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Effects of Nicotine and Alcohol on Affective Responses to Emotionally Toned

Film Clips

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Abstract

Rationale: Smoking abstinence can result in decreased affective reactions to positively valenced stimuli and this can be reversed via smoking. Given their shared ability to trigger nucleus accumbens dopamine release, a priming dose of alcohol may likewise augment positive affective responses during abstinence.

Objectives: To replicate our previous finding that compared to satiation, abstinence from smoking will be associated with decreased ‘happiness’ responses to positively valenced film clips (study 1); and to explore whether a priming dose of alcohol can substitute for nicotine by concomitantly enhancing such responses (study 2). In both studies ‘sadness’ responses to negatively valenced clips were also included.

Methods: 32 and 77 smokers respectively in studies 1 and 2 were randomly allocated to abstain from smoking for 10 hours (abstinent smokers) or smoke as usual (satiated smokers).

Participants then rated the extent to which they felt a list of emotions in response to each of 16 film clips. In study 2, participants were additionally allocated to an alcohol manipulation in which they received either alcohol or placebo.

Results: In Study 1, nicotine administration increased abstinent smokers’ ratings of happiness and sadness to the corresponding film clips. In Study 2, nicotine and alcohol both enhanced positive reactivity to happy clips, and their effects were not additive. Alcohol but not nicotine likewise enhanced sadness responses to sad clips.

Conclusions: Abstinence from smoking can result in blunting of affective responses to positively toned stimuli, an effect that can be ameliorated by both nicotine and alcohol. The impact of nicotine on negative reactivity appears to be less robust.

Keywords: Smoking, nicotine, alcohol, abstinence, affective responses, emotional stimuli, priming, cross-priming, happiness, sadness

Introduction

Abstinence from regular smoking can result in diminished responsiveness to rewarding and emotionally-toned stimuli (Dawkins et al. 2006; Dawkins et al., 2007; Powell et al. 2002), and affective symptoms including anhedonia, dysphoria, depressed mood and anxiety (APA 1994; West et al. 1991). Our group has previously found that compared to their performance after smoking, acutely abstinent smokers show reduced allocation of attention to ‘pleasurable’ words on a Stroop task (Dawkins et al., 2006) and reduced responsiveness to a financial incentive on a card-sorting task (the Card Arranging Reward Responsivity Objective Test [CARROT]; Dawkins et al. 2006; Powell et al. 2002); this latter effect was not replicated, however, by Kalamboka et al. (2009) using a variant of the task. We have also found abstinent smokers to be more anhedonic than satiated smokers, showing muted affective responses to everyday pleasures as assessed both by a self-report questionnaire (Dawkins et al. 2006; Powell et al. 2002; Powell et al. 2004) and in an experimental paradigm where they rated their mood state following presentation of happy, sad and emotionally neutral film clips. Thus Dawkins et al. (2007) found that abstinent smokers reported less increase in ‘happiness’ after watching positively valenced clips; there was no effect of recency of smoking on responses to sad or neutral clips. However, Spring et al. (2008) found that nicotine enhanced both the effects of exposure to happy music on smokers’ positive mood and cognitions and the effects of exposure to sad music on their negative mood and cognitions.

These cognitive and affective effects have been putatively linked to underlying neuropharmacological processes, particularly in the mesolimbic dopamine (DA) circuitry – often referred to as the brain reward system – which has been implicated in the attribution of motivational salience to external stimuli: that is, ‘analysis of the information that carries emotive, evaluative and, in the long-term, survival significance for the individual’ (Goldstein and Volkow, 2002). Addictive substances, including nicotine and alcohol, are believed to exert their rewarding effects by stimulating release of nucleus accumbens DA and opioid peptides (Gianoulakis, 2004; Soderpalm, et al., 2000). Relatedly, chronic substance use is associated with hypoactivity in the mesolimbic DA pathway (Altmann et al., 1996; Volkow et al., 2004) and the opioid system (Drews and Zimmer, 2010). It therefore follows that during acute abstinence, when neurochemical activity is not being artificially enhanced by substance use, smokers are likely to show reduced capacity for salience attribution and that this should manifest in motivational and emotional blunting.

