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# Smoking and negative affect

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## Abstract

Approximately 25 percent of adults in the United Kingdom smoke despite awareness of the significant health risks. There is much debate about why individuals continue to smoke (e.g. appetitive effects of nicotine, habitual behaviour). Many addicted individuals describe coping with negative affect as key reason for continued drug-use. A review of the literature suggests that negative affect is an important feature of smoking behaviour. However, there is a lack of theory driven research that explores the underlying mechanisms which might moderate and maintain this relationship.

A recent model of addiction is able to account for many facets of the empirical evidence surrounding addictions and suggests some testable mechanisms which underlie dependence; namely, that negative affect will increase drug craving and bias selective attention towards drug-related cues (Baker, Piper, McCarthy, Majeskie and Fiore, 2004). Research was carried out to investigate whether negative affect in smokers would increase craving and attentional bias towards drug-related cues.

The present findings suggest that negative affect does increase craving and bias selective attention in favour of drug-cues. Furthermore, that biases in selective attention may occur specifically in the early stages of stimulus evaluation rather than in the maintenance of attention. There was some evidence of maintained attention to drug-related cues, however, this was related to smoking status (e.g. whether a smoker or non-smoker) rather than

mood state. These findings suggest that different underlying processes may mediate the relationship between attentional bias for drug-related cues, smoking status and negative mood states. Further research is required to clarify this issue.

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# Smoking motivation: the role of negative affect

Literature Review

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## Abstract

Attempts to understand addictions have focused upon motivational processes involved in drug use. There is a growing body of evidence suggesting that negative affect is an important feature of smoking behaviour. Current theoretical models of addiction are reviewed in this paper, with particular emphasis placed upon the predictions made regarding the role of negative affect in the maintenance of nicotine use. Several important key questions are considered; do smokers and non-smokers differ in measures of affect? Are smokers more vulnerable to affective distress? Does negative affect have the ability to directly cue drug use? What underlying mechanisms moderate the effect of negative affect upon drug use?

The evidence appears to suggest that compared to non-smokers, smokers experience higher rates of negative affect. Smoking also appears to be associated with several socioeconomic and environmental variables which may contribute to psychological distress. However, there is ambiguity surrounding the causal nature of these relationships. There is evidence that negative affect can directly prime craving and smoking. However, there is a lack of theory driven research which explores the underlying mechanisms which might moderate and maintain this relationship. Future research and clinical implications are put forward in light of these findings.

## Introduction

A large proportion of the population in the United Kingdom (approximately 25%) smoke cigarettes despite the potential health risks (Coulthard, Farrell, Singleton & Meltzer, 2002). It is estimated that over 13 people an hour die in the United Kingdom from cigarette smoking (Department of Health, 1998). Cigarette smoking is a complex behaviour that is highly reinforcing and can lead to compulsive use. Nicotine, which is thought to be the principle psychoactive agent in cigarette smoke, is highly addictive and many smokers experience unpleasant withdrawal symptoms, such as urges to smoke, irritability, and restlessness if they abstain from further nicotine use. In fact, individuals are commonly reported to smoke to avoid these unpleasant withdrawal symptoms rather than to enjoy the effects of the drug. Smoking can develop into a chronic problem for individuals. There is a high relapse rate (Hunt, Barnett & Branch, 1971) associated with smoking and substantial costs to society, as heavy use (more than twenty cigarettes per day) is associated with more frequent use of generic health services and specialist mental health services (Coulthard et al., 2002). Given these high costs why do individuals continue to smoke?

Attempts to understand addictions have focused upon motivational processes involved in drug-use. Research thus far has concentrated upon smoking as a learned behaviour. Numerous models have been put forward which will be reviewed later. Each model proposes different mechanisms about how this learning takes place and is reinforced. As yet there is not enough

evidence to confirm one absolute model of addiction, as each model is unable account for all the nuances of addictive behaviour (e.g. acquisition, maintenance). There is a need to develop a better understanding of the mechanisms underlying addictions, as this should help shape more effective smoking prevention initiatives and treatments for nicotine dependence.

