



Impulsivity and Attentional Bias to Smoking Cues in Non, Light and Heavy Smokers

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ABSTRACT

Impulsivity is a multidimensional construct that has been implicated in the initiation, maintenance and relapse of nicotine addiction. However, the mechanism by which impulsivity operates in the addiction process is not yet fully understood. One possible explanation is that it functions with other characteristic factors of addiction such as response to drug cues. The current study explores trait impulsivity, delayed gratification and disinhibition across non, light and heavy smokers and attempts to ascertain an interaction with attentional bias to drug related stimuli assessed with the visual dot-probe paradigm. Significant differences across the smoking groups were established for the trait impulsivity BIS-11 subscales and sensitivity to delay gratification, suggesting smokers to be more impulsive than non-smokers. The disinhibition go/no-go task suggested light smokers to have the greatest state motor impulsivity. A significant interaction was not demonstrated between smoking status and response to smoking stimuli on the dot-probe task. However, scores for the delayed gratification and trait motor impulsivity were shown to correlate with attentional bias, to suggest these measures of impulsivity may be related to responsiveness to smoking cues. The discussion considers how the significant results are suggestive of an interplay between impulsivity and attentional bias in the addiction process and how such findings could benefit smoking cessation treatment programmes.

KEY WORDS	ATTENTIONAL BIAS	VISUAL DOT- PROBE	NICOTINE ADDICTION	TRAIT IMPULSIVITY	BEHAVIOURAL IMPULSIVITY
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Introduction

Every year, over 100,000 smokers in the UK die from smoking related causes and despite profound public awareness of the damaging health effects, approximately 10 million adults in the UK still smoke. In 2010, the Government spent £83.9m on services to help people stop smoking and a further £61.8m on stop smoking medication. However, surveys show that two-thirds of current smokers would like to stop smoking but have not received sufficient intervention (ASH, 2011). Such statistics highlight the importance of a comprehensive understanding of the addictive process to enable more effective nicotine cessation treatments to be developed. Addiction has been associated with appetitive reward seeking behaviours such as impulsivity (Mitchell, 1999). The multifaceted construct of impulsivity has been operationally defined as the “inability to stop a behaviour that has negative consequences, a preference for immediate over delayed rewards, tendency to engage in risky behaviours, heightened novelty seeking, behaviour without forethought or consideration of outcome, impatience when forced to wait, and difficulty persisting at an activity” (Perry, Carroll, 2008). However, in much addiction literature, impulsivity has been considered as a homogeneous concept and thus the complexity of its involvement in the addiction process is not yet fully understood (Doran et al. 2009).

Trait definitions of impulsivity have been developed through personality research (e.g. Eysenck et al., 1985). Based on these conceptualisations of impulsivity, Patton et al. (1995) developed the Barratt Impulsiveness Scale (BIS-11) a self-report questionnaire, quantifying trait impulsivity according to three component dimensions, motor, attention and non-planning impulsivity. The scale is an established measure in addiction research used to demonstrate an association between high impulsivity and smoking (Spinella, 2002; Mitchell, 1999; Doran et al., 2004; Vuchinich & Simpson 1998). Studies such as these imply that individuals with more impulsive personality types will be more vulnerable to nicotine use. Doran et al. (2004) also found that smokers who scored higher for impulsivity were more likely to relapse after abstinence, indicating trait impulsivity may contribute to nicotine dependence. However, questionnaire measures rely heavily on self-awareness and the ability of an individual to objectively understand their behaviours. Operant laboratory based tools attempt to overcome this problem and assessments of behaviour enable the exploration of a more circumscribed definition of impulsivity than questionnaires (Mitchell, 1999).

Users of addictive substances often demonstrate impulsive behaviour by showing quicker discounting of the value of delayed rewards and a preference for short term rewards (Bickel et al., 1999). This is recognised in drug abuse behaviour, whereby the perceived value of the initial rewarding effect of the drug is of greater value than long term gains of better health and longer life expectancy (Perry & Carroll, 2008). Therefore, laboratory delayed discounting paradigms consist of presenting a participant with a choice of a smaller immediate reward and a larger reward delivered after a delay. The delay at which the smaller, immediate reinforcer and the larger delayed reinforcer are chosen equally can be calculated and this is referred to as the indifference point (Johnson & Bickel, 2002). The hyperbolic decay model (Equation 1) is typically applied to the indifference points to assess the rate of decay of the reward value over time:

$$V_p = \frac{V}{1 + kD}. \quad (1)$$

However the exponential model can also be applied:

$$V_p = V e^{-kD} \quad (2)$$

Where V_p is the present (discounted) value of the reward, V is the objective (undiscounted) value of the reward, D is the delay from the choice until the receipt of the reward and k is a free parameter. In the exponential equation, e is Euler's number.

Greater discounting of delayed reward has been seen in users of varying drugs of abuse compared with non-users, e.g. opioid-dependant individuals (Madden et al. 1999), cocaine users (Coffey et al., 2003), alcohol abusers (Vuchinich & Simpson, 1998). Ohmura et al., (2005) also found that the discounting for both cigarette and monetary rewards was proportional to cigarettes smoked per day, suggesting that impulsivity was correlated with nicotine intake. Bickel et al. (1999) found that current smokers discounted the value of delayed money more than never-smokers and ex-smokers. For current smokers, delayed cigarettes lost subjective value more rapidly than delayed money, suggesting a rapid loss of subjective value for delayed outcomes, particularly for the drug of dependence. It was postulated that never-smokers and ex-smokers showed similar patterns of discounting because the effects of smoking on delayed discounting were reversible. However, it could be suggest that smokers, less impulsive for delay discounting, are more successful in quit attempts than impulsive smokers. Dallery and Raiff (2007) postulated delay discounting was predictive of latency to smoke after abstinence, suggesting more impulsive smokers had the most difficulty abstaining. Research that considers different smoking status groups is crucial in exploring the role of impulsivity through different stages of the addiction cycle. Koob and Le Moal (2001) suggest that development of drug use through phases of initiation, escalation, maintenance and reinstatement after abstinence, is evidence of changes in the underlying mechanism that regulates drug taking behaviour.