It is now well established that during abstinence, a small dose of a previously abused drug can reinstate drug seeking behavior in animals (de Wit, 1996). In humans likewise, one drink can ‘whet the appetite’ and increase subsequent drinking (de Wit and Chutuape, 1993; Stockwell et al., 1982). This phenomenon, known as ‘priming’, is thought to be mediated via increasing extracellular DA activity in the nucleus accumbens (Robinson and Berridge, 1993). ‘Cross-priming’ between different addictive substances has also been demonstrated: that is, the phenomenon whereby a small dose of one drug can enhance craving or responding for another, putatively due to their common activation of the mesolimbic DA system (Robinson & Berridge, 1993). Thus for instance, nicotine administration has been shown to increase alcohol consumption in both rats (Lopez-Moreno et al. 2004) and humans (Barrett et al. 2006); whilst

conversely a priming dose of alcohol has been found to increase smoking behaviour, as indexed by puff volume, rate of smoking, and change in expired carbon monoxide (Mintz et al. 1985; Zacny 1990). Similar alcohol/nicotine cross-priming effects have been reported in relation to elevation of craving (Hillemacher et al. 2006) and cue-elicited craving (Burton and Tiffany 1997; Erblich et al. 2009), although there are also contradictory findings (Palfai et al. 2000).

There is some evidence that the two substances have synergistic effects on the overall level of pleasure experienced. Thus smokers report that drinking alcohol enhances the subjective rewarding effects of smoking (Glautier et al. 1996; Zacny 1990), and nicotine may also enhance the subjective effects of ethanol (Kouri et al. 2004). This may reflect additive neuropharmacological effects on shared neurochemical systems; for example, they both target nicotinic acetylcholine receptors (Spanagel, 2009), nicotine via selective effects on α and β subunits, and alcohol as part of wide-ranging effects on multiple neurotransmitter systems (see e.g. Vengeliene et al. 2008; Wang et al. 1994)

To date, no published studies have investigated whether these cross-priming effects apply to affective and motivational responses to stimuli which are not directly drug-related stimuli; if the depression of brain reward pathways seen during abstinence indeed has a generalised effect on salience attribution, as suggested by the previous behavioural studies in humans described above, then such responses should be enhanced by administration of any addictive substance. Consistent with this hypothesis, pilot work conducted in our laboratory and described in a doctoral thesis (McFie, 2005) has indicated that administration of alcohol (but not placebo) to abstinent smokers increases their responsiveness to financial incentive on the CARROT. We therefore set out here to determine, using the methodology developed in McFie's pilot work, whether priming doses of

nicotine and alcohol would individually affect emotional reactivity to affectively toned film clips and whether any separate effects would be additive.

The present paper describes two studies. The first aimed to replicate our previous finding that in regular smokers, ‘happiness’ responses to positively-valenced film clips are attenuated during abstinence and/or augmented after smoking, and additionally to investigate whether this effect is confined to affectively positive responses or whether it extends also to affectively negative (sadness) responses. The second study investigated possible cross-priming between alcohol and nicotine in the same paradigm, testing the hypothesis that a small dose of alcohol would augment abstinent smokers’ ‘happiness’ responses to positively valenced stimuli; the effects on affective responses to negatively valenced (sad) clips were also explored.

STUDY 1

Materials and methods

Participants

Thirty-two smokers (21 female) were recruited; all were aged between 18 and 32 (mean: 22) and reported smoking 10 or more cigarettes a day for at least the previous year. All were students at Goldsmiths, University of London, and received either ‘course credits’ or £10 for their participation. They gave written informed consent, and the study was approved by Goldsmiths Ethics Committee.

