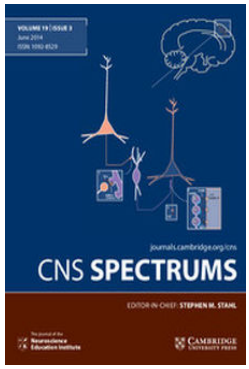

The clinical relevance of attentional bias in substance use disorders

Matt Field, Reshmi Marhe and Ingmar H. A. Franken

CNS Spectrums / Volume 19 / Issue 03 / June 2014, pp 225 - 230
DOI: 10.1017/S1092852913000321, Published online: 13 May 2013

CNS Spectrums (2014), **19**, 225–230.
doi:10.1017/S1092852913000321

REVIEW ARTICLE

The clinical relevance of attentional bias in substance use disorders

Matt Field,[1]* Reshmi Marhe,[2,3] and Ingmar H. A. Franken[2]

[1] *School of Psychology, University of Liverpool, Liverpool, UK*
[2] *Institute of Psychology, Erasmus University Rotterdam, Rotterdam, The Netherlands*
[3] *Department of Child and Adolescent Psychiatry, VU University Medical Centre, Amsterdam, The Netherlands*

Individuals with substance use disorders typically show an "attentional bias" for substance-related cues: Those cues are able to grab and hold the attention, in preference to other cues in the environment. We discuss the theoretical context for this work before reviewing the measurement of attentional bias, and its relationship to motivational state and relapse to substance use after a period of abstinence. Finally, we discuss the implications of this research for the treatment of substance use disorders. We conclude that attentional bias is associated with subjective craving, and that moment-by-moment fluctuations in attentional bias may precede relapse to substance use. The evidence regarding the predictive relationship between attentional bias assessed in treatment contexts and subsequent relapse is inconsistent. Furthermore, there is currently insufficient evidence to endorse attentional bias modification as a treatment for substance use disorders. Clinical implications and suggestions for future research are highlighted.

Received 25 February 2013; Accepted 5 April 2013; First published online 13 May 2013

Key words: Addiction, attentional bias, attentional bias modification, relapse, Stroop task, visual probe task.

Clinical Implications

- Attentional bias should be discussed with substance abusing patients as a component of psychosocial interventions. They should be advised that it may function as an "early warning signal" for imminent relapse, so that they can act accordingly (for example, distancing themselves from a high-risk situation) if they become aware of it.
- Based on the ambiguous evidence to date, we do not recommend assessing attentional bias in treatment settings for clinical purposes (for example, in order to identify those who are at risk of treatment dropout or relapse).
- Based on the available evidence, we do not recommend incorporating attentional bias modification (ABM) into treatment programs, until the intervention has been evaluated in a large-scale clinical trial.

Introduction

Motivationally salient cues attract and hold selective attention, and this "attentional bias" is related to individual differences in appetitive and aversive motivation. For example, anxious emotional states and anxiety disorders are reliably characterized by an attentional bias for threatening information.[1] Attentional biases for substance-related cues are seen in those with substance use disorders, and the strength of attentional bias for alcohol cues is proportional to individual differences in alcohol consumption in nondependent alcohol consumers.[2] The purpose of this review is to explain the theoretical context of attentional bias research related to substance use disorder, describe some of the ways in which it is measured, and provide a critical discussion of the relevance of attentional bias in clinical settings.

*Address for correspondence: Prof. Matt Field, School of Psychology, University of Liverpool, Liverpool, L69 7ZA, UK.
(Email: mfield@liv.ac.uk)

Theoretical Background

Attentional bias for drug-related cues is thought to develop as a consequence of a classical conditioning process. Repeated pairings of drug-related cues (conditioned stimuli) with the rewarding effects of those drugs (unconditioned stimuli) results in the elicitation of conditioned responses when exposed to those drug cues. Conditioned responses may include attentional orienting toward the cue, increased subjective craving, physiological arousal, and drug-seeking behavior. According to some theoretical models, the attentional orienting response (attentional bias) may directly modulate the other responses to drug-related cues.[2,3]