In addition to maximising benefits of long term outcomes, correctly functioning self-control inhibits responses to reward (Hayes et al., 1996) and therefore, behavioural inhibition tasks measure an individual's ability to stop a prepotent response. The go/no-go task (Fox et al., 2002) requires subjects to either execute a response upon the presentation of the "go" target stimulus or inhibit a response upon the presentation of "no-go" target stimulus. These targets are presented in a random order and in quick succession. Addiction research has shown poor inhibitory control in alcoholics (Noel et al., 2007) and cocaine users (Fillmore & Rush, 2002; Hester & Garavan, 2004). Spinella (2002) suggested that the quantity of cigarettes smoked per day was positively correlated to inhibitory response failures. Perkins et al. (2008) found that non-smokers who demonstrated higher behavioural impulsivity were more responsive to the rewarding effects of a nicotine nasal spray, suggesting that disinhibition may also act as a vulnerability to drug use.

Although the existence of a relationship between impulsivity and the initiation, maintenance and relapse of drug abuse has been consistently evidenced, little is known about the underpinnings of this relationship. Evidence from addiction literature has suggested the existence of a

relationship between impulsivity and subjective craving. Doran et al. (2007) found high impulsive smokers showed greater increases in subjective craving after being presented with smoking paraphernalia than low impulsive smokers. It was suggested that impulsive smokers both anticipate and perceive greater reinforcement from smoking and therefore, they may experience stronger craving in response to cues. Reactivity to substance-related cues is commonly associated with regular substance use and dependence (Carter & Tiffany, 1999). Robinson and Berridge (1993) postulated that the rewarding effects of drugs of abuse become associated with environmental cues that are present at the time of drug administration. Field and Duka (2002) provided evidence for the role of conditioning, demonstrating that arbitrary cues are able to grab attention after relatively few pairings with drug administration. Response to cues was previously explained by this classical conditioning of positive effects of reward (Sigel & Ramos, 2002) or negative effects of withdrawal (Stewart et al., 1984). However, Carter and Tiffany (1999) meta-analysis of cue reactivity research, did not find evidence to support either explanation.

Robinson and Berridge (1993) suggested that the rewarding effects of drugs are not just simply conditioned, but have the ability to alter brain chemistry of systems that are involved in the process of natural reward and motivation. Neuroadaptations render these brain reward systems hypersensitive to drugs and drug associated stimuli. However, the affected brain systems do not mediate the pleasurable effects of the drug i.e. liking of a drug, instead a subcomponent of reward, termed incentive salience, sensitises the motivational "wanting" of a drug. Klinger (1975) postulates that having goals effects attention by making individuals receptive to goal associated cues. Therefore, substance-related cues "grab attention, become attractive and wanted and thus guides behaviour to the incentive" (Robinson & Berridge, 1993, p 261). Incentive-sensitization theory suggests that subjective craving and attentional bias are emotional and cognitive outputs of the sensitised dopaminergic system and both motivate substance-seeking behaviour (Robinson & Berridge, 1993). The most recent conceptualisation of the role of attentional bias in addiction suggests that it has a mutually excitatory relationship with subjective craving. When substance-related cues become the focus of attention, subjective craving increases; this in turn increases the "attention-grabbing" properties of the cues and so on until ultimately the substance is sought out and self-administered (Franken, 2003). It is suggested that responses to smoking cues maintain nicotine use and deter attempts to quit (Waters & Feyerabend, 2000) by increasing craving and decreasing the time to the next cigarette (Droungas et al. 1995).

Early assessments of attentional bias used the modified Stroop tasks, the addiction Stroop (e.g. Cox et al., 2006) infers attentional bias when performance on colour is impaired due to the pairing with substance related words. It is suggested that in users of the drug in question, attention is diverted to the automatic processing of the semantic content of the drug-related word, which impairs colour naming. Munafò et al. (2003) found smokers but not non-smokers, were slower to colour name smoking-related words than neutral words. However, this approach has been criticised for its ambiguity in justifying attentional bias as other explanations can also be attributed to the slower reaction times. Klein (2007) suggested that alcohol abusers instructed to suppress thoughts about alcohol, showed slower colour naming than those who were not, therefore attempts to avoid elaborative processing could account for the difference. Algom et al. (2004) suggest that induced craving upon seeing substance related words may generally slow down cognitive performance, as craving has been suggested to tax cognitive function (Tiffany, 1990).

Recent research has focussed on more direct measures of the allocation of visuo-spatial attention with the use of the visual dot probe (Field & Cox, 2006). A substance related stimulus

and a matched control are presented simultaneously on a computer screen and then removed to reveal a visual probe in the location of either stimulus. Reaction times to the probes replacing the drug-related stimulus are compared with reaction times to probes replacing the neutral stimulus. The central tenet of the task is that participants respond faster to probes that appear in the region of a visual display to which they are attending (Posner et al., 1980). Therefore, attentional bias to substance related cues is inferred when participants respond faster to probes that replace drug related cues than neutral or control stimuli. Attentional bias effects have been demonstrated using the visual dot-probe paradigm in users of opiates (Lubman et al., 2000) and alcohol (Townshend & Duka, 2001). Bradley et al. (2003) found a significant difference in attentional bias of smokers compared with non-smokers using a dot-probe of smoking related stimuli.

Reaction time to a visual probe only provides information about the allocation of attention at the time of stimulus offset (Bradley et al. 2003). Luck et al. (2000) postulate the attention system is not unitary and different cognitive mechanisms underpin an initial shifting of attention to a stimulus and the disengagement of attention from a stimulus. These two processes can be assessed by altering the duration of presentation of the stimuli, known as the stimulus onset asynchrony (SOA). Perception research suggests, approximately 50ms is required to shift attention to a presented cue and at least 150ms is required to disengage and redirect attention to another simple cue (Theeuwes, 2005). Therefore, where two stimuli are presented simultaneously in the visual dot-probe task for less than 200ms, any attentional bias observed would be explained as initial orientation of attention. Accordingly, SOAs longer than 200ms will allow time for multiple shifts in attention between different stimuli and therefore attentional bias implies maintenance of attention to a stimulus. This method employed by Bradley et al. (2003) found that smokers showed an attentional bias for smoking images presented for long SOAs but not short SOAs. This suggests smoking cues did not automatically orientate attention, but once they were attended to, they held the attention of smokers. However, Bradley et al. used a short SOA of 500ms and so the lack of effects demonstrated could be because this does not fit with the revised knowledge of timings for attentional shifts.

Townshend and Duka (2001) and Field et al. (2004) considered the relationship between substance-related attentional bias and the quantity and frequency of substance use. Both studies found that heavy drinkers had a significantly greater attentional bias for alcohol related pictures than light drinkers. However, in the case of nicotine research the results have been less conclusive, Mogg and Bradley (2002) found a positive association between nicotine intake and response to smoking stimuli, Bradley et al. (2003) found a negative association and Munfo et al. (2003) reported no significant findings of a relationship between extent of smoking behaviour and attentional bias.

