 10 training blocks, with each block containing 20 trials with a distractor rendered in the high-value colour, 20 trials with a distractor in the low-value colour, and 8 distractor-absent trials, on which there was no coloured circle in the display. Trials occurred in random order, and target location and distractor location were randomly determined on each trial, with the constraint that the distractor could never appear in a location adjacent to the target. Participants took a short break after every two blocks.

On each trial, a small circular region of interest (ROI) (diameter 3.5°) was defined around the diamond target and a larger ROI (diameter 5.1°) was defined around the distractor. A response was registered when 100 ms of gaze-time inside the target ROI had accumulated. If a response was registered within 600 ms of the onset of the stimulus display, and if no gaze was recorded in the distractor ROI prior to the response, then a reward was delivered. If the high-value distractor was present, 500 points were awarded; if the low-value distractor was present, 10 points were awarded; on distractor-absent trials, a reward of 10 points or 500 points was equally likely. If any gaze fell inside the distractor ROI prior to a response being registered, zero points were earned. When this happened, the trial was recorded as an *omission trial*. On distractor-absent trials, one of the grey circles (that was not adjacent to the target) was chosen at random; gaze falling inside an ROI around

the selected grey circle caused an omission trial in exactly the same way as if the selected circle had been a distractor. Responses with response times slower than 600 ms also earned zero points.

The session began with 8 practice trials. On these trials, the distractor was yellow and no rewards were available. Participants were then told that on subsequent trials they would earn points depending on 'how fast and accurately' they moved their eyes to the diamond. They were also told that the number of points that they earned would determine their monetary pay-off at the end of the experiment, which would typically be between \$5 and \$15, but were not told the exact conversion rate between points and money. [This was $payment (AUD) = 0.00015 \times points - 1$].

Each trial began with the presentation of the fixation display, on which participants' gaze location was superimposed as a small yellow dot. Once 700 ms of gaze-time had accumulated inside the circle surrounding the fixation cross, or if 5 s had passed, the cross and circle turned yellow and the dot marking gaze location disappeared. After 300 ms the screen blanked, and after a random interval of 600–800 ms, the search display appeared. The trial terminated when a response was registered or after 2 s (timeout). The feedback display then appeared for 1400 ms, which showed the reward earned on the current trial (either 0 points, 10 points, or 500 points) and total earnings so far. If response time was greater than 600 ms, then the message 'Too slow' also appeared. Inter-trial interval was 1400 ms.

2.4. Goal-directed selective attention (GDSA) task: stimuli and procedure

Following the VMAC task, participants completed a goal-directed selective attention (GDSA) task presented using Inquisit Millisecond Software. On each trial, stimuli appeared in two of the four corner positions of the screen (each corner position was equidistant from the screen centre). One of these stimuli was a target (a black square frame, side length 4.2° visual angle), and the other was a distractor (a black equilateral triangle frame, side length 4.7°). The screen background was light grey. Participants' task was to indicate whether the target stimulus appeared on the left or right side of the screen by pressing a corresponding key on the keyboard (C or M respectively) as quickly as possible. Since target and distractor stimuli were of similar salience in this GDSA task, good performance relied on participants using top-down, goal-directed attention to select the target and ignore the distractor on each trial. Our measure of cognitive control was given by mean response time on the GDSA task (termed GDSA score), with lower scores indicating greater cognitive control (top-down control of selective attention).

Each display remained on the screen until a response was made, at which point the screen blanked. The inter-trial interval was 4000 ms. Target and distractor location were determined randomly on each trial. There were 64 trials in total, with a short break after 32 trials.

2.5. Other procedures/measures

Following the GDSA task, participants completed a questionnaire presented using Inquisit Millisecond Software. This questionnaire asked about general demographic information, participants' use of tobacco, alcohol, and other drugs, and impulsivity. Specifically, for each substance, participants were asked if they had ever used it and, if so, if they had used it in the past month. Participants who responded that they had used a substance in the past month were further asked about the number of days of use in the last month. Impulsivity was measured using the Barratt Impulsiveness Scale (BIS-11; [Patton and Stanford, 1995](#)), a thirty-item questionnaire designed to assess self-reported impulsiveness.