Design and Procedure

In this mixed design, smokers were randomly assigned to either a 10 hour ‘abstinent’ condition (N = 15), or a ‘satiated’ condition (N = 17) in which they smoked as usual and within the 15

minutes prior to the testing session. Expired carbon monoxide (CO) levels were measured to verify compliance with experimental instructions. Abstinence was verified by CO readings of ≤ 10 ppm, and satiety by $\text{CO} \geq 10$ ppm (see Jarvis 1987; Jo and Oh 2003).

Participants were tested in mixed groups of 5 to 12 satiated and abstinent smokers in a small seminar room with easy chairs. After completing some baseline questionnaires and providing the breath carbon monoxide sample they watched sixteen film clips varying in affective valence, rating their emotional responses after each clip without talking to or conferring with other participants.

Assessments of dependence and withdrawal symptoms

The Fagerström Test of Nicotine Dependence (FTND; Heatherton et al. 1991): Total scores on this 6-item self-report scale range from 0 (low dependence) to 10 (high dependence).

The Mood and Physical Symptoms Scale (MPSS; West and Hajek 2004): This measures the severity of 7 signs and symptoms associated with withdrawal (depression, irritability, anxiety, drowsiness, restlessness, hunger, poor concentration). Participants rated each item on a 5-point scale (0-4), the total score thus ranging 0 to 28 with a higher score reflecting greater symptom severity.

Experimental assessment of emotional reactivity

16 film clips were selected on the basis of a pilot study in which volunteers rated the emotions they elicited. Six were categorized as 'happy' and three as 'sad' on the basis that they elicited significantly higher ratings of 'happiness' or 'sadness' respectively than of 'anxiety' 'disgust'

and 'contentment'; two 'neutral' clips had mean and modal ratings which were undifferentiated across emotions (Dillon 2007, doctoral thesis; Gross and Levenson 1995). The other five clips (disgust, anxiety and contentment) are not of interest here but were included as filler stimuli to camouflage the obvious contrast between happy and sad.

Clips averaged 136 seconds (range: 30-252 seconds); this did not differ significantly between clip type. They were presented on a 19 inch TV monitor in one of two different orders to which participants were assigned randomly; no two clips of the same affective valence were contiguous. During a 1 minute pause after each clip participants rated the extent to which they had felt a list of emotions including happiness and sadness. '0' indicated 'not the slightest bit' and '8' 'the most they had ever felt' (Gross and Levenson 1995).

Mean 'happiness' and 'sadness' ratings were computed for the six 'happy' and three 'sad' clips respectively, and were compared with the mean 'happiness' and 'sadness' ratings associated with the two neutral clips.

Statistical analysis

Two repeated measures analyses of variance (one for happiness and one for sadness) were conducted in SPSS with the between-subjects factor of GROUP (2 levels: satiated smokers vs abstinent smokers) and the within-subjects factor of CLIPTYPE (2 levels: happy or sad vs neutral). The GROUP X CLIPTYPE interaction is of specific interest here, since it is hypothesized that satiated smokers will show a more pronounced affective response to the emotionally valenced clips than will abstinent smokers.

Given the possible influence of the two orders of clip presentation, we initially ran the analyses with ORDER as an additional between-subjects factor. As it did not yield any interactive effects, it is not considered further here.

Results

Descriptive statistics are displayed in Table 1. The 15 abstinent smokers and 17 satiated smokers did not differ from each other in age ($t_{30} < 1$, ns), FTND scores ($t_{30} < 1$, ns) or sex ratio ($\chi^2 < 1$, $df = 1$, ns). Against expectation, neither did they differ in baseline mood and physical symptom scale scores (MPSS: $t_{30} < 1$, ns).

- TABLE 1 ABOUT HERE -

Affective ratings to emotionally-toned and neutral film clips

Mean affective ratings in response to the happy and sad clips along with the corresponding affective rating to neutral clips are illustrated graphically in Figure 1 (a & b).