Past research has shown that both impulsivity and cue reactivity are fundamental factors in the process of addiction, but the relationship between the two is yet to be fully understood. Neurobiological evidence suggests that the concepts are related by dysfunctional dopamine mechanisms. High impulsivity has been connected to the disruption of dopamine receptor functioning in the prefrontal cortex, whilst attentional bias has been attributed to the interference of natural reward dopamine pathways in the limbic region (Volkow et al., 2009). However, it is yet to be realised how the manifestations of these dysfunctional systems interact to propagate drug abuse.

A comprehensive understanding of the dissociable facets of impulsivity could enable the development of clinical interventions informed by individual differences, providing more adept treatments for low or high impulsive individuals. Recent research has also already begun to

develop the attentional bias paradigm into a treatment design, attempting to explore the possibility of reducing craving through attentional bias training (Attwood et al., 2008). Results have been mixed as to the efficacy of this as a treatment (Field et al. 2009) and therefore more in depth research into the variations of attentional bias previously mentioned, is required to develop the most effective methods. Combining enhanced knowledge of these approaches to addiction, could lead to the development of more successful smoking cessation treatments.

Drawing on previous research, the current study explored separate dimensions of impulsivity in an attempt to isolate their interaction with nicotine addiction. Therefore, trait measures of impulsivity along with measures of delay discounting and disinhibition were considered. Evidence suggests that cue reactivity is also a factor of drug abuse and the current study attempted to develop this concept in terms of nicotine research. Evaluation of past research suggested that attentional bias measures, using the visual dot-probe, are viable exploratory tools in assessing response to cues, therefore the current study will implement this paradigm. Expanding on past research in this field, the current study will also attempt to further define the attentional processes involved in cue reactivity by exploring responses to smoking and neutral stimuli presented for short and long SOAs. As there is evidence to suggest impulsivity and attentional bias are interactive in a system of addiction, the current study will explore the relationship between the two constructs. Due to the lack of research to explain the involvement of these two mechanisms at different levels of smoking behaviours, the current study will attempt to explore how impulsivity and attentional bias function separately and interact together, in non, light and heavy smokers.

Method

Design

A between groups design was carried out exploring the differences in the dependant variable of impulsivity, between three levels of the independent variable of smoking status, (i.e. non-smokers, light smokers and heavy smokers). The measures of impulsivity were the three subscales scores and total scores of the BIS-11 questionnaire, delay discounting k values, percentage correct and incorrect responses for the go/no-go task along with correct and incorrect response latencies. It was hypothesised that both light smokers and heavy smokers would show higher impulsivity than the non-smokers for each of the measures of impulsivity. A visual dot-probe task was used to carry out mixed between-within groups design, considering the effect of smoking status of reaction time to smoking and neutral stimuli. There were two 3x2 designs, one for 200ms SOA condition and another for 2000ms SOA condition. The between subjects independent variable was smoking status, with three levels of non, light and heavy smoker. The within subjects independent variable was stimulus type, i.e. neutral or smoking picture. It was hypothesised that both heavy and light smokers would demonstrate quicker reaction times to smoking images than non-smokers, in both conditions. It was also hypothesised that a smoking status and stimulus-type interaction would show a difference in reaction time between the neutral and smoking stimuli for the light smokers and heavy smokers, but not the non-smokers. It was hypothesised this interaction would occur in the 200ms and 2000ms SOA condition.

A correlational design was then implemented to explore the relationship between difference scores of reaction time to neutral and smoking stimulus for the 200ms and 2000ms SOAs and all the previously detailed measures of impulsivity. It was hypothesised that the difference

scores would show positive correlations with each measure of impulsivity, suggesting that as impulsivity increases so does attentional bias to smoking cues.

Participants

Thirty three participants, 11 non-smokers, ($M = 26.82$, $SD = 12.77$) 11 light-smokers, ($M = 21.64$, $SD = 3.04$) and 11 heavy-smokers ($M = 23.36$, $SD = 2.29$) were recruited from Leeds Metropolitan University, via poster advertisement or by opportune sampling of people on campus. Non-smokers were “never” smokers and were recruited as the control group. Participants were recruited as light smokers if they smoked <10 cigarettes per day, heavy smokers were recruited as those who smoked >10 cigarettes per day (Shavelle et al., 2008). Pre-testing questioning excluded social smokers, i.e. those who did not smoke at least one cigarette a day and any smokers attempting to quit, in case the testing process encouraged relapse. Difference in smoking dependence between the two groups was confirmed using scores from the Fagerstrom Test of Nicotine Dependence Questionnaire (FTND; Heatherton et al., 1991) and analysed with a t-test. Heavy smokers demonstrated a higher dependence to nicotine ($M = 6.36$, $SD = 1.43$) than light smokers ($M = 1.18$, $S.D. = 1.40$) and this difference was significantly established ($t(20) = -8.57$, $p < 0.05$). This difference was also confirmed by carbon monoxide (CO) ppm readings, which suggested the heavy smokers, ($M = 18.27$, $SD = 7.73$) smoked significantly more than light smokers ($M = 4.54$, $SD = 1.81$, $t(20) = -5.73$, $p < 0.01$).

Materials

The Fagerstrom Test for Nicotine Dependence was implemented as a self-report measure of nicotine dependence comprised of six multiple choice questions assessing smoking behaviour by latency to first cigarette of the day, number of cigarettes per day, ease of refraining from smoking, time of day when craving is strongest and when smoking is most frequent and the need to smoke even when ill (see appendix 1). Pomerleau et al. (1994) suggested the FTND demonstrated moderate internal consistency with a Cronbach alpha coefficient reported at 0.68. The questionnaire showed similar internal consistency in the current study with a Cronbach alpha of 0.69.