2.6. Data analysis

Preliminary data-screening for the VMAC task followed our previous

protocols when analysing data from this procedure (Le Pelley et al., 2015; Pearson et al., 2015; Pearson et al., 2016). Specifically, the first two trials, and the first two trials after each break, were discarded. Timeouts were also discarded (4.7% of remaining trials). Finally, we also excluded all trials on which valid gaze location was registered in less than 25% of samples between presentation of the search display and registering of a response (2.8% of remaining trials). For included trials, averaging across participants, valid gaze location was registered in 95.3% (SD = 7.4%) of samples, suggesting high fidelity of the gaze data on these trials.

As in previous work with this procedure (Le Pelley et al., 2015; Pearson et al., 2015; Pearson et al., 2016), our measure of attentional capture in the VMAC task was the proportion of omission trials for displays featuring high- versus low-value distractors; that is, how often participants looked at each type of distractor and hence caused omission of the reward. More specifically, we were interested in the difference in the proportion of omissions for trials featuring high- and low-value distractors, which was calculated by subtracting the proportion of omissions for trials with a low-value distractor from that for trials with a high-value distractor. A larger score (reflecting a greater difference in omissions between high- and low-value conditions) suggests greater attentional capture by the distractor associated with high reward compared to that paired with low reward. That is, a higher score indicates a greater influence of reward prediction on attentional capture. As noted earlier, GDSA score (our index of top-down attentional control) was given by mean correct response time on the GDSA task, with higher scores indicating poorer top-down control.

First, two overall Poisson regressions were run – one on each dependent variable of interest: 1) alcohol use (number of days alcohol was consumed in the last month) and 2) illicit drug use (number of illicit drugs ever used – this measure of drug use has been shown to be associated with impulsivity-related measures, including delay-discounting (Kollins, 2003) and personality traits in non-clinical, university populations (Franken and Muris, 2006; Newbury-Birch et al., 2000)). Poisson regressions were conducted because both dependent variables (alcohol use and illicit drug use) were count variables and fitted a Poisson distribution. In both of these models, independent variables were: VMAC score (the difference in the proportion of omissions between high- and low-value trials); GDSA score (our proxy measure of cognitive control); the interaction between VMAC and GDSA scores; sex; age; self-reported impulsivity (overall BIS score); tobacco use (number of days tobacco was smoked in the past month); and illicit drug use or alcohol use (depending on model). These variables were entered to control for their influence due to research suggesting their influence on attention and/or value-related attentional capture specifically (Anderson et al., 2011; Heishman et al., 2010; Roper et al., 2014). Mean VMAC and GDSA scores were centred around the mean to avoid multicollinearity in the interaction. A significant interaction effect was followed up by splitting participants into two groups based on GDSA score (median split): a high- and a low cognitive control group, and re-running the above regression looking at the variable in question as well as entering any other variables with $p < 0.05$. Importantly, a) there was no evidence of multicollinearity in any of the regression models (all variable inflation factors < 2) and b) slight overdispersion (in the alcohol use model, 4.5) was addressed by using the robust estimator covariance matrix and Pearson chi-square scale parameter method for all analyses.

3. Results

Participants had a mean age of 19.2 years (SD = 1.9; range 18–31), and 62% were female. Median alcohol use was 2 days (range 0–16) and median drug use was 0 (range 0–4). Twenty-eight percent of participants had ever used illicit drugs, with the maximum number of illicit drugs ever used being 4. The most commonly used illicit drug was cannabis, with 27% of participants having ever used it. Mean BIS-11

Table 1
Results of overall Poisson regression on illicit drug use.

	B	S.E.	Wald	Sig
Sex	−0.68	0.49	1.92	0.166
Age	0.24	0.05	20.14	0.000
Tobacco use	−0.05	0.06	0.54	0.462
Response time	−0.00	0.00	0.07	0.788
VMAC score	2.98	2.06	2.10	0.148
Interaction	0.06	0.03	4.58	0.032
BIS	0.06	0.02	6.81	0.009
Alcohol use	0.11	0.04	6.46	0.011

Note: Bold font indicates variables that have a significant association with the outcome, $p < 0.05$. 'BIS': overall score – Barratt Impulsiveness Scale Version 11 (Patton and Stanford, 1995). 'Alcohol use': number of days used alcohol in the past month. 'Tobacco': number of days smoked tobacco in the past month. 'Response time': mean response time on GDSA task. 'VMAC score': difference in the proportion of omissions between high- and low-value trials.

total score was 64.1 (SD = 8.8; maximum possible score = 120). Six participants reported using an illicit drug in the past month (cannabis), of whom none reported use in the past 48 h. Re-running all analyses while excluding these six participants returned the same results.