There is a growing body of evidence suggesting that mood is an important feature of addictive behaviours (Kassel, Paronis & Stroud, 2003). Addicted individuals frequently report that affective states are the main antecedent to relapse (Marlatt & Gordon, 1985). Baker, Brandon & Chassin (2004a) suggest that although both positive and negative affect are influential in prompting drug-taking behaviour, negative affect is thought to have a more important role in motivating drug-use. Evidence exists which supports this assumption. For example, negative affective imagery has been associated with greater increases in craving compared to positive and neutral affective imagery (Tiffany & Drobes, 1990). Additionally, relapse can be predicted by negative reinforcement expectancies about drug-use (e.g. relief from negative affective states) but not by positive reinforcement expectancies (e.g. stimulation and pleasurable relaxation) (Baker et al., 2004a). Thus, this review will focus upon the role of negative affect in smoking. Negative affect is defined as a "general dimension of subjective distress and unpleasurable engagement that subsumes a variety of aversive mood states, including anger, contempt, disgust, guilt, fear and nervousness, with low negative affect being a state of calmness and serenity" (Watson, Clarke & Tellegen, 1988, pg 1063). Many studies within the

literature base refer to mood states (such as those just described) as stress. As stress and stressors will also be referred to within this paper it is helpful to define these. Stressors can be defined as "situations in which environmental demands tax the adaptive capacity of the individual" (Kassel et al., 2003, pg 273) and stress is a response (cognitive, behavioural, physiological & emotional) that occurs after exposure to such a stressor.

Shadel, Shiffman, Niaura, Nichter and Abrams (2000) suggest that, when reviewing the literature on smoking, it is important to distinguish between processes that govern initiation of use and those that lead to nicotine dependence. This paper will focus upon the maintenance and relapse stages of smoking, as these stages are particularly pertinent to the development of effective treatments for nicotine dependence.

This paper will consider current theories of addictions with specific reference to what these models say about negative affect and the effect it may have on the mechanisms maintaining addictions. A review of the empirical evidence will then be presented concerning the role of negative affect in smoking. Specifically, it will consider various questions, such as; do smokers and non-smokers differ in levels of negative affect? Does negative affect prime smoking and does smoking alleviate negative affect? The later part of this paper will consider both the clinical and research implications for the future study and treatment of addictions.

## Theoretical models

As previously stated there are a number of theoretical models of addiction. It is beyond the scope of this paper to review them all. This review will therefore focus upon current influential models of addiction, as well as older models that have influenced current thinking and those which have been influential in the treatment of addictions. Each model will be reviewed in terms of the proposed motivational mechanisms in operation. In this section, the extent to which each model discusses the role of negative affect will also be discussed.

### Withdrawal based models

Withdrawal based models of addiction pioneered by Wikler in the late 1940s have had a lasting influence in this field. These models propose that the key motive for drug-use is escape and avoidance of the aversive symptoms of drug withdrawal. Classical conditioning is proposed to occur through repeated use of drugs and addicts learn that withdrawal symptoms are rapidly reduced by further drug-use (Wikler, 1948).

Subsequent authors (Solomon,1977; Seigel, 1989) have emphasised the role of opponent processes in maintaining addiction. For example, according to this view if a drug has a positive effect on the body (e.g. euphoria), the body produces a negative affective process to cancel it out. Although, positive reinforcement of the direct drug effect occurs initially, negative affective states (involved in withdrawal) are predicted to develop in response to drug cues over

time. This negatively reinforces further drug-use, as the body has a drive to return to neutrality.

There is some supporting evidence for the role of withdrawal in motivating drug-use. Withdrawal symptoms have been associated with increased urge and intention to take drugs (Baker, Morse & Sherman, 1986; O'Brien, 1975; Wikler, 1980). However, withdrawal-based models are frequently criticised as they do not account for the data highlighting that individuals often relapse after withdrawal has subsided (Robinson & Berridge, 1993). Additionally, only certain drugs have a withdrawal syndrome and few individuals report the main precursor to relapse as being due to the physiological signs of withdrawal (McAuliffe, 1982).

A strength of these models, however, is that they do consider negative affect to be an important feature of drug addiction; albeit this is restricted within the context of drug withdrawal. Withdrawal models therefore posit that an individual uses drugs and then becomes sensitised to negative affect through the unpleasant symptoms of drug withdrawal. More recent evidence exists for cognitive and incentive models of addiction and these will be considered next.