General models of addiction describe neuroadaptations and psychological changes that underlie the shift from recreational substance use to the loss of control over use that is arguably the defining feature of substance use disorders.[4] For example, sensitization of

dopamine function may occur in subcortical structures that govern reward and associative learning, resulting in strong cravings and an increase in the incentive-motivational "pull" of substance-related cues, such as the sight and smell of alcoholic drinks.[5] In addition, hypoactivity in distinct subregions of the prefrontal cortex results in reduced inhibition of subcortical structures, which manifests as executive dysfunction and loss of control over behavior.[6] Cognitive models of addiction make predictions that can be overlaid on these neurobiological models. For example, dual-process models[7] posit that repeated substance use leads to an increase in automatic appetitive processing of substance cues, including attentional biases and automatic approach behavior directed toward those cues, combined with suppression of cognitive control processes that ordinarily mitigate the impact of automatic processes on behavior. Finally, a hybrid neurobiological–cognitive model[3] proposes that "a drug stimulus produces an increase in dopamine levels in the corticostriatal circuit … which in turn serves to draw the subject's attention toward a perceived drug stimulus. This process results in motor preparation and a hyper-attentive state towards drug-related stimuli that, ultimately, promotes further craving and relapse" (p. 563). A schematic overview of this model is shown in Figure 1.

When we combine these theoretical models, the following predictions emerge:

1. Attentional bias for substance-related cues is a characteristic of substance use disorders.
2. The bias is associated with the strength of craving at that moment in time.
3. Strong attentional bias increases the risk of relapse to substance use in those who are attempting to remain abstinent or to reduce their substance use.

These predictions will be evaluated in this article.

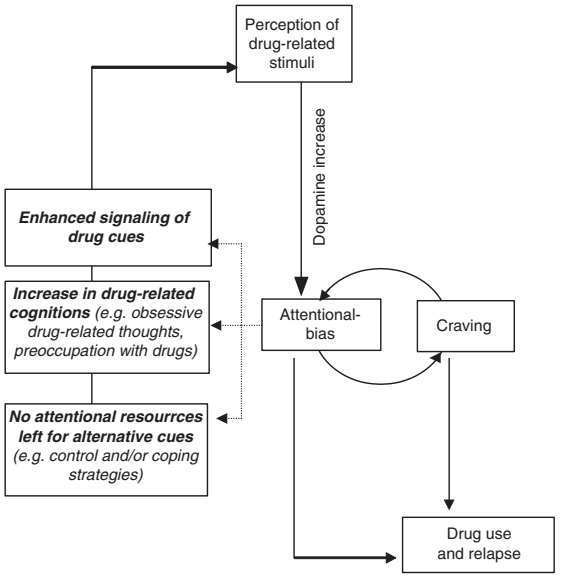

Figure 1. Schematic illustrating the activation of attentional bias and its possible relationship with craving, drug use, and relapse. A drug-related cue is perceived and evokes an increase in dopaminergic activity in subcortical structures, causing it to be flagged as "salient," and thereby grabbing the attention. The individual then ruminates on drug use, is unable to engage coping mechanisms, finds it difficult to disengage from the cue, and experiences an increase in craving. This causes further increases in craving, creating a vicious cycle that may ultimately result in relapse. Reproduced with permission from Franken (2003)[(3)].

Measurement of Attentional Bias

Attentional bias for substance-related cues can be measured directly (for example, by monitoring eye movements), or it can be indirectly inferred with reaction time or other measures. Indirect measures of attentional bias, particularly the modified Stroop and visual probe tasks, are the most commonly used tasks in part because of their ease of administration. In the word version of the addiction Stroop task, participants are presented with words that are printed in different colors and they are required to name the ink color in which the words are printed while ignoring the semantic content of the words. Substance abusers, but not control subjects, are generally slower to name the color of substance-related words (eg, "pipe," "crack") compared to matched neutral words (eg, "desk," "chair"), which is interpreted as involuntary automatic processing of the drug-related words.[8] In a typical visual probe task, a drug-related picture (eg, a pack of cigarettes) is presented alongside a matched neutral picture (eg, a pack of pens) on a computer screen for a short period (typically between 50 and 2000 milliseconds). The pictures are then removed from the display to be immediately replaced by a visual probe that appears on either the left or right of the screen, such that it appears either in the spatial location that had been occupied by the drug-related picture or on the opposite side of the screen. Participants are instructed to respond to this probe as quickly as possible: If they are consistently faster to respond to probes that replace drug pictures compared to neutral pictures, we assume that they were looking at the drug-related picture (ie, attentional bias). Studies using this task have generally revealed attentional bias for drug cues among those with tobacco, opiate, or cocaine dependence.[2] The picture in alcohol abusers is more complex: light drinkers show no bias, heavy drinkers who are not seeking treatment generally show attentional bias for alcohol cues, whereas alcohol-dependent

patients who are receiving treatment may show overt attentional avoidance of alcohol cues.[9]