- FIGURE 1 ABOUT HERE -

There was a highly significant main effect of CLIPTYPE for both happiness and sadness responses ($F_{1,30} > 50.0$, $p < 0.0001$ in both cases) with the affectively toned clips eliciting stronger mood states than the neutral clips. Satiated smokers also reported overall stronger emotional responses than abstinent smokers, reflected in significant main effects of GROUP for both happy ($F_{1,30} = 10.39$, $p < 0.005$) and sad ($F_{1,30} > 6.87$, $p = 0.01$) clips. More importantly, the GROUP X CLIPTYPE interactions were also significant. That is, by comparison with ratings to neutral clips, the increases in happiness and sadness elicited by happy and sad clips respectively

were significantly less pronounced in abstinent smokers than in satiated smokers (happiness, $F_{1,30} = 8.71$, $p < 0.01$; sadness, $F_{1,30} = 9.77$, $p < 0.005$). Post-hoc between-subject t-tests confirmed that affective ratings to both happy and sad clips differed significantly between abstinent and satiated smokers (happy: $t_{30} = 3.18$, $p < 0.005$; sad: $t_{30} = 2.98$, $p < 0.01$) whilst the corresponding emotional ratings to neutral clips did not ($t_{30} < 1$, ns in both cases).

STUDY 2

Materials and methods

Participants

Seventy-seven smokers aged between 18 and 57 (mean: 29), of whom 51 were women, were recruited. All described themselves as social drinkers (i.e. they consumed alcohol at least once a week but had no self-reported alcohol-related problems) and reported having smoked 10 or more cigarettes a day for at least the last year. Approximately half were students at the University of East London (UEL) and received 'course credits' for their participation. The remaining participants, recruited using a snowballing technique, were friends of the students and experimenters and were given a £10 voucher. All participants provided written informed consent and the study was approved by the University of East London Ethics Committee.

Design and Procedure

Participants were randomized between four experimental conditions defined by pre-test consumption of nicotine (via cigarette), alcohol, neither, or both. Thus there were two between-subjects factors: SMOKE (satiated vs. abstinent) and ALCOHOL (alcohol vs. placebo). This yielded four groups: satiated and alcohol (NIC+ALC); satiated and placebo (NIC+PLB);

abstinent and alcohol (AB+ALC); abstinent and placebo (AB+PLB). Smoking abstinence and satiety instructions and verification were exactly as described in Study 1.

Both experimenter and participants were blinded to the alcohol manipulation, an independent research assistant preparing the drinks). Following a protocol developed by Glautier et al (1992), for the alcohol drink 15ml of vodka (Smirnoff; 37.5% vol) was mixed with 45ml of Indian tonic water; in the placebo drink, vodka was replaced by spring water (15ml). Both beverages were flavoured with 1.5ml of Angostura bitters, and were administered in white plastic disposable cups. At the end of the experiment participants were asked to indicate which beverage they thought they had received.

15ml of vodka is less than a standard 25ml pub unit and thus well below the amount most people would choose to drink socially. It yields less than 0.1g of alcohol per kg of body weight, a dose previously found by Duka et al (1998) to increase subsequent alcohol consumption in social drinkers, and which does not produce overt inebriation or sedation likely to counteract or camouflage any priming effects. In the unpublished study by McFie (doctoral dissertation, 2005) this dose was found to significantly elevate responsiveness to reward on the CARROT in both male and female smokers, suggesting that the effect was not related to body mass index. For these reasons, we opted for the simplicity of utilising a fixed dose rather than adjusting it to body weight; although it is possible that priming effects might be to some extent dose-dependent, it was beyond the scope of the present study to investigate this.