Across all participants, the mean proportion of omission trials for displays featuring a high-value distractor was 0.158 (SEM = 0.014), and for displays featuring a low-value distractor was 0.119 (SEM = 0.012). This difference was significant, $t(65) = 3.24$, $p = 0.002$. So averaging across all participants there was strong evidence of an influence of reward prediction on attentional capture, with greater capture by the high-value distractor than the low-value distractor. That is, across all participants there was a clear VMAC effect.

The alcohol use model was not significant overall ($p = 0.250$). Within this model, tobacco use was the only independent variable significantly associated with alcohol use, with greater tobacco use being significantly associated with greater alcohol use (Wald Chi-Square = 23.9, $p < .001$).

In contrast, the illicit drug use model was significant overall ($p < .001$). Table 1 presents the results from the Poisson regression on illicit drugs. Table 2 presents the results of follow-up regressions on illicit drugs. Here, older age (Wald Chi-Square = 20.1, $p < .001$), greater overall BIS score (Wald Chi-Square = 6.8, $p = 0.009$), and more frequent alcohol use (Wald Chi-Square = 6.5, $p = 0.011$) were significantly associated with having used more illicit drugs. More importantly, the interaction between VMAC score and GDSA score was significant (Wald Chi-Square = 4.6, $p = 0.032$). Follow-up Poisson regressions conducted

separately for each GDSA group (high cognitive control and low

Table 2
Results of follow-up Poisson regressions on illicit drug use, split by group (high cognitive control versus low cognitive control).

	B	S.E.	Wald	Sig
High control				
Age	0.24	0.04	43.52	0.000
VMAC score	0.87	3.83	0.05	0.820
BIS	0.04	0.02	5.20	.023
Alcohol use	0.10	0.04	4.78	0.029
Low control				
Age	0.57	0.47	1.48	0.224
VMAC score	6.94	1.54	20.40	.000
BIS	0.16	0.09	3.32	0.068
Alcohol use	−0.13	0.10	1.63	0.201

Note: Bold font indicates variables that have a significant association with the outcome, $p < 0.05$. 'BIS': overall score – Barratt Impulsiveness Scale Version 11 (Patton and Stanford, 1995). 'Alcohol use': number of days used alcohol in the past month. 'Tobacco': number of days smoked tobacco in the past month. 'Response time': mean response time on GDSA task. 'VMAC score': difference in the proportion of omissions between high- and low-value trials.

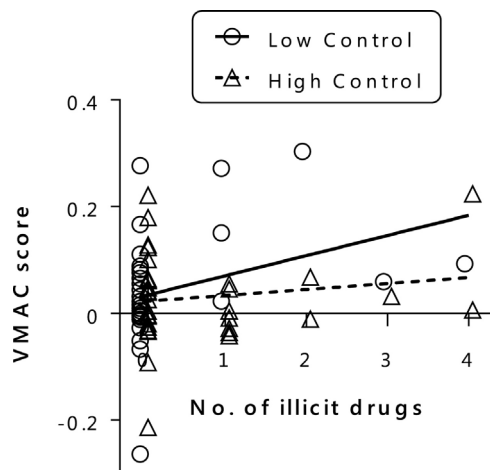


Fig. 2. VMAC score (see text for details) as a function of the number of types of illicit drugs ever used. Triangles show data for participants whose response times in the goal-directed selective attention task (GDSA score) were faster than the median (high cognitive control group), suggesting relatively good cognitive control. Circles show data for participants whose GDSA scores were higher than the median (low cognitive control group), suggesting relatively poor cognitive control. Lines show lines of best fit for each group.

cognitive control) thus included age, BIS score, alcohol use, and VMAC score. Both group models were significant in predicting illicit drug use overall (High, $p = 0.001$; Low, $p = 0.009$). In the high cognitive control group model, older age (Wald Chi-Square = 43.5, $p < 0.001$), more frequent alcohol use (Wald Chi-Square = 4.8, $p = 0.029$), and overall BIS score (Wald Chi-Square = 5.2, $p = 0.023$) were associated with illicit drug use. VMAC score was not (Wald Chi-Square = 0.052, $p = 0.820$). In the low cognitive control group, illicit drug use was significantly associated only with greater VMAC score (Wald Chi-Square = 20.4, $p < 0.001$), with a trend towards a significant association with BIS score (Wald Chi-Square = 3.3, $p = 0.068$). Fig. 2 shows a bivariate plot of VMAC scores as a function of illicit drug use, along with a line of best fit, for each group separately. This bivariate correlation was significant in the low cognitive control group, $r = 0.468$, $p = 0.006$ and not in the high cognitive control group, $r = -0.036$, $p = 0.843$.