## Cognitive models

### *Cognitive Social Learning Theory (Marlatt & Gordon, 1985)*

This model proposes that key factors in the decision to use drugs involve the expectations individuals have about drug-use, its consequences, and how

confident an individual is in his/her ability to cope when faced with drug cues that have triggered them into using drugs in the past. Situations which are "high risk" are proposed to be characterised by negative emotional states, interpersonal conflicts and social pressure. Support for this model exists as relapse has been associated with low self efficacy (Baer, Holt & Lichtenstein, 1986), expectancies (Jones, Corbin & Fromme, 2001; Julliano & Brandon, 2002), increased negative affect (Litt, Kadden, Cooney & Kabela, 2003) and poor coping skills (Shiffman, Patty, Guys, Kassel & Hickcox, 1996).

The model has often been criticised for being hierarchical (e.g. some factors, such as negative emotional states, are more important than others). However, recently Witkeiwitz and Marlatt (2004) have reformulated the model so that all factors have the ability to be equally influential. The extent to which each factor influences the process depends upon the individual and the particular dispositional factors regarding the context of past and current drug-use, experiences such as readiness to change, and risks and coping resources. These are all proposed to be key factors in the maintenance of drug-use (Witkeiwitz & Marlatt, 2004).

Niaura (2000) has also proposed an updated model. Niaura (2000) suggests drug cues may be influenced by affect and drug-use may occur via physiological activation (which he suggests is an appetitive motivational process), urges, outcome expectancies, self-efficacy and cognitive behavioural coping attributions.

Beck, Wright, Newman and Liese (1993) have also expanded upon Marlatt and Gordon's (1985) original ideas, suggesting that in these "high risk" situations both internal cues (e.g. boredom, anxiety, depression etc.) and external cues (e.g. peer group) can influence the decision about drug-use. Negative affect triggers beliefs about drug-use (e.g. "I can't stand discomfort, I need to smoke to make it go away") and, consequently, craving. These beliefs and subsequent drug-use over time generate a craving-using pattern. Once drug beliefs are activated, addicted individuals experience a "cognitive blockage" where attention becomes biased towards drug-associated stimuli above all other stimuli in the environment (Beck et al., 1993).

Cognitive social learning theories highlight that negative affect plays an influential role in cuing drug-use via expectancies about the ability of the substance to regulate affect (Marlatt & Gordon, 1985), via negative belief systems and attentional bias to drug cues (Beck et al. 1993) or via appetitive processes (Niaura, 2000). These models also predict that environmental factors contribute to "High Risk" situations and therefore cue drug-use.

Niaura (2000) and Beck et al. (1993) have proposed that some automatic processing may be involved in motivating drug-use. Another cognitive model places more emphasis upon the role of automatic processes involved in motivating drug-use. This model will be discussed next.

*Cognitive Processing Model (Tiffany, 1990)*

Drug addiction is proposed to be a learned habit (Tiffany, 1990). Tiffany postulates that, over time, skills involved in the acquisition and consumption of drugs are stored in memory as "drug-use action plans" (i.e. cognitive representations of skills). If drugs are readily available, drug-use, occurs outside of conscious awareness and requires little cognitive effort. However, if drug-use is interrupted, more effort and processing are required to overcome the barriers to use. Craving is seen not as a trigger for actual drug-use, but as an indicator of this process. Tiffany suggests that these drug-use action plans contain the necessary information for drug-use to take place. For example, the precise details of the stimulus conditions which trigger drug-use, both environmental (e.g. location) and internal (e.g. physical and emotional states), the physical skills and the problem-solving strategies to be implemented, if drug-use is impeded.

Tiffany suggests that negative affect may be one of many important factors in triggering action plans for drug-use. This would depend upon the circumstances in which each individual formed their habit. Individuals who frequently used drugs whilst in negative mood states would have this information stored within their "drug-use action plan". Tiffany states that negative affect does not promote drug-use directly by stimulating craving. Rather negative affect can increase drug-use by initiating an individual's drug-use action plan. This is in contrast to other theorists who believe that affective cues (Baker, Piper, McCarthy, Majeskie & Fiore, 2004a; Beck et al., 1993;

Niaura, 2000) and expectancies about the effects of drug-use on mood (Marlatt & Gordon, 1985) can directly elicit drug urges which then lead to drug-taking behaviour.

Tiffany's model has some empirical support (see Tiffany & Conklin, 2000, for review). It may, however, be difficult to separate and measure some of the abstract concepts (e.g. whether craving initiates drug-use action plans or whether craving is an index of this). The model has also been criticised for failing to account fully for the compulsive nature of drug-use (Robbinson & Berridge, 2003). Robbinson and Berridge (2003) state that "habit learning theories mistake automatic performance for motivational compulsion. However, habits are not intrinsically compulsive in any motivational sense, no matter how automatic they are." (Robbinson & Berridge, 2003, pg 33). Appetitive models propose alternative mechanisms for understanding the compulsive nature of drug-use and will be discussed next.