The Barratt Impulsiveness Scale (BIS-11; Patton et al., see appendix 2) was used to assess trait impulsivity. Each of the 30 component self-report items pertained to one of three components of impulsivity. The eight items of the attention impulsiveness subscale assessed attention and cognitive impulsivity (e.g. I get bored easily when solving thought problems). The “motor” subscale, measuring motor impulsiveness and perseverance, consisted of eleven items (e.g. I am restless at the theatre or lectures) and the remaining eleven items assessed self-control and cognitive complexity in the “nonplanning” subscale (e.g. I spend or charge more than I earn). Participants made their responses on a four level low to high Likert Scale (i.e. 1 = 2 = “occasionally”, 3 = “often” and 4 = “almost always/always”) 11 of the items were reverse scored. An initial explanation section instructed the participants not to spend too much time on the questionnaire and answer “quickly and honestly”. An average score was recorded for each of the subscales and an overall score for each participant was also calculated. According to Doran et al. (2007) the BIS-11 has demonstrated good internal consistency, Cronbach's $\alpha = 0.82$. Good internal consistency was also established in the present study, Cronbach's $\alpha = 0.89$.

The delay discounting task design was taken from Johnson and Bickel (2002). The task was performed on a computer running Microsoft Visual Basic 6.0 and began with an instruction

screen informing the participants that they were going to be asked to choose between hypothetical rewards (see appendix 3) which were presented to the participant using the choice algorithm. The screen displayed two adjacent large command buttons in which the choices were presented. The left button always displayed an immediate reward (e.g. £500 now) and the right button displayed a delayed reward (e.g. £1000 in a week). The selection of the choice was made by operating the cursor. Centrally aligned above the two choice buttons was a circle that was green when the command buttons were available to register a response and then turned red after a response was made. The circle remained red for 2 seconds during which time the two choice buttons were visible, but were disabled and the participant was forced to wait before responding. After 2 seconds, the circle turned green again. There was no programmed limit to the time the participant could wait before making a response. The choice of the hypothetical delayed reward remained fixed at £1000 for each trial and was compared with a hypothetical short term reward which altered in value until an indifference point was established. The short term reward was offered immediately and the delay time of the reward altered in each of the 7 trials (i.e. 1 day, 1 week, 1 month, 6 months, 1 year, 5 years and 25 years).

Taking from the method adopted by Fox et al. a go/no-go task was setup in E-Prime consisting of ten blocks of 18 symbols appearing rapidly on the screen. Half of the symbols were “targets” and half were “non-targets” and comprised of either letters (A-G) or numbers (2-9). Participants were informed of the target stimulus for each block and told to respond as quickly as possible using the space bar (see appendix 4). The target alternated between letters and numbers following every two blocks. The initial two blocks were practise trials where the target was counterbalanced across participants. The order of the blocks (i.e. either letter target or number target) was counterbalanced across the participants to control for order effects. The stimuli were displayed on a black background in white type, font courier new, size 30. Each stimulus was displayed for 300ms followed by an interval of 900ms. A correct “go” response was made if the space bar was tapped during the presentation of the target stimulus or during the interval that followed. An incorrect “go” was recorded if the participant failed to respond. A correct “no-go” was recorded if the participant withheld their response at the presentation of a non-target stimulus. An incorrect “no-go” was recorded when a response was made when a non-target stimulus was presented or in the interval that followed.

The visual dot-probe task was run in E-Prime, on a separate PC to that on which the impulsivity tasks were run. The E-Prime was the only application running on the PC to ensure the accuracy of the reaction time recording. At the beginning of the task, a screen of instructions was presented to the participant, informing them they would see a series of picture pairs on the screen, when the picture pairs were removed they would be presented with the probe on either side of the screen and they were to press “UP” or “DOWN” on the keyboard, corresponding to the direction in which the arrow was pointing. Participants were instructed to respond as quickly as possible and to have their dominant hand placed over the appropriate keys ready to make a response (appendix 5).

Visual stimuli in the visual dot-probe consisted of 20 colour photographs of smoking-related images (e.g. a woman putting a cigarette to her lips, a hand holding a cigarette beside an ashtray etc.). Each was paired with another photograph of another scene, matched as closely as possible for image complexity, colour saturation, brightness and content, but lacked any smoking related cues (e.g. a woman applying lipstick, a hand holding a pen by a writing pad). An additional 20 picture pairs of furniture, chosen for their lack of emotive content, were prepared for use as fillers. The pictures were digitalised and converted to an indexed 256-colour palette and they were adjusted in size so that each was approximately 125mm wide and 100mm

high when displayed on the screen. When presented side by side the distance between them was 60mm. The background display was black. The probes used were white arrows, pointing either up or down, presented on a black background and were 30mm in height when displayed on the screen. The probe was presented in the centre of the position previously occupied by either pictorial stimulus, thus the probe locations were 150mm apart. Each of the picture pairs were shown four times to allow every combination of picture type and probe congruence to be presented. This combination of picture and probe was displayed once each for 200ms SOA and 2000ms SOA. The filler pictures were presented four times in total for the 200ms and 2000ms condition, with the four probe and picture combinations shown for each pair. In total there were 160 critical trials and 80 filler trials, randomly presented. Four of the filler pairs were used for a 16 trial practice block.

Each trial began with a fixation mark presented in the centre of the screen for 500ms. A delay of 250ms was interjected before presentation of a picture pair for either 200ms or 2000ms. The pictures were replaced immediately with a black screen with the arrow presented on either side. Once the participant had made their response to the arrow it was removed from the screen and a black screen was presented for 1000ms as an inter-trial interval. When half of the trials had been presented the task programmed a one minute break for the participant, after which they were able to restart the task by selecting any key on the keyboard.

Procedure

Ethical approval was granted by the Leeds Metropolitan Ethics Board. Each participant was presented with an information sheet prior to testing outlining the experimental method and explaining the anonymity of the results and details of how their results could be withdrawn. Informed consent was given by each participant. Smokers were given the FTND questionnaire and their carbon monoxide reading was taken. Each participant then completed the BIS-11 questionnaire. Participants were then seated at the PC to complete the delay discounting and the go/no-go tasks. The task order was counterbalanced for each participant to counteract order effects. Participants were instructed to inform the experimenter when each task had finished. Participants were then asked to move onto the second computer where they completed the dot-probe task. Upon completion of the task each participant was given a debrief providing an explanation of the aims of the experiment and reinstating the participant's right to withdraw their results. Participants completed the task individually in a quiet computer lab at Leeds Metropolitan University. One researcher was present for the duration, although care was taken to ensure the participant did not feel under observation. The entire procedure took approximately 1 hour.

Data Analysis

Mean scores were calculated for total scores of the BIS-11, with the necessary items reverse scored. Subscale scores for attention, motor and nonplanning impulsivity were also calculated into mean scores for each participant. A one-way ANOVA was performed on total scores and each of the subscales to see if a significant difference occurred between the smoking status groups (i.e. non, heavy, light smokers).