In recent years, both of these tasks have been criticized owing to their poor internal reliability,[10] and due to ambiguity about the attentional subprocesses that they measure.[2] These criticisms may not apply to more direct measurements of attentional bias, such as eye movement monitoring.[11] For example, direct measurement of participants' eye movements while they complete a visual probe task reveals that substance abusers hold their gaze on substance-related cues for longer than on neutral cues, and this bias tends to be highly correlated with the reaction time index of attentional bias that is obtained from the task.[12] Although direct measurement of attentional biases using eye-tracking may be preferable to indirect measures due to the superior reliability and construct validity of eye-tracking, indirect measures may be preferred for practical purposes, including the high cost of eye-tracking equipment.

In addition to using reaction time tasks to measure attentional bias, investigators have recently studied the neural substrates of attentional bias using functional magnetic resonance imaging (fMRI) and electroencephalography (EEG) measures. Two specific electrophysiological indices of cognitive processing, the P300 and slow potential (SP) components of the event-related brain potential (ERP), are associated with the deployment of attentional resources to visual stimuli. A recent meta-analysis[13] showed that the P300 and SP amplitudes in response to substance cues are typically larger in substance abusers than controls. In addition, some recent fMRI studies showed that activation in the anterior cingulate cortex,[14–18] dorsolateral prefrontal cortex,[15,18,19] insula,[14,16,18] nucleus accumbens,[17] and amygdala[16,18,20] is associated with attentional bias for substance-relevant stimuli as compared to neutral stimuli. Therefore, these specific EEG and fMRI indices of brain activation could also be used as indirect measures of attentional bias.

Does Attentional Bias Predict Who Will Relapse?

In addition to the numerous studies that show cross-sectional associations between attentional bias and substance use, one study revealed that individual differences in attentional bias for alcohol cues predicted future drinking behavior in a sample of heavy drinkers who were not seeking treatment. In this study, participants in whom attentional bias was low at baseline showed larger reductions in drinking at 6-month follow-up compared to participants in whom attentional bias was high at baseline.[21] Based on this, we might expect that attentional bias, assessed during or shortly after treatment for substance dependence, would be a useful predictor of the likelihood that patients could maintain abstinence in the long-term. Individuals with elevated attentional bias should be less likely to maintain abstinence in the long-term.

Some studies appear to support this prediction: Individual differences in drug Stroop interference assessed in the clinic predict the likelihood of remaining abstinent after discharge.[16,22–26] However, several studies that used either the visual probe task or the modified Stroop (or both) failed to find this relationship between behavioral measures of attentional bias assessed in the clinic and subsequent relapse.[27–29] The inconsistency may be partly attributable to variations in methods (eg, attentional bias measure used, duration of follow-up) and populations studied. For example, closer inspection of individual studies suggests that attentional bias may be a more robust predictor of relapse in the first few days after treatment, but at longer follow-up periods (typically, several months), this predictive relationship is more inconsistent. Overall, the evidence that attentional bias for drug cues assessed during treatment is a reliable predictor of subsequent relapse is equivocal, and more studies are needed before synthesizing this research in a systematic review.

Some recent findings suggest a resolution to this issue. For example, a recent study showed that anterior cingulate cortex activation during performance of a cocaine Stroop task predicted cocaine use at 3 month follow-up, whereas behavioral performance on the cocaine Stroop task did not.[29] This suggests that patterns of brain activity that are correlated with attentional bias may be more sensitive predictors of future drug use than behavioral measures of the bias. In addition, two recent studies measured attentional bias using experience sampling techniques, which involved participants completing a drug Stroop task on a handheld computer when prompted to do so at random intervals throughout the day, or when they experienced a temptation to use the drug. These studies revealed that attentional bias was elevated in the hours before participants reported being tempted to use heroin and cocaine,[30] and elevated attentional bias preceded relapse.[31] See Figure 2. We await replication of these findings, but they suggest that attentional bias for drug cues may peak in the days before participants relapse, which would be consistent with the theoretical model depicted in Figure 1.[3] On the other hand, when attentional bias is assessed in a treatment setting using only behavioral measures, this may not be a robust predictor of relapse to drug use several months later. We address this topic in the following section.