Since there were relatively few participants who reported having used multiple types of illicit drugs, we followed up this analysis—which quantified illicit drug use as a Poisson count—with a similar analysis in which drug use was instead treated as a binary variable, distinguishing between participants who reported never having used illicit drugs (coded as 0), versus those who reported ever having used one or more types of illicit drug (coded as 1). The results of this analysis were very similar to those of the Poisson regression described in the previous paragraph. Table S1 in Supplementary Online Materials shows the detailed results of this analysis. Overall, the model fit was significant, Chi Square = 25.0, $p = 0.002$. Older age ($B = 0.814$, $p = 0.017$) and overall BIS score ($B = 0.094$, $p = 0.029$) were significantly associated with being more likely to have used illicit drugs. Importantly—and as for the Poisson regression—the interaction between VMAC and GDSA score was significant ($B = 0.220$, $p = 0.014$). Follow-up binary logistic regressions were conducted for each GDSA group. Both group models were significant in predicting likelihood of illicit drug use overall (high cognitive control group, $p = 0.009$; low cognitive control group, $p = 0.017$). For the high cognitive control group, there was a trend towards alcohol being associated with illicit drug use ($B = 0.20$, $p = 0.068$), and VMAC was not significant ($B = -5.1$, $p = 0.390$). For the low cognitive control group, illicit drug use was significantly associated only with VMAC score ($B = 17.7$, $p = 0.030$).

4. Discussion

Overall, participants were more likely to look at a distractor that signalled availability of high reward as compared to one that signalled availability of low reward, even though looking at the distractor led to omission of the reward. This replicates the value-modulated attentional capture (VMAC) effect previously reported (Le Pelley et al., 2015; Pearson et al., 2015, 2016) and suggests that Pavlovian signals of reward are more likely to elicit automatic attentional capture even when this capture is counterproductive to participants' goal of maximising their payoff.

Importantly, the VMAC effect was related to participants' illicit drug use (in terms of the number of illicit drugs they had ever used) only among participants showing relatively poor goal-directed selective attention (low cognitive control group); VMAC was not associated with illicit drug use among participants performing better on this latter measure. In other words, the association between VMAC and illicit drug use was moderated by participants' goal-directed selective attention ability.

The association between illicit drug use and VMAC seen in the low cognitive control group may be interpreted in several ways. First, it may be that exposure to illicit drugs leads to greater reward-related attentional capture, perhaps via sensitisation of neural pathways related to reward processing, but this may be expressed only in those with poor cognitive control. Indeed, the incentive-sensitisation model of addiction (Robinson and Berridge, 2000) proposes that addictive drugs may cause changes in reward-related brain areas to make them become more responsive to reward in general (i.e., not just to drug reward) and cues associated with the availability of reward (Robinson and Berridge, 2000). Animal research supports this possibility; for instance, rats pre-exposed to amphetamine show enhanced responding for a non-drug reward (in extinction) in the presence of a stimulus previously paired with sucrose (through Pavlovian conditioning), compared with rats that have not been pre-exposed to amphetamine (Wyvell and Berridge, 2001). Importantly, animals with such a predisposition (sign-trackers) have been shown to be more susceptible to drug-induced sensitisation of psychomotor behaviour (Flagel et al., 2008).