Appetitive models

Appetitive models of drug-use focus upon brain reward circuitry (Franken, 2003; Robinson & Berridge, 1993; 2000; 2001; 2003). Drugs are believed to alter the neurobiological systems in the brain involved in reward. This results in these systems becoming hypersensitive to specific drug effects and (through the process of classical conditioning) stimuli associated with drug-use. Robinson and Berridge (1993) refer to this process as incentive sensitisation, where cues associated with drug-use have greater incentive value

and result in drug "wanting". Drugs and associated cues "grab attention" and lead to a conscious desire for drugs and drug taking. Robinson and Berridge (1993; 2000; 2001; 2003) propose that drug "wanting" and "liking" result from different neural systems (e.g. an addict can want drugs even when he/she no longer likes using the substance). It is worth noting that other authors propose that these concepts of wanting and liking will co-vary and are governed by the same neural processes (Franken, 2003).

Although these appetitive models do not make direct predictions about negative affect, several factors are proposed to influence the neurobehavioural sensitisation and conditioning process. These include, for example, genetics, hormones, drug dose, and environmental and psychological factors such as learning and stress (Robinson & Berridge, 2000; 2001; 2003). These factors are proposed to greatly affect an individual's vulnerability to sensitization and the manifestation of it (Robinson & Berridge, 2000). Thus, it is possible that individuals vulnerable to negative affect may be more likely to become sensitised to drug-use and the development of addictions as according to Robinson and Berridge (2000; 2001; 2003) they are likely to be more vulnerable to the sensitisation process.

Recent evidence supports this appetitive view of drug cues and their role in the maintenance of addictions. Several studies have confirmed the presence of attentional biases (measured by reaction time and eye movement data on the visual probe task) for drug cues (Field, Mogg & Bradley, 2004; Mogg, Bradley, Field & De Houwer, 2003; Bradley, Mogg, Wright & Field, 2003; Waters,

Shiffman, Bradley & Mogg, 2003). There is also evidence that attentional biases for drug cues are associated with high levels of drug craving (Mogg et al. 2003), behavioural response tendencies to approach drug related stimuli (Mogg et al. 2003) and more unsuccessful attempts to abstain (Bradley et al. 2003).

The main criticism of the appetitive models of addiction is that they fail to account for the evidence suggesting that cognitive factors such as expectancies of a desired drug effect appear to moderate drug-taking behaviour (Cohen, McCarthy, Brown & Myers, 2002). Expectancies about negative affect reduction have been found to be related to the magnitude of dependence, severity of withdrawal symptoms, and treatment outcome (Copeland, Brandon & Quinn, 1995; Wetter et al., 1994). A recent model of drug motivation attempts to take account of this issue and this shall be discussed next.

Negative reinforcement model of negative affect

Baker et al. (2004b) have sought to reformulate negative reinforcement accounts of addictions and, in addition, draw from both cognitive and appetitive models. The primary motivation for drug-use is purported to be the avoidance and escape of negative affect (as opposed to negative physical sensations associated with drug withdrawal). Baker et al. (2004b) propose a number of underlying information processing systems involved in the production and maintenance of drug-taking behaviour. They hypothesise that, through repeated withdrawal/drug taking cycles, individuals become sensitive to negative affect, detecting this when physiological sensations associated with

nicotine use lessen in the body. When the level of negative affect is relatively mild (where drugs are readily available or drug-use is expected to take place), an individual is unconsciously motivated to escape or avoid it. This motivational state does not depend on conscious awareness of the signals of negative affect as drugs are readily available and drug-use is expected to take place.

However, when negative affect is high (e.g. when drugs are absent or there are significant stressors), individuals become more aware of their negative affective state. This is proposed to affect individuals in several ways. Biases in selective attention and response selection occur, for example, drug cues have increased incentive value and grab attention, whereas alternate (non-drug) reinforcers have decreased incentive value and attention is directed elsewhere. Such processes are proposed to lead to drug seeking-behaviour and drug-use. In addition, alternative (non-drug related) cues and potential coping strategies are ignored and given no reinforcement.