Participant's choices in the delay discounting task were calculated into indifference points for each of the seven trials. Each participant's indifference points were plotted on separate graphs and an exponential model was applied and a k value was devised. An r^2 value was also produced to denote the goodness-of-fit of the model to the data. For all 33 participants mean $r^2 = 0.78$ (s.d.=0.12) suggesting the model demonstrated a good fit to the data sets. The k values

from all the participants were analysed with smoking status in a one-way ANOVA to look for difference across the smoking groups.

Regarding the go/no-go task, the number of incorrect no-go responses were calculated and expressed as a percentage of total responses for each participant, providing the main index of impulsivity. Percentage incorrect go responses, means of correct response latency and incorrect response latency were also recorded for each participant. Each of these four variables were computed in a one-way ANOVA against the independent variable of smoking status to establish difference.

Mean reaction times calculated for each probe position and SOA (i.e. probe congruence with the neutral image 200ms SOA, probe congruence with neutral image 2000ms SOA, probe congruence with the smoking image SOA 200ms and probe congruence with the smoking image 2000ms SOA). Trials with errors or reaction times <200ms or >2000ms were removed, (Bradley et al., 2003) in <3% of data was removed. A 3x2 ANOVA was used to analyse response time to the neutral and smoking congruent probes, in relation to the IV of smoking status and IV of stimulus type, for the 200ms SOA and the 2000ms SOA condition.

Difference scores were also calculated between reaction times to the neutral and smoking stimulus congruent probes presented for the same duration. Difference scores at 200ms and 2000ms SOAs were then correlated with all impulsivity variables using a Spearman's rho.

Results

Tests of normality and homogeneity of variance were carried out on each of the variables. Normality was assumed if the significance of the Shapiro-Wilk value was >0.05 or the kurtosis statistic was >+/-0.2 (Miles & Shelvin, 2001). Significantly non-normal data was transformed with a Log10 transformation. Percentage data from the go/no-go variables was transformed using an arc-sine transformation. The Levene's test indicated that the homogeneity of variance was not violated for any of the data sets, thus ANOVA analysis could be applied to the data.

Descriptive statistics are displayed in Table 1 for the subscale scores and total scores of the BIS-11.

Table 1
Means standard deviations for scores on the BIS-11 and subscales of the smoking status groups

	BIS-11 Total		Attention		Motor		Non-Planning	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Non-Smoker	1.97	0.28	1.85	0.44	2.08	0.27	1.94	0.37
Light Smoker	2.45	0.26	2.32	0.40	2.69	0.30	2.31	0.30
Heavy Smoker	2.67	0.43	2.82	0.47	2.41	0.36	2.81	0.71

The mean scores indicate that light smokers and heavy smokers scored higher than non-smokers for total scores and all three subscales. Heavy smokers scored higher than light smokers for all measures with the exception of motor impulsivity for which light-smokers appear the most impulsive.

A one way ANOVA was performed on the total scores for the BIS-11 and the subscales. A significant difference was found between smoking groups for BIS-11 total scores ($F(2, 30) = 12.83, p < 0.01$) with a large effect size (eta squared = 0.46). Post-hoc comparisons of the BIS-11 total scores using the Tukey HSD suggested that light smokers scored higher for impulsivity than non-smokers (mean difference = 0.48, $p < 0.05$) as did heavy smokers (mean difference = 0.70, $p < 0.01$). However there was no significant difference between the impulsivity scores between light and heavy smokers (mean difference = 0.22, $p > 0.05$). This suggests that the smokers were significantly more impulsive than non-smokers, however there was no distinction between the smoking groups for this measure of impulsivity.

A significant difference was found for the BIS-11 Attention subscale ($F(2, 30) = 13.48, p < 0.01$) with a large effect size (eta squared = 0.47). Post-hoc comparisons using Tukey HSD tests indicated that non-smokers scores for attentional impulsivity were significantly lower than light smokers (mean difference = 0.47, $p < 0.05$) and heavy smokers (mean difference = -0.97, $p < 0.05$). Light smokers also scored significantly lower than heavy smokers (mean difference = 0.5, $p < 0.05$). This suggests the smokers showed greater attention impulsivity than non smokers and that heavy smokers were the most attentionally impulsive group.

There was also a statistically significant difference in the BIS-11 Motor subscale scores between the smoking -status levels ($F(2, 30) = 10.19, p < 0.01$) with a large effect size (eta squared = 0.43). Post-hoc comparisons using Tukey HSD for motor impulsivity showed that heavy smokers had scores significantly higher than non-smokers (mean difference = 0.06, $p < 0.01$), as did the light smokers (mean difference = 0.11, $p < 0.01$). This indicates that both heavy and light smokers show more motor impulsivity than non-smokers, however, no significant difference of impulsivity between light and heavy smokers was established in this measure (mean difference = 0.05, $p < 0.05$).

The BIS-11 Non-planning subscale also showed a significant difference across the smoking status groups ($F(2, 30) = 8.76, p < 0.01$) with a large effect size (eta squared = 0.37). Post-hoc comparisons using Tukey HSD for the non-planning subscale indicated the heavy smokers had significantly higher scores than non-smokers (mean difference = 0.88, $p < 0.01$). No significant differences were established between light and heavy smokers (mean difference = 0.51, $p > 0.05$) or light and non-smokers (mean difference = 0.37, $p > 0.05$). This suggests that heavy smokers exhibited higher non-planning impulsivity than non-smokers and light smokers.

Mean k values and standard deviations are displayed in Table 2 for each smoking status condition.

Table 2
Means and standard deviations for k values across the smoking status groups

k Value

	<i>M</i>	<i>SD</i>
Non-Smoker	0.20	0.13
Light Smoker	0.55	0.20
Heavy Smoker	0.53	0.19

The means indicate that light smokers and heavy smokers had higher *k* values than non-smokers suggesting faster discounting of delayed reward and hence higher impulsivity. The means suggest the light smokers are marginally more impulsive than the heavy smokers.

A one-way ANOVA showed a significant difference across the three smoking conditions ($F(2, 30) = 13.18, p < 0.01$) with a large effect size (eta Squared = 0.47). Tukey HSD was implemented as post-hoc analysis and it was found that results from both light-smokers (mean difference = 0.35, $p < 0.01$) and heavy smokers (mean difference = 0.32, $p < 0.01$) discounted reward to a greater extent than non-smokers. A significant difference was not established between light and heavy smokers (mean difference = 0.03, $p > 0.05$).