Groups of 2 to 5 participants from any combination of the experimental conditions were tested in a small seminar room. Upon arrival, participants completed some baseline questionnaires (as in

Study 1) and provided a breath carbon monoxide sample. Following consumption of their drink participants were required to wait for 30 minutes. During this time they were permitted to read but were not permitted to leave the premises. They then watched the same sequences of clips and gave ratings after each, exactly as in Study 1.

Assessment Measures

As for Study 1.

Statistical Analysis

As in Study 1, separate repeated measures analyses of variance were conducted for ratings of happiness and sadness with the within-subjects factor of CLIPTYPE (happy or sad vs neutral) and the two between-subjects factors of SMOKESTATUS (satiated vs abstinent) and ALCSTATUS (alcohol vs. placebo drink). Although the 2-way SMOKESTATUS X CLIPTYPE and ALCSTATUS X CLIPTYPE interactions are reported, it is the 3-way CLIPTYPE X SMOKESTATUS X ALCSTATUS which is of principal interest here since this tests the hypothesis that alcohol and nicotine intake will produce either additive or separate but non-additive effects on affective responses.

Results

Descriptive statistics are displayed in Table 2. The four groups did not differ from each other in age ($F_{3,76} < 1$, ns), FTND scores ($F_{3,76} = 1.14$, ns) or sex ratio ($\chi^2 = 3.90$, $df = 3$, ns). The apparently slightly lower dependence (FTND) scores of smokers assigned to the abstinent than the satiated conditions was not significant ($t = -1.66$, $p = 0.10$). Independent samples t-test

revealed significantly higher withdrawal symptoms in abstinent smokers ($M = 6.05$; $SD = 3.61$) than satiated smokers ($M = 3.92$; $SD = 3.81$; $t_{75} = 2.52$, $p = 0.01$).

-TABLE 2 ABOUT HERE -

Alcohol/Placebo manipulation

Participants performed at chance level in guessing whether they had received alcohol or placebo ($\chi^2 = 1.05$; $df = 1$; ns).

Affective ratings to emotionally-toned and neutral film clips

Mean happiness responses to happy and neutral clips are illustrated graphically in Figure 3a and mean sadness responses to sad and neutral clips in Figure 3b.

- FIGURE 3 a & b ABOUT HERE -

(a) Happy vs neutral clips:

There was as expected a highly significant main effect of CLIPTYPE ($F_{1,73} > 50.0$, $p < 0.0001$) with happy clips eliciting stronger happiness than the neutral clips.

There were no main effects of either SMOKESTATUS or ALCSTATUS, nor did either interact with CLIPTYPE ($F_{1,73} < 1.5$, ns, in all cases). There was, however, a significant 3-way CLIPTYPE x SMOKESTATUS x ALCSTATUS interaction ($F_{1,73} = 5.20$, $p < 0.05$). Follow up 2 X 2 ANOVAs revealed a marginally significant SMOKESTATUS x ALCSTATUS interaction ($F_{1,73} = 3.27$, $p = 0.075$) for positive affective responses to happy clips but no significant

interaction for such responses to neutral clips ($F_{1,73} < 1$, ns). Post hoc t-tests confirmed that alcohol significantly enhanced positive affective responses to happy clips in abstinent smokers ($t_{37} = -1.99$, $p < 0.05$) but had no such effect in satiated smokers ($t_{37} < 1$, ns). Likewise, nicotine enhanced positive affective responses to happy clips in participants who had consumed the placebo beverage ($t_{35} = -1.90$, $p = 0.06$) but not in those who had consumed alcohol ($t_{38} < 1$, ns).

(b) Sad vs neutral clips

The effect of CLIPTYPE was highly significant, as expected ($F_{1,73} > 50.0$, $p < 0.0001$), with sad clips eliciting stronger sadness ratings than neutral clips.