Baker et al. (2004b) suggest that particular drug-use decisions may also be influenced by other motivational factors, such as expectancies about drug-use and social pressures. However, these are not suggested to be fundamental motivational elements in decision-making about drug-use but rather modulate this process. Baker et al. (2004b) propose that, in addition to negative affect associated with withdrawal states, other everyday stressors also cue drug-use. For example, social stressors, work pressures and negative self-referential thoughts are all cited as potential triggers for negative affect and therefore drug-use.

The negative reinforcement model of negative affect is able to account for many facets of the empirical evidence surrounding addictions (e.g. information processing biases and cognitive factors) and suggests some testable mechanisms which underlie dependence (e.g. negative affect resulting in drug cues having inflated value).

Summary of theoretical models

Current theoretical models of addictions give rise to several similar predictions about the role of negative affect in maintaining drug-use. However, there are also some differences between these models, for example, in the degree to which they emphasise the importance of negative affect in motivating drug-taking behaviour. The majority of models view negative affect as one of several motivational factors which might vary in potency within each individual (Wikler; 1948; Marlatt & Gordon, 1985; Beck et al., 1993; Niaura, 2000; Witkeitwitz & Marlatt, 2004; Robinson & Berridge, 1993; 2000; 2001; 2003). However, Baker et al. (2004b) propose that, negative affect is the primary motivation for drug-use for all addicts.

With the exception of Tiffany (1990), there does appears to be a general consensus that negative affect (occasioned either by a stressor or through withdrawal) does have the ability to prime drug craving which subsequently leads to drug-use (Wikler; 1948; Marlatt & Gordon, 1985; Beck et al., 1993; Niaura, 2000; Witkeitwitz & Marlatt, 2004; Robinson & Berridge, 1993; 2000; 2001; 2003; Baker et al., 2004b). Several authors hypothesise that a range of

cognitive factors also have a motivational role, such as beliefs (Beck et al. 1993), expectancies (Baker et al., 2004b; Marlatt & Gordon, 1985; Niaura, 2000; Witkeitwitz & Marlatt, 2004), and problem solving and coping strategies (Baker et al., 2004b; Marlatt & Gordon, 1985; Niaura, 2000; Tiffany, 1990; Witkeitwitz & Marlatt, 2004). However, the degree to which these variables hold motivational potency differs within each model.

Despite consensus regarding a relationship between negative affect and drug-use, there is disparity between models regarding the development of this relationship. Some models may view the relationship as resulting from a general vulnerability to negative affect which increases one's risk of developing an addiction (Robinson & Berridge, 1993) whereas other models appear to view the process in reverse; repeated drug-use increases the frequency of heightened levels of negative affect (Wikler; 1948; Solomon, 1977; Seigel, 1989; Baker et al., 2004b).

There is a growing body of research investigating these theoretical similarities and differences within the smoking literature. The key questions appear to be whether smokers and non-smokers differ on measures of affect? If this relationship were to exist, what is the direction of this relationship? Does negative affect have the ability to directly cue drug-use? Are there specific variables (e.g. attentional processing of drug cues) which moderate this relationship? These questions will now be discussed through the presentation of the empirical evidence regarding the role of negative affect in nicotine dependence.

## Empirical Research

Do smokers differ from non-smokers in negative affect?

Several studies have investigated the association between smoking and specific mental health problems. In a recent survey of psychiatric morbidity among adults in the UK, individuals experiencing depression, phobias and obsessive-compulsive disorder were reported to be twice as likely to be smokers than non-smokers (Coulthard et al., 2002). Several other studies have found that smoking is more common in individuals meeting diagnostic criteria for anxiety (Hughes, Hatsukami, Mitchell & Dahlgren, 1986; John, Meyer, Rumpf, Hapke, 2004; Degenhardt & Hall, 2001), depression (Hughes et al., 1986; John et al., 2004; Degenhardt & Hall, 2001), social anxiety (Sonntag, Wittchen, Hofler, Kessler and Stein, 2000), anti-social personality disorder (Coulthard et al., 2002) and those experiencing probable psychosis (Coulthard et al., 2002, Glass, 1990; Hughes et al., 1986). In addition, smokers are more likely than non-smokers to have co-morbid diagnoses (Coulthard et al., 2002, Degenhardt & Hall, 2001).