Means and standard deviations of percentage correct go and percentage correct no-go responses are shown below in Table 3.

Table 3
Means and standard deviations of percentage of correct go and no-go responses of the smoking status groups

	% Correct No-Go		% Correct Go	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Non-Smoker	92.30	7.01	97.85	2.51
Light Smoker	85.61	7.43	93.56	4.78
Heavy Smoker	88.64	5.44	94.95	3.00

The means suggests that light smokers and heavy smokers made fewer correct no-go and correct go responses than the non-smokers, with the light smokers making the most errors.

A one-way ANOVA revealed a significant difference was established between smoking groups for percentage correct no-go responses ($F(2, 30) = 3.35, p < 0.05$, eta squared = 0.19). Post hoc analysis using Tukey HSD revealed a significant difference between the means of non-smokers and light smokers (mean difference = 6.69, $p < 0.05$) indicating that light smokers made significantly fewer correct no-go responses than non-smokers. However, there was no significant difference established between non-smokers and heavy smokers (mean difference = 3.66, $p > 0.05$) or between the heavy and light smokers (mean difference = 3.03, $p > 0.05$).

The results of a one-way ANOVA of correct go responses revealed a significant difference between groups ($F(2, 30) = 6.25, p < 0.05$, eta squared = 0.29). Post-hoc analysis using Tukey HSD revealed that percentage correct go responses were significantly lower in light smokers than non-smokers (mean difference = 4.29, $p < 0.05$) and significantly lower for the heavy smokers compared with the non-smokers (mean difference = 2.90, $p < 0.05$). This suggests both light smokers and heavy smokers demonstrated less accuracy in their performance than the

non-smokers. A difference between light and heavy smokers was not significantly established for percentage correct go scores (mean difference = 1.39, $p > 0.05$).

Descriptive statistics are presented below in Table 4, for correct and incorrect response latencies in the go/no-go task.

Table 4
Mean and standard deviations for response latencies of the smoking status groups

	Correct Response Latency		Incorrect Response Latency	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Non-Smoker	404.83	31.74	323.85	58.00
Light Smoker	382.33	24.26	337.99	58.20
Heavy Smoker	373.51	23.40	330.72	18.41

The means suggests that the non-smokers showed greater response latency for correct responses, compared with the light and heavy smokers, indicating a slower rate of responding. Heavy smokers appear to have made the fastest correct responses. Light and heavy smokers exhibit greater incorrect response latency than non-smokers.

The ANOVA of correct response latency also showed a significant difference between groups ($F(2,30) = 4.01$, $p < 0.05$) with a large effect size ($\eta^2 = 0.21$). Post-hoc analysis with Tukey HSD revealed percentage correct response latency was significantly lower for heavy smokers compared with the non-smokers (mean difference = 31.31, $p < 0.05$). This suggests that heavy smokers had made significantly faster responses than the non-smokers on the go/no-go task compared with non-smokers. However, a significant difference was not established between light and heavy smokers (mean difference = 8.82, $p > 0.05$) and light and non-smokers (mean difference = 22.49, $p > 0.05$).

There was no significant difference of incorrect response latency between light, heavy and non-smokers when a one-way ANOVA was implemented ($F(2,30) = 0.23$, $p > 0.05$).

Mean reaction times for the visual dot-probe stimuli presented at 200ms SOA are presented below in Table 5.

Table 5
Means and standard deviations of reaction times to dot-probe at 200ms SOA for smoking status groups

	Response Time to Neutral Stimuli		Response Time to Smoking Stimuli	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Non-Smoker	604.17	95.97	592.43	79.69
Light Smoker	577.91	45.40	561.80	49.06
Heavy Smoker	567.98	56.47	543.72	40.44

The means indicates that light and heavy smokers made quicker responses to the smoking stimuli presented for 200ms than the non-smokers. Non-smokers exhibited the slowest reaction time in each stimulus condition and demonstrated the least variation in response time according to the stimuli shown.

Reaction time scores from the visual dot-probe were compared in two separate two-way ANOVAs, for neutral and smoking stimuli probe congruence at 200ms exposure and neutral and smoking stimuli probe congruence at 2000ms exposure.

A two-way ANOVA was performed to ascertain differences in reaction time in the 200ms SOA condition. A main effect was established for stimulus (neutral or smoking picture congruence of the probe) ($F(1, 30) = 5.63, p < 0.05$, eta square = 0.16) indicating a significant difference for reaction times to the smoking or neutral image. Post hoc pairwise comparisons revealed that the participants' reaction times were significantly quicker in response to the smoking congruent image compared with the neutral congruent image (mean difference = 17.37, $SD = 7.32, p < 0.05$). There was no main effect of smoking status ($F(2, 30) = 1.39, p > 0.05$). An interaction between picture and smoking status was not significantly established ($F(2, 30) = 0.25, p > 0.05$).

Mean reaction times for the visual dot-probe stimuli presented at 2000ms SOA are presented below in Table 6.

Table 6
Means and standard deviations of reaction times to dot-probe at 2000ms SOA for smoking status groups

	Response Time to Neutral Stimuli		Response Time to Smoking Stimuli	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Non-Smoker	617.57	76.77	616.01	71.80
Light Smoker	600.97	58.27	571.38	42.93
Heavy Smoker	573.66	67.03	546.15	54.15

The means suggest that again light and heavy smokers made quicker responses to the smoking stimuli presented for 2000ms than the non-smokers. Non-smokers exhibited the slowest reaction time in each stimulus condition and demonstrated the least variation in response time according to the stimuli shown. Light smokers showed the greatest difference in response time to the neutral and smoking stimuli.

In the 2000ms exposure there was a main effect for picture type ($F(1, 30) = 5.48, p < 0.05$ eta squared = 0.15), post hoc pairwise comparisons revealed that participants responded significantly faster to the smoking stimuli than the neutral stimuli (mean difference=19.56, $SD = 8.36, p < 0.05$). A main effect of smoking status was not significantly established ($F(2, 30) = 2.64, p > 0.05$). A significant interaction was not established between picture type and smoking status ($F(2, 30) = 1.17, p > 0.05$).

Difference scores were calculated for reaction times between the two stimuli (smoking and neutral) for both exposure times (200ms and 2000ms). Shapiro Wilk analysis revealed that the difference scores calculated from the dot-probe reaction times were not normally distributed and a Spearman's rho was used to correlate difference scores with the variables of impulsivity.

Spearman's correlation co-efficients between the BIS-11 total and subscale scores and difference scores at 200ms and 2000ms SOA are shown in Table 7.