There were marginally significant main effects for both SMOKESTATUS ($F_{1,73} = 3.23$, $p = 0.08$) and ALCSTATUS ($F_{1,73} = 3.41$, $p = 0.07$), with participants who had consumed either substance reporting greater sadness overall. For ALCSTATUS this was qualified by an interaction with CLIPTYPE ($F_{1,73} = 3.30$, $p = 0.07$), follow-up t-tests indicating that alcohol significantly enhanced sadness responses to sad clips ($t_{75} = -1.97$, $p < 0.05$) but not to neutral clips ($t_{75} < 1$, ns). Neither the SMOKESTATUS X CLIPTYPE nor the 3-way interaction were significant ($F_{1,73} \leq 1$, ns in both cases).

Discussion

The results of Study 1 replicated our previous findings (Dawkins et al 2007) that abstinence from smoking attenuates the self-reported happiness responses to positively valenced film clips shown by satiated smokers. Moreover, 'sadness' responses to sad clips were also significantly attenuated in abstinent smokers. Whilst this latter result conflicts with the absence of such an effect in our 2007 study, it is consistent with the findings from Spring et al.'s (2008) mood

induction study of reduced positive and negative reactivity in smokers who were tested after smoking a denicotinized cigarette. The present study had a larger sample and more robust design than the earlier one, which may have lacked statistical power to detect the effect of nicotine in amplifying negative affect responses. Thus, these data suggest that in dependent smokers nicotine enhances both positive and negative affective reactions and /or abstinence blunts them (although see Study 2).

An anomaly of this study was that the abstinent smokers did not report more severe nicotine withdrawal symptoms than satiated smokers. When the mean symptom scores of participants in this study are compared with those in the second study – where the expected elevation of scores in abstainers was observed – it appears that whilst abstainers in the two studies scored similarly (6.5 and 6 respectively) the satiated smokers in Study 1 reported markedly more severe symptoms than those in Study 2 (7 vs 4). Nicotine withdrawal symptoms, as listed in the assessment tool used here (MPSS), are on the whole rather non-specific (e.g. depression, anxiety, poor concentration) and it is possible that some of the non-specific symptoms were elevated in a proportion of the participants for reasons unconnected with their smoking status, for example general ill health; with sample sizes of under 20 in each group, such chance vagaries can have a disproportionate effect. Whatever the explanation, however, the fact that abstainers were not more symptomatic than satiated smokers means that the blunting of their affective responses cannot be attributed simply to general withdrawal-associated malaise or low mood. The neurobiological model outlined in the introduction accordingly gains strength as a potential explanation for the observed effect: that is, during abstinence there is a dampening of the mesolimbic DA circuitry believed to underpin the attribution of motivational salience to external stimuli (Goldstein and Volkow 2002). There is substantial preclinical evidence that this system

mediates affective responses to both pleasurable and aversive stimuli (e.g. Di Chiara, 1995; Salamone, 1994), and in humans Fear and Healy (1996) have reported that neuroleptic treatment (which blocks dopamine activity) is associated with reduced sensitivity to interference from threat-related words in a modified Stroop paradigm.

Whilst the salience attribution account is compelling, other plausible explanations could implicate different cognitive processes with other neurobiological correlates. For example, the attenuation of emotional responses might reflect generalized impairments of attention emanating from disturbances in other neurochemical systems modulating arousal such as the nicotinic acetylcholine system which both nicotine and alcohol target (Spanagel, 2009). In any event, now that the effect of nicotine on affective responding has been replicated, future empirical investigation could seek to distinguish between competing underlying cognitive mechanisms; for instance, the possible role of attentional impairment could be tested by asking participants factual questions about the clips after having watched them.

Study 2 tested, and confirmed, the hypothesis that a very small ‘priming’ dose of alcohol would restore emotional responding in abstinent smokers. The double blind placebo-controlled design entailed administering alcohol or placebo beverages, indistinguishable to participants, to smokers who were either abstinent or satiated; administration of alcohol augmented both the positive and the negative emotional reactivity of abstainers to happy and sad clips respectively. There were significant effects of both alcohol and nicotine on happiness responses, and a significant effect of alcohol on sadness responses; in neither case were the effects of the two substances additive. Interestingly, and in contrast to Study 1, nicotine did not increase negative reactivity to sad clips.