Research also suggests that several individual differences may exist between smokers and non-smokers. In terms of personal characteristics smokers have been found to be higher in traits of neuroticism and psychoticism than non-smokers (Canals, Blade & Domenech, 1997; Gilbert & Gilbert, 1995; Pritchard & Kay, 1993; McChargue, Cohen, & Cook, 2004).

The evidence suggests that compared to non-smokers, smokers do experience higher rates of negative affect and moreover have more complex

mental health needs (Degenhardt & Hall, 2001). Perhaps the higher rate of negative affect is due to a general vulnerability to distress that precipitates the initiation of smoking behaviour and then subsequently the maintenance of use?

Do smokers experience more socioecconomic variables, which are associated with psychological distress than non-smokers?

There are also differences in the experience of several socioeconomic variables. Smokers are reported to experience more financial problems (Coulthard et al., 2002; Kassel et al., 2003), have a lower general household income (Coulthard et al., 2002), and have fewer educational attainments. (Coulthard et al., 2002)

In addition, smokers and non-smokers have been reported to experience differences in levels of social support which is a known moderator of psychological distress (Kassel et al., 2003). Smokers are more likely to perceive a severe lack of social support, have a primary support group of less than four people, and have had fewer social contacts in the previous week compared to non-smokers (Coulthard et al., 2002).

Smokers also appear to experience more stressful life events and daily stressors (Kassel et al. 2003). In fact, those who smoke more than twenty a day are more likely to have been previously homeless, and experienced separation and or divorce. They are also more likely to have been the victim of personal injury, illness, assault, violence in the home, and previous sexual abuse (Coulthard et al., 2002; Kassel et al., 2003).

Thus, in addition to experiencing greater levels of negative affect, smokers also appear to be exposed to many of the moderators of affective distress. However, it is unclear from the current literature as to the mechanisms that underlie these relationships. For example, are smokers more vulnerable to negative affect and therefore smoke to cope with this? Are socially disadvantaged people more likely to experience mental and physical health problems, and also more likely to smoke (e.g. because of poorer education or another unknown variable)?

What is the relationship between negative affect and amount of nicotine use within smokers?

The degree of stress one experiences may be positively linked to level of nicotine use (Billings & Moos, 1983). Evidence suggests that affective distress can predict the transition from experimental smoker into regular smoker (Hirschman, Leventhal & Glynn, 1984; Koval, Pederson, Mills, McGrady & Carvajal, 2000; Olrlando, Ellikson & Jinnett, 2001; Siqueira, Diab, Bodian & Rolnitzky, 2000). Therefore, increased stress may be associated with increased risk of becoming dependent on nicotine (Stein, Newcomb and Bentler, 1996). Research undertaken during wartime in Bosnia highlighted that, despite increased cost of cigarettes and having less money, individuals reported smoking more (Creson, Schmitz, & Arnoutovic, 1996). Another study exploring quit rates during pregnancy found that failure to quit co-varied with several indices of stress (Dejin-Karlsson et al., 1996).

The amount of nicotine use is also positively correlated with specific personality traits such as neuroticism, hostility, anger and dysphoria (Anda et al., 1990; Degenhardt and Hall, 2001) and with specific mental health disorders such as depression (Breslau, Kilbey & Andreski, 1991; 1994) anxiety (Dierker, Avenevoli, Merikangas, Flaherty & Stolar, 2001; Johnson et al., 2000) and externalising behaviours (Cornelius, Lynch, Martin, Cornelius & Clark, 2001). In fact, Breslau, Kilbey and Andreski (1993) found that smokers with a history of depression had a two fold increased risk of becoming nicotine dependent. Furthermore, co-morbidity of diagnoses within a group of smokers was positively associated with the daily amount of cigarettes smoked (e.g. heavy smokers experienced more co-morbidity than light smokers) (Coulthard et al., 2002). Smoking and nicotine dependence have also been associated with the subsequent development of mental health problems such as anxiety (Johnson et al. 2000; Orlando et al., 2001; Breslau et al., 1993) and depression episodes (Brown, Lewinsohn, Seeley & Wagner, 1996; Windle & Windle 2001).

It does appear that increased nicotine use is associated with greater affective distress. However, the directional nature of the relationship still remains unclear. Are there other variables which moderate this relationship? Studies that have examined the direct effect of nicotine on negative affect will be discussed in more detail later in this review. Firstly, laboratory studies investigating the direct effect of negative affect upon smoking will be reviewed.