Attentional Bias Fluctuates with Motivational State

There is much debate about the conceptualization of subjective drug craving, its measurement, and its

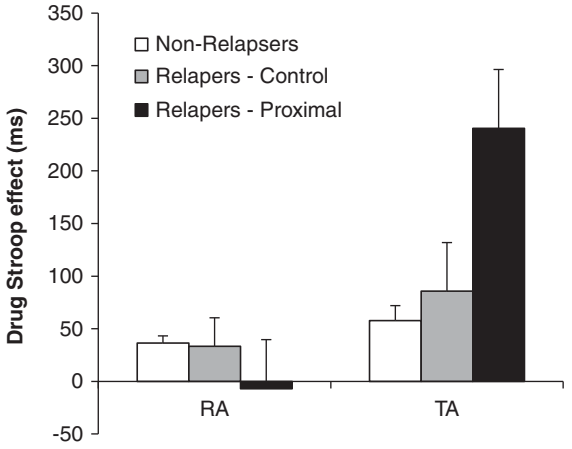

Figure 2. Attentional bias for drug cues assessed with a modified Stroop task administered on a handheld device to participants while they were in their first week of residential treatment. Participants completed the task at randomly prompted intervals (RAs) or when tempted to use the drug (TAs). Participants who relapsed versus those who did not were distinguished. During TAs (but not RAs), attentional bias was elevated in those who subsequently relapsed versus those who did not. Furthermore, there was a marked peak in attentional bias when assessed soon before participants relapsed (proximal assessment) compared to when assessed at other times (control assessment). Overall, this study shows that attentional bias is elevated in participants who relapse compared to participants who do not, and bias is particularly high in the days that precede a relapse. Reproduced with permission from Marhe *et al.* (2013)[(31)].

clinical significance.[3,5] As predicted by Franken[3] (see Figure 1), there is a small but robust association between the magnitude of attentional bias and the strength of subjective craving for the drug.[32] Importantly, the magnitude of the correlation between the two increases when attention is measured directly (for example, with eye movements) rather than being indirectly inferred from reaction time measures (such as the drug Stroop task). Therefore, attentional bias seems to be associated with the strength of drug craving, but the two are not synonymous. Given that craving fluctuates within individuals over time,[33] we might expect attentional bias to do the same, and indeed the aforementioned experience sampling studies suggest a large degree of within-subject variation in the magnitude of attentional bias.[30,31] Experimental studies with heavy drinkers and tobacco smokers reveal that attentional biases are increased as a consequence of stress,[34] alcohol intoxication,[35,36] withdrawal (from tobacco),[37] exposure to drug cues,[38,39] and having an imminent opportunity to consume the substance.[40,41]

Each of these factors (eg, stress, alcohol intoxication, opportunity) is known to increase the desire in the laboratory to consume the drug. Furthermore, self-report data from patients who relapsed indicate that these factors can be a trigger for relapse after a period of abstinence. Therefore, when considered alongside recent experience sampling studies,[30,31] attentional bias may peak during "high-risk" situations for relapse alongside increases in subjective craving. This may also clarify why we see mixed findings regarding the predictive validity of attentional bias when assessed in treatment contexts. In these contexts, most of these triggers are likely to be absent: Withdrawal symptoms are likely to be well managed (by pharmacotherapy), and there are no drug cues or opportunities to use the drug within the treatment facility. Furthermore, when patients are tested during or shortly after completing a treatment program, they are likely to view their drug use negatively, which may explain why alcoholic inpatients show attentional avoidance of alcohol cues in these circumstances.[9] Considering these observations, we would not predict any robust predictive relationship between attentional bias and relapse if attentional bias is assessed in a treatment context, but relapse occurs weeks or months later in very different contexts. We await experience sampling studies in naturalistic settings that can address these issues.