An alternative possibility is that greater reward-modulated attentional capture in general (i.e., not specific to drug reward) is associated with a greater risk of illicit drug exposure, particularly in those with less cognitive control (cf. Wiers et al., 2015). It is important to note however that this is not saying that cognitive control per se is related to less drug use. In fact, the present data suggest that the high cognitive control group used more drugs, albeit not significantly (mean of 0.67 vs 0.36, $p = 0.115$). Rather, the mechanisms that ultimately influence behaviour may differ between groups. Indeed, as explained by Hofmann et al. (2008), motivation also plays a significant role in behaviour, with explicit goals and motivations playing a greater role in influencing behaviour among those with higher cognitive control. Interestingly, self-reported impulsivity was associated with illicit drug use, especially in the high cognitive control group, which at first glance seems to go against dual-process accounts. However, a closer look at items within the BIS suggests that many of these reflect *explicit* motivations and values. In particular, many items might be said to reflect the extent to which an individual values stability and future security over, say, experiencing new and different things (e.g., I plan for job security; I save regularly; I plan trips well ahead of time; I change jobs; I change hobbies; I am future oriented; I am more interested in the present than the future). To the extent that individuals differ in the extent that they value trying new things over stability, they would score differently on such items and likewise, be differently motivated to try illicit drugs. To the extent that self-reported impulsivity is tapping into differences in explicit values and motivations, then the current findings would be in line with other research showing that such factors have more influence in predicting behaviour (eating-related) among individuals with higher

levels of cognitive control than those with low cognitive control (Hofmann et al., 2008).

More generally, the finding that impulsivity was related to illicit drug use supports the conceptualisation of impulsivity as a construct existing on a continuum, associated with AOD use even in university samples (for a similar argument, see Kollins, 2003). This finding is thus consistent with the idea that examining impulsivity (and related constructs such as VMAC) in non-clinical samples can provide important insights into the mechanisms associated with AOD use.

That said, we should emphasise that the current sample was non-clinical. These findings pave the way for future research with clinical populations (e.g., individuals seeking/attending treatment for AOD use problems) to establish whether the current findings reflect how VMAC and cognitive control interact to influence AOD use and/or problems among individuals with a history of prolonged AOD use and/or AOD use problems. Further research in a clinical population could also establish whether VMAC and cognitive control relate to treatment outcomes.

Returning to the interpretation of our findings, earlier we raised two possibilities: that exposure to drugs increases reward-based attentional capture; or that greater general sensitivity of attention to reward increases the likelihood of exposure to, or repeated use of, illicit drugs. The cross-sectional design of the current study means that we cannot currently decide between these two interpretations (that exposure to drugs increases reward-based attentional capture; or that greater general sensitivity of attention to reward increases the likelihood of exposure to, or repeated use of, illicit drugs). Longitudinal studies are needed in order to test whether VMAC predicts future illicit drug use among individuals with cognitive control deficits, or whether AOD use precedes and predicts changes in VMAC and whether this effect is moderated by cognitive control.

Another limitation of the current study is that alcohol and drug use data was based on self-report, which is potentially subject to bias and random error (Babor et al., 1990); however, previous studies have found self-reported alcohol and other drug use to be valid (Hesselbrock et al., 1983; Martin et al., 2005). Finally, a weakness of the current study was the use of alcohol use frequency as the only measure of alcohol use, without reference to quantity. Indeed, a more detailed assessment of alcohol use might have revealed an association between VMAC and alcohol use.

The finding of maladaptive attentional capture in the current study and its association with illicit drug use in participants with poor cognitive control highlights the potential of this procedure as a human analogue of maladaptive learned behaviour (i.e., sign-tracking) in the animal literature (Flagel et al., 2009; Robinson and Flagel, 2009). Particularly, this paradigm offers a novel approach to studying and understanding automatically triggered sensitivities (or more specifically, individual differences in incentive salience attribution), which could learn from and progress in parallel with animal research in this area. Importantly, the interaction between cognitive control and VMAC in relation to actual illicit drug use provides strong support for dual-process models of behaviour. Further research is needed to fully understand the learning mechanisms that drive maladaptive attentional capture in the current procedure, as well as how they are influenced by cognitive control manipulations. Such an understanding may improve current knowledge on what drives problematic AOD use as well as offer novel approaches for treatment and targeted prevention efforts. In particular, the current findings suggest that better treatment effects might be achieved through customising strategies depending on an individual's cognitive profile. That is, while cognitive control training might work best for individuals with low cognitive control, strategies targeting explicit substance use motivations and attitudes might work better for individuals with strong cognitive control (as previously noted, e.g., Wiers et al., 2013). Finally, the current findings have implications for prevention and early intervention efforts by making possible the detection of high risk prior to any substance use exposure.

Conflict of interest

No conflict declared.

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Contributors

LA, RWW, and MLP conceived the idea and methodology of this study. LA performed all data analyses. All authors contributed to the writing of the article and have approved the final manuscript.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <http://dx.doi.org/10.1016/j.drugalcdep.2017.01.041>.

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