Laboratory studies have used different measurement procedures (e.g. self-reported craving and drug liking, physiological markers, duration and

frequency of cigarette puffs, relapse back into use and factors affecting the success of smoking cessation treatments) to examine whether negative affect increases craving and drug taking behaviour? The limitations of such measures will be discussed after the presentation of the empirical evidence.

*Effects of negative affect upon regular daily smokers*

There are a number of studies which have directly manipulated affect and drug use in the laboratory. These studies have the benefit of controlling for extraneous variables such as retrospective recall and the type of affective stressor. Payne, Schare, Levis and Colletti (1991) used a learned helpless task to manipulate negative affect. Smokers in the negative affect condition reported greater desire to smoke than controls. This was evidenced by both a higher puff rate and a longer duration of puff when given the opportunity to smoke (Payne et al., 1991). Similar increases in cravings have been found using a comparable manipulation (Brandon, Wetter & Baker, 1996). Social stressors have also been used successfully to manipulate negative affect in smokers. Social anxiety ratings were also found to be associated with significantly greater urge to smoke (Juliano & Brandon, 2002; Niaura et al., 2002).

Negative affect was manipulated in both a group of deprived (twenty-four hour abstinence period) and non-deprived smokers with a social stressor. Craving was reported to have increased in both groups to the same degree. However, in non-deprived smokers, increased positive affect led to increased craving whereas decreased positive affect led to increased craving in the

abstaining group (Zinser, Baker, Sherman & Cannon, 1992). In another study, a musical mood induction procedure was used to induce either negative or elated mood in smokers. Induced negative affect was associated with increased craving (measured by the Questionnaire of Smoking Urges; Cox, Tiffany & Christen, 2001) and drug taking behaviour (measured by a progressive-ratio operant procedure) relative to an elated control condition (Willner & Jones, 1996). However, this was only evident in smokers who had been instructed to abstain from smoking for 24 hours. Their findings suggest that negative affect may have enhanced motivational importance in the context of withdrawal.

In another study, imagery scripts were used to manipulate affect in both deprived (six to twenty-four hours abstinence) and non-deprived smokers. In both groups, negative affective imagery scripts increased self-reported urge to smoke relative to positive and neutral scripts (Maude-Griffin & Tiffany, 1996). However this effect was more pronounced in the abstaining group (Maude-Griffin & Tiffany, 1996).

Negative affect does appear to be motivationally potent in its own right, for example in increasing craving; however, this effect appears more pronounced in abstaining individuals. The next section reviews the effect of negative affect upon individuals who make a decision to abstain.

*Effects of negative affect upon smokers attempting to quit.*

As hypothesised by Baker et al. (2004b) high levels of negative affect have been associated with increased likelihood of relapse (Glassman et al.,

1990; Hall, Munoz, Reus & Sees, 1993). In one study, smokers who called a quitting help line were asked to identify why and when they had smoked and/or relapsed. Seventy-five percent of the callers reported that negative affect precipitated their relapse (Shiffman, 1989). Furthermore, of those who relapsed, high levels of stress were associated with the highest probability of relapse (Shiffman, 1989). Using Marlatt and Gordon's (1979) categorisation of the determinants of relapse, Hodgins, Elguebaly & Armstrong (1995) found that over half of major relapses were reported to be due to negative emotional states and interpersonal conflicts. In addition, a significant proportion of minor lapses were categorised by social stressors, such as pressure from others to smoke (Hodgins et al., 1995). These findings lend support to the negative reinforcement model of negative affect (Baker et al., 2004b). However, other researchers have questioned the importance of negative affect and stress in triggering relapse (Tiffany, 1990), given the high incidence of relapse that does not seem to be triggered in this way.

Does smoking relieve negative affect?

In the early nineties, Brandon & Baker developed a scale to measure the subjective value of nicotine use for individuals. Four important themes emerged; reduction of negative affect, positive reinforcement and stimulation, negative consequences of smoking (physical withdrawal) and appetite and weight control (Brandon & Baker, 1991). Of these four themes, reduction in negative affect, distinguished daily smokers from other less addicted subtypes (Brandon &

Baker, 1991) suggesting that reduction of negative affect by smokers may be a key factor in the development of high levels of nicotine dependence. This kind of survey data is limited as it relies upon retrospective accounts of drug use where there may be inaccuracies in self-report.

Several studies have investigated the direct effect of smoking on measures of affect in the laboratory. Within this literature there have been conflicting findings in relation to nicotine's effects on both physiological and