It is unclear why this was the case but it suggests that the effect of nicotine on negative reactivity is less reliable than its effect on positive reactivity.

Whilst little is yet known about the dose dependency or time course of priming effects, it is notable that the fixed alcohol dose of 15ml vodka used here was lower even than the small dose of 0.1g per kg of body weight found by Duka et al. (1998) to increase alcohol consumption ten minutes later. As noted previously, in a pilot study in our laboratory using the same protocol as employed here, McFie (2005) found this dose to increase abstinent smokers' reward responsivity on the CARROT. Given the 30 minute delay between consuming the very small dose of alcohol (15 ml vodka) and exposure to the film clips, blood alcohol levels would have been close to zero by the time of testing. The observed enhancement of emotional reactivity is thus unlikely to reflect an acute effect of alcohol on brain function, but is consistent with its ingestion having triggered a sustained increase in the reactivity of the circuitry underlying such responses via either a direct effect in stimulating neurochemical activity or indirectly via associative learning (e.g. a conditioned response to the presence of alcohol in the system). In the absence of biological markers in the present study this interpretation is necessarily speculative, but is given some weight by the lack of additivity between the effects of alcohol and nicotine. Thus, participants receiving both substances did not show elevated emotional responses compared to those receiving only one, suggesting that priming was an all-or-none rather than a dose-dependent phenomenon. It would, however, be important to seek replication of this in future research, and explicitly to investigate whether variations in dose and timing affect the magnitude of any priming effects.

Overall, then, Study 2 suggests that in smokers, a low dose of alcohol can substitute for nicotine in enhancing affective reactivity. This might increase the appeal of alcohol during the early stages of a quit attempt. However, whilst drinking might be beneficial in normalising hedonic tone and emotional reactivity, it has been found elsewhere that alcohol cross-primes the urge to smoke (Hillemacher et al. 2006; Piasecki et al. 2008), cue-elicited craving (Burton and Tiffany 1997; Erblich et al. 2009), and the subjectively rewarding effects of smoking (Glautier et al. 1996; Zacny 1990). Alcohol consumption during abstinence is therefore a double-edged sword which seems on balance more likely to increase than decrease the risk of relapse to smoking. Elsewhere we have found that hedonic tone / affective responsiveness improves to normal levels within a few weeks of successful smoking cessation (Dawkins et al., 2009). It may therefore be of value in smoking cessation programmes to educate clients about the likelihood and time course of changes in affective state, and in this context to discuss the possible risks of alcohol consumption.

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Table 1: Demographic and smoking-related information for the two groups (study 1)

	Satiated Smokers (N = 17)	Abstaining Smokers (N = 15)	t/χ^2	p
Age				
Mean (SD)	22.18 (4.53)	22.27 (3.63)	-0.06	0.95
Range	18-32	19-30		
Sex Ratio (M:F)	5:12	6:9	0.40	0.53
FTND				
Mean (SD)	4.12 (2.06)	4.33 (2.80)	-0.25	0.80
Range	1-9	0-9		
CO level (ppm)				
Mean (SD)	16.38 (7.41)	2.77 (1.96)	7.05	0.00*
Range	10-40	1-9		
MPSS				
Mean (SD)	7.06 (5.96)	6.47 (6.14)	0.28	0.78
Range	0-18	0-13		

Figure 1: Mean affective ratings for the 2 groups for emotionally-toned and neutral film clips (study 1). Error bars are 1SE