Treatment Implications

Attentional bias is a feature of substance use disorders, it fluctuates in line with motivational state, and it may peak in the days before people relapse to substance use after a period of abstinence. It is important to consider whether attentional bias could be a viable target for novel treatment interventions. In order to answer this question, we must first ask whether attentional bias has a causal influence on the risk of relapse or the strength of drug craving. A number of studies investigated this issue by first experimentally manipulating attentional bias for alcohol or tobacco cues in the laboratory, and then exploring the effects of these manipulations on subjective craving and drug-seeking behaviors that were measured immediately after the training. Despite some promising initial findings,[42] a series of studies have failed to demonstrate robust effects of attentional bias modification (ABM) on craving or drug-seeking assessed in the laboratory.[43–46] However, these studies involved only a single session of ABM that was administered to college students who were not motivated to reduce their drinking or smoking behavior, which leaves open the possibility that ABM may be an effective treatment for substance abusers who are motivated to reduce their substance use, or abstain altogether. Two studies investigated the efficacy of multiple sessions of ABM in alcohol abusers. One study[47] investigated a Stroop-based ABM

procedure and reported that heavy drinkers who received it reported reductions in alcohol consumption at follow-up. However, the absence of a control group or any kind of mediation analysis in this study makes it impossible to rule out nonspecific effects as an explanation for the observed reduction in drinking. Another study[48] administered a visual probe–based ABM procedure or a suitably matched control intervention to alcohol-dependent patients who were attempting to remain abstinent from alcohol. Groups did not differ on overall relapse rates at follow-up, although time-to-relapse was delayed in the group receiving ABM compared to the control group. However this was a small pilot study. A well-powered clinical trial (using a similar methodology) is required to obtain a more conclusive answer on the potential clinical effectiveness of ABM as a treatment intervention for substance use disorders. In this context, it is notable that ABM for threat-related information has shown some promise as an adjunct treatment for anxiety disorders.[49] One suggestion for future research is to investigate the effectiveness of ABM if delivered via handheld devices such as smartphones, so that patients can benefit from a brief session of ABM if they feel tempted to use the drug.[30]

Conclusions

Attentional bias for drug-related cues is a reliable feature of substance use disorders. The bias fluctuates in line with motivational state: It is correlated with the strength of craving, and it increases in response to manipulations such as stress, withdrawal, and cue exposure, which are known to increase the risk of relapse to drug use after a period of abstinence. If we assess attentional bias in a treatment setting, it does not seem to be a reliable predictor of relapse several months later. However, moment-by-moment fluctuations in attentional bias are meaningful, because substance abusers are more likely to be tempted to use drugs, and to relapse, when attentional bias is high. At present, there is no convincing evidence to recommend the use of attentional bias modification as an adjunct treatment for substance use disorders, although larger trials and alternative methodologies are required to investigate this issue.

Disclosures

The authors do not have anything to disclose.