Table 7
Correlation matrix of visual dot-probe difference scores and BIS-11 total and subscale scores

	Difference Scores 200ms SOA	Difference Scores 2000ms SOA	Attention	Motor	Nonplanning	BIS-11 Total
Difference Scores 200ms SOA						
Difference Scores 2000ms SOA	0.23					
Attention	0.13	0.13				
Motor	0.02	0.33*	0.49**			
Nonplanning	0.20	0.27	0.62**	0.47**		
BIS-11 Total	0.15	0.25	0.86**	0.68**	0.88**	

* Correlation is significant at the 0.05 level

** Correlation is significant at the 0.01 level

Difference scores for the 2000ms picture exposure showed a medium positive correlation with BIS-11 subscale for motor impulsivity ($r=0.33$, $n=33$, $p< 0.05$) suggesting the higher the rating for motor impulsivity the greater the difference between the reaction time scores to smoking and neutral images, i.e. attentional bias for smoking cues increases with motor impulsivity.

The Spearman's rho correlation coefficients are between difference scores and delay discounting k values are shown in Figure 8.

Figure 8
Correlation matrix of *k* values and visual dot-probe difference scores

	Difference Scores 200ms SOA	Difference Scores 2000ms SOA	<i>k</i> value
Difference Scores 200ms SOA			
Difference Scores 2000ms SOA	0.23		
<i>k</i> value	0.09	0.36*	

* Correlation is significant at the 0.05 level

** Correlation is significant at the 0.01 level

A significant, medium positive correlation was also established between *k* value on the delay discounting task and difference scores for the 2000ms exposure time ($r=0.36$, $n=33$, $p<0.05$). This suggests the quicker the reward discounting, (i.e. the higher impulsivity), the greater the difference in reaction time scores between the smoking and neutral images, indicative of attentional bias.

Spearman's rho correlation co-efficient between the four variables of the go/no-go task and difference scores are shown in Table 9.

Table 9
Correlation matrix of go/no-go variable and visual dot-probe difference scores

	Difference Scores 200ms SOA	Difference Scores 2000ms SOA	% Correct Go	% Correct No-Go	Correct Response Latency	Incorrect Response Latency
Difference Scores 200ms SOA						
Difference Scores 2000ms SOA	0.23					
% Correct Go	-0.14	-0.12				
% Correct No-Go	-0.24	-0.02	0.67**			
Correct Response Latency	-0.11	-0.11	0.58**	0.35*		
Incorrect Response Latency	-0.06	-0.37	0.21	-0.01	0.38*	

* Correlation is significant at the 0.05 level

** Correlation is significant at the 0.01 level

No significant correlations were established between the go/ no-go task variables and the dot-probe difference scores. The only significant correlations observed were between the variables of the go/no-go task.

Discussion

The BIS-11 overall scores suggest light smokers and heavy smokers were more impulsive than non-smokers, supporting the previous findings in addiction literature, that smokers show greater

trait impulsivity than non-smokers (e.g. Mitchell, 1999; Doran et al., 2004). Attentional impulsivity, as scored on the BIS-11 showed significant increases from non, to light, to heavy, smokers. de Wit (2008) suggests that attentional impulsivity or inattention, may increase difficulty in abstaining from drug use, as lapses in perseverance and attention result in lapses to drug use. As attentional impulsivity is observed in both levels of smoking status, it may have influence in the progression of nicotine addiction. It could be suggested that higher attentionally impulsive individuals may be more likely to initiate nicotine use that then escalates into heavy smoking behaviours. However, less attentionally impulsive light smokers are less vulnerable to the escalation of smoking behaviour. Subscale measures for motor impulsivity reinstated the smoker and non-smoker difference, however, no difference was found between light and heavy smokers. This could suggest that motor impulsivity is a dispositional predictor of smoking behaviour, as suggested by Sher et al. (2000) but it does not regulate the strength of addiction or mediate smoking behaviour. However, conflicting evidence from Spinella (2002) suggests that motor impulsivity does increase with dependence, demonstrating a correlation with BIS-11 motor subscale scores and cigarettes smoked per day, indicating that impulsivity increases with dependence.

Non-planning impulsivity was significantly higher amongst the heavy smokers than light or non-smokers. Doran (2007) postulated that as smokers have less consideration for negative future consequences, they are more likely to adopt dangerous smoking habits than those who engage in forward thinking. Vuchinich and Simpson (1998) suggested that light smokers were more future orientated than heavy smokers and concluded that light smokers may curtail their smoking behaviours as they have more forethought for future consequences. This is reflected in the current study whereby light smokers did not exhibit non-planning impulsivity above that of the non-smokers.

As hypothesised, the delay discounting paradigm also indicated a significant difference in impulsive choice between smokers and non-smokers. Both heavy and light smokers discounted delayed reward significantly quicker than non-smokers, substantiating previous research that smokers exhibit more impulsive choice by valuing short term rewards higher than long term gains (e.g. Bickel et al. 1999; Mitchell, 1999). As de Wit and Richards (2004) suggest, this impulsivity manifests in smokers' behaviour in placing greater value on the short term rewards of a drug, over the long term rewards of better health or longer life expectancy. No significant difference was established between the rate of discounting between light and heavy smokers, suggesting that delay gratification is associated with the maintenance of smoking behaviour but is not a factor in the escalation of use. A similar result was established by Vuchinich and Simpson (1998) which found no significant difference in discounting rates between heavy and light drinkers. This would suggest that differences in consumption of drugs such as nicotine are not accounted for by impulsive discounting of delayed reward, although this may have influenced initial use.

However, the lack of a significant effect could also expose flaws in the experimental design of the current study. Johnson and Bickel (2002) suggests the use of either the hyperbolic or exponential models to establish the k value, however, the hyperbolic model is considered more accurate (Vuchinich & Simpson 1998). In the current study the exponential model was applied and this may have reduced the accuracy of the k values and therefore difference was not detected between the groups. Also, smokers were asked to arrive ready for testing in a sated condition, having maintained their normal smoking habits. All heavy smokers had had a cigarette prior to testing which was confirmed by the carbon monoxide readings. However, as testing sessions took place in the morning, many of the light smokers, having maintained their

normal smoking behaviour, had not had a cigarette within the twelve hours prior to testing, which was also suggested by the lower carbon monoxide readings. It could therefore be suggested that the light smokers would have been in a mild state of withdrawal. Field et al. (2006) and Mitchell (2004) suggest that impulsivity in a delayed discounting task increases during nicotine withdrawal, thus the light smokers may have been performing more impulsively, which in turn increased their scores to a level comparable with heavy smokers in a sated state. Another consideration is that the mean age of the heavy smoker group was higher than that of the light smoker group. Reynolds (2006) suggested that impulsivity in delay gratification scenarios decreases with age as impulsive choice becomes less pronounced. Therefore, although a heavy smokers' k value might be elevated as a function of smoking behaviour, the effect of age would simultaneously reduce it, with the opposite effects being demonstrated for younger light smokers.