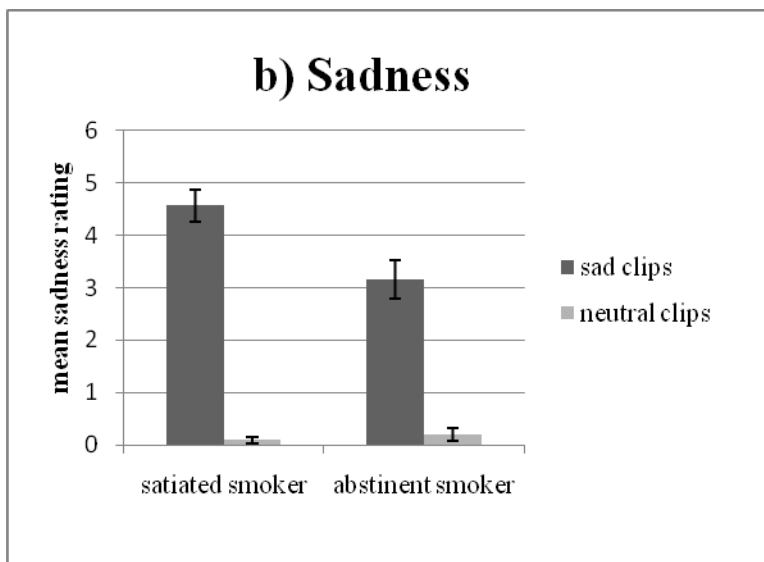
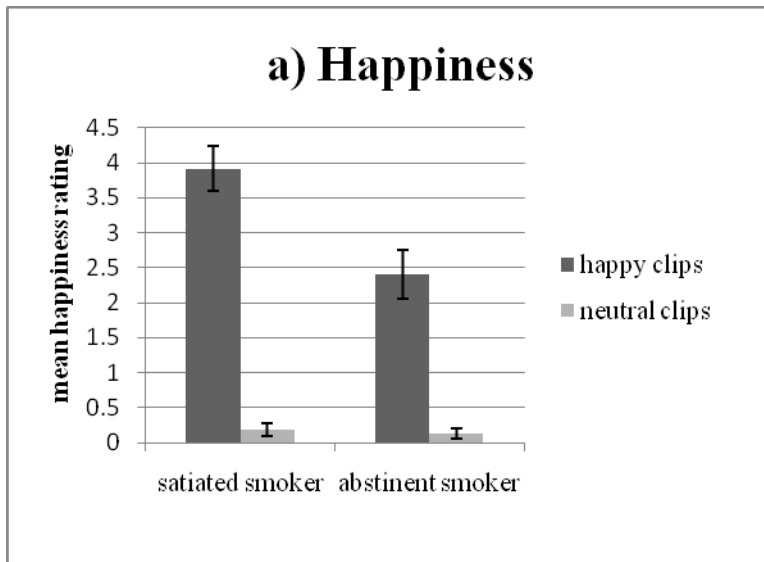


Table 2: Demographic and smoking related information for the four groups (study 2)

	Smoke/Alcohol	Smoke/Placebo	Abstinent/Alcohol	Abstinent/Placebo	F/ χ^2	p
Age						
Mean (SD)	29.20 (10.55)	29.22 (8.92)	31.50 (12.36)	26.32 (7.43)	0.87	0.46
Range	18-50	19-53	18-57	18-43		
Sex ratio (M:F)	8:12	5:13	4:16	9:10	3.90	0.27
FTND						
Mean (SD)	4.35 (2.81)	3.83 (2.33)	3.40 (2.52)	3.00 (1.76)	1.14	0.34
Range	0-8	0-7	0-7	1-6		
CO level (ppm)						
Mean (SD)	15.30 (4.94)	16.33 (5.56)	4.30 (2.66)	4.00 (2.49)	51.50	0.00*
Range	10-31	10-28	1-9	1-8		
MPSS						
Mean	3.55 (2.61)	4.33 (4.86)	5.80 (3.87)	6.32 (3.40)	2.28	0.08
Range	0-9	0-18	1-15	2-15		

Figure 2: Mean affective ratings for the four groups for emotionally-toned and neutral film clips (study 2). Error bars are 1SE