References

1. Cisler JM, Koster EHW. Mechanisms of attentional biases towards threat in anxiety disorders: an integrative review. *Clin Psychol Rev*. 2010; **30**(2): 203–216.
2. Field M, Cox WM. Attentional bias in addictive behaviors: a review of its development, causes, and consequences. *Drug Alcohol Depend*. 2008; **97**(1–2): 1–20.
3. Franken IHA. Drug craving and addiction: integrating psychological and neuropsychopharmacological approaches. *Prog Neuropsychopharmacol Biol Psychiatry*. 2003; **27**(4): 563–579.
4. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. Washington, DC: American Psychiatric Association; 2013.
5. Robinson TE, Berridge KC. The neural basis of drug craving: an incentive-sensitization theory of addiction. *Brain Res Brain Res Rev*. 1993; **18**(3): 247–291.
6. Goldstein RZ, Volkow ND. Dysfunction of the prefrontal cortex in addiction: neuroimaging findings and clinical implications. *Nat Rev Neurosci*. 2011; **12**(11): 652–669.
7. Stacy AW, Wiers RW. Implicit cognition and addiction: a tool for explaining paradoxical behavior. *Annu Rev Clin Psychol*. 2010; **6**: 551–575.
8. Cox WM, Fadardi JS, Pothos EM. The addiction-stroop test: theoretical considerations and procedural recommendations. *Psychol Bull*. 2006; **132**(3): 443–476.
9. Noël X, Colmant M, Van Der Linden M, *et al*. Time course of attention for alcohol cues in abstinent alcoholic patients: the role of initial orienting. *Alcoholism Clin Exp Res*. 2006; **30**(11): 1871–1877.
10. Ataya AF, Adams S, Mullings E, *et al*. Internal reliability of measures of substance-related cognitive bias. *Drug Alcohol Depend*. 2012; **121**(1–2): 148–151.
11. Field M, Christiansen P. Commentary on 'Internal reliability of measures of substance-related cognitive bias.' *Drug Alcohol Depend*. 2012; **124**(3): 189–190.
12. Mogg K, Bradley BP, Field M, De Houwer J. Eye movements to smoking-related pictures in smokers: relationship between attentional biases and implicit and explicit measures of stimulus valence. *Addiction*. 2003; **98**(6): 825–836.
13. Littel M, Euser AS, Munafò MR, Franken IHA. Electrophysiological indices of biased cognitive processing of substance-related cues: a meta-analysis. *Neurosci Biobehav Rev*. 2012; **36**(8): 1803–1816.
14. Luijten M, Veltman DJ, den Brink WV, *et al*. Neurobiological substrate of smoking-related attentional bias. *Neuroimage*. 2011; **54**(3): 2374–2381.
15. Luijten M, Veltman DJ, Hester R, *et al*. Brain activation associated with attentional bias in smokers is modulated by a dopamine antagonist. *Neuropsychopharmacology*. 2012; **37**(13): 2772–2779.
16. Janes AC, Pizzagalli DA, Richardt S, *et al*. Brain reactivity to smoking cues prior to smoking cessation predicts ability to maintain tobacco abstinence. *Biol Psychiatry*. 2010; **67**(8): 722–729.
17. Nestor L, McCabe E, Jones J, Clancy L, Garavan H. Differences in "bottom-up" and "top-down" neural activity in current and former cigarette smokers: evidence for neural substrates which may promote nicotine abstinence through increased cognitive control. *Neuroimage*. 2011; **56**(4): 2258–2275.