Difference was also found across smoking status groups for percentage correct no-go responses in the go/no-go task. The mean scores supported the hypothesis that light and heavy smokers showed a higher error rate than non-smokers, with the light smokers displaying significantly lower percentage of correct no-go response than non-smokers, demonstrating poorer inhibitory control. According to Hayes et al. (1996) behavioural disinhibition results in the inability to inhibit responses to reward and therefore would increase drug taking behaviour. Spinella (2002) and Billieux et al. (2006) suggest that smokers display poorer inhibitory control than non-smokers providing evidence to support the theory that increases in disinhibition correlate with quantity of nicotine intake. In the current study however light smokers demonstrated poorer inhibitory control than heavy smokers. Harrison et al. (2009) postulated that deprivation also increases errors in inhibitory control, therefore, if the light smokers were experiencing mild withdrawal this may have increased their impulsivity to a level above that of heavy smokers. Heavy smokers demonstrated a significantly faster response time with lower correct response latency than non-smokers, suggestive of increased motor impulsivity (Fox et al. 2002) as was hypothesised. However, the light smokers did not exhibit such an effect. The percentage go errors were also significantly higher for the heavy and light smokers than the non-smokers. A deprivation in the light smokers could have reduced accuracy as Harrison et al. also observed an increased go error rate in deprived smokers and postulated that the less impulsive smokers were more affected by deprivation. Inaccuracy in the performance of heavy smokers could be attributed to a lack of attention. As the previous BIS-11 scores identified heavy smokers to be high for attentional impulsivity, these results may reflect the deficits in attention and perseverance that define this type of impulsivity.

It was hypothesised that both heavy and light smokers would demonstrate attentional bias to smoking cues at 200ms SOA, compared with non-smokers, however, the results did not reveal any such interaction. Bradley et al. (2003) also reported a lack of significant difference between smokers and non-smokers for attentional bias for short SOAs, therefore, it could be suggested that smoking stimuli do not automatically grab the attention of smokers any more than non-smokers. However, overall, participants responded significantly quicker towards the smoking stimuli, as suggested by the significant main effect. The descriptive statistics suggested the greatest differences in reaction time between the stimuli, were shown by the light and heavy smokers. The lack of a significant interaction could be attributed to the small sample size used, therefore, further testing with a larger sample size could increase the power of these effects.

Bradley et al. (2003) instead suggested that smokers showed attentional bias towards smoking cues at longer SOAs and mean reaction time scores for the 2000ms SOA in the current study demonstrated this trend, however statistical significance was not reached. This could again be

due to the small sample size used and increasing the number of participants in the study could improve the significance. Consideration also has to be given to the possibility of varying deprivation levels across the groups having an impact on the results. Field et al. (2006) suggested nicotine deprivation increases attentional bias, therefore, the light smokers with low mean CO ppm readings may have been more responsive to smoking cues than the satiated heavy smokers with higher ppm readings. Repeating the design with both groups in a state of deprivation may show more pronounced effects of attentional bias. However, it could also be considered that this insignificant result may suggest that smokers are more responsive to internal cues such as craving induced by withdrawal. The mean reaction times suggest that the greatest difference in response time between stimuli was demonstrated by the light and not the heavy smokers. Koob and Le Moal (2001) suggested that individuals with a high nicotine dependence have induced a permanent dysregulation of the homeostatic reward systems and nicotine intake will be motivated by the need to regain homeostasis after withdrawal. Therefore, if smoking behaviour is motivated by a regular pattern of reward and withdrawal, external cues may carry less incentive salience for heavy smokers as they will have a nicotine intake regardless of the presence of conditioned cues.

The correlational analysis of difference scores suggested that BIS-11 self report scores for motor impulsivity correlated with attentional bias difference scores at 2000ms. This suggests, the more trait motor impulsivity an individual exhibits, the more susceptible they will be to a drug cue holding their attention. Mitchell (1999) suggested that such impulsive individuals have difficulty controlling their responses to reward, therefore, when presented with a reward cue impulsive individuals will have difficulty disengaging their attention and thus display the attentional bias observed in the current study. As no correlations were found between the non-planning and attention subscales, it could be suggested that these facets of impulsivity are not associated with attentional bias. Doran et al. (2007) found no association between cue-induced craving and "lack of premeditation" and suggested this impulsivity was related to cigarette consumption but not craving in response to cues.

Attentional bias at 2000ms correlated with delay discounting k values, suggests that the faster discounting of delayed reward is related to fixation of attention on smoking cues. It could be suggested readily available cues hold attention as they suggest the possibility of immediate reward. Therefore, less motivation would be given to the disengagement from a cue in the pursuit of a delayed reward. Doran et al. (2007) suggested that smoking cue exposure made it more difficult for more impulsive smokers to resist an instant cigarette reward over an increased amount of cigarettes after a delay. Doran et al. (2007), therefore, suggests that response to cues regulates impulsivity, however, cause and effect cannot be inferred in the correlation design of the current study. Further research in the area could attempt to define this relationship as the next step towards developing clinical nicotine addiction interventions. If impulsivity is considered a predictor of responsiveness to cues, then the control of impulsive behaviour could be addressed in cognitive or behavioural therapies for addiction. If attentional bias for smoking cues is found to increase impulsivity and encourage drug intake, then attentional bias training could be developed to help smokers to recognise and control their responses to smoking cues.

To the author's knowledge, this is the first evidence to support an association between specific components of impulsivity and attentional bias. By investigating impulsivity as dissociable constructs, better understanding of the interplay between impulsivity and addiction could be investigated. Similarly, a more comprehensive approach to types of attentional bias has provided evidence for the effects of different mechanisms of attention in response to stimulus-related cues. The effects seen in this study could also be re-examined in samples of withdrawn

or abstinent smokers in order to develop an understanding of how these processes contribute to relapse. Developments of the concepts addressed in this research could be implemented in development of effective smoking cessation treatments.

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