18. Vollstädt-Klein S, Loeber S, Richter A, *et al*. Validating incentive salience with functional magnetic resonance imaging: association between mesolimbic cue reactivity and attentional bias in alcohol-dependent patients. *Addict Biol*. 2012; **17**(4): 807–816.
19. Hester R, Garavan H. Neural mechanisms underlying drug-related cue distraction in active cocaine users. *Pharmacol Biochem Behav*. 2009; **93**(3): 270–277.
20. Janes AC, Pizzagalli DA, Richardt S, *et al*. Neural substrates of attentional bias for smoking-related cues: an fMRI study. *Neuropsychopharmacology*. 2010; **35**(12): 2339–2345.
21. Cox WM, Pothos EM, Hosier SG. Cognitive-motivational predictors of excessive drinkers' success in changing. *Psychopharmacology*. 2007; **192**(4): 499–510.
22. Carpenter KM, Schreiber E, Church S, McDowell D. Drug Stroop performance: relationships with primary substance of use and treatment outcome in a drug-dependent outpatient sample. *Addict Behav*. 2006; **31**(1): 174–181.
23. Cox WM, Hogan LM, Kristian MR, Race JH. Alcohol attentional bias as a predictor of alcohol abusers' treatment outcome. *Drug Alcohol Depend*. 2002; **68**(3): 237–243.
24. Marissen MAE, Franken IHA, Waters AJ, *et al*. Attentional bias predicts heroin relapse following treatment. *Addiction*. 2006; **101**(9): 1306–1312.
25. Waters AJ, Shiffman S, Sayette MA, *et al*. Attentional bias predicts outcome in smoking cessation. *Health Psychol*. 2003; **22**(4): 378–387.
26. Powell J, Dawkins L, West R, Powell J, Pickering A. Relapse to smoking during unaided cessation: clinical, cognitive and motivational predictors. *Psychopharmacology*. 2010; **212**(4): 537–549.
27. Waters AJ, Shiffman S, Bradley BP, Mogg K. Attentional shifts to smoking cues in smokers. *Addiction*. 2003; **98**(10): 1409–1417.
28. Field M, Mogg K, Mann B, Bennett GA, Bradley BP. Attentional biases in abstinent alcoholics and their association with craving. *Psychol Addict Behav*. 2013; **27**(1): 71–80.
29. Marhe R, Luitjen M, van de Wetering BJM, Smits M, Franken IHA. Individual differences in anterior cingulate activation associated with attentional bias predict cocaine use after treatment. *Neuropsychopharmacology*. 2013; **38**(6): 1085–1093.
30. Waters AJ, Marhe R, Franken IHA. Attentional bias to drug cues is elevated before and during temptations to use heroin and cocaine. *Psychopharmacology*. 2012; **219**(3): 909–921.
31. Marhe R, Waters AJ, van de Wetering BJM, Franken IHA. Implicit and explicit drug-related cognitions during detoxification treatment are associated with drug relapse: an ecological momentary assessment study. *J Consult Clin Psychol*. 2013; **81**(1): 1–12.
32. Field M, Munafò MR, Franken IHA. A meta-analytic investigation of the relationship between attentional bias and subjective craving in substance abuse. *Psychol Bull*. 2009; **135**(4): 589–607.
33. Preston KL, Vahabzadeh M, Schmittner J, *et al*. Cocaine craving and use during daily life. *Psychopharmacology*. 2009; **207**(2): 291–301.
34. Field M, Powell H. Stress increases attentional bias for alcohol cues in social drinkers who drink to cope. *Alcohol Alcohol*. 2007; **42**(6): 560–566.
35. Schoenmakers T, Wiers RW, Field M. Effects of a low dose of alcohol on cognitive biases and craving in heavy drinkers. *Psychopharmacology*. 2008; **197**(1): 169–178.
36. Fernie G, Christiansen P, Cole JC, Rose AK, Field M. Effects of 0.4g/kg alcohol on attentional bias and alcohol-seeking behaviour in heavy and moderate social drinkers. *J Psychopharmacol*. 2012; **26**(7): 1017–1025.
37. Field M, Mogg K, Bradley BP. Eye movements to smoking-related cues: effects of nicotine deprivation. *Psychopharmacology*. 2004; **173**(1): 116–123.
38. Field M, Rush M, Cole J, Goudie A. The smoking Stroop and delay discounting in smokers: effects of environmental smoking cues. *J Psychopharmacol*. 2007; **21**(6): 603–610.
39. Cox WM, Yeates GN, Regan CM. Effects of alcohol cues on cognitive processing in heavy and light drinkers. *Drug Alcohol Depend*. 1999; **55**(1–2): 85–89.
40. Field M, Hogarth L, Bleasdale D, *et al*. Alcohol expectancy moderates attentional bias for alcohol cues in light drinkers. *Addiction*. 2011; **106**(6): 1097–1103.
41. Jones A, Hogarth L, Christiansen P, *et al*. Reward expectancy promotes generalized increases in attentional bias for rewarding stimuli. *Q J Exp Psychol (Hove)*. 2012; **65**(12): 2333–2342.
42. Field M, Eastwood B. Experimental manipulation of attentional bias increases the motivation to drink alcohol. *Psychopharmacology*. 2005; **183**(3): 350–357.
43. Field M, Duka T, Eastwood B, *et al*. Experimental manipulation of attentional biases in heavy drinkers: do the effects generalise? *Psychopharmacology*. 2007; **192**(4): 593–608.
44. Field M, Duka T, Tyler E, Schoenmakers T. Attentional bias modification in tobacco smokers. *Nicotine Tob Res*. 2009; **11**(7): 812–822.
45. Attwood AS, O'Sullivan H, Leonards U, Mackintosh B, Munafò MR. Attentional bias training and cue reactivity in cigarette smokers. *Addiction*. 2008; **103**(11): 1875–1882.
46. McHugh RK, Murray HW, Hearon BA, Calkins AW, Otto MW. Attentional bias and craving in smokers: the impact of a single attentional training session. *Nicotine Tob Res*. 2010; **12**(12): 1261–1264.
47. Fadardi JS, Cox WM. Reversing the sequence: reducing alcohol consumption by overcoming alcohol attentional bias. *Drug Alcohol Depend*. 2009; **101**(3): 137–145.
48. Schoenmakers TM, de Bruin M, Lux IFM, *et al*. Clinical effectiveness of attentional bias modification training in abstinent alcoholic patients. *Drug Alcohol Depend*. 2010; **109**(1–3): 30–36.
49. MacLeod C, Mathews A. Cognitive bias modification approaches to anxiety. *Annu Rev Clin Psychol*. 2012; **8**: 189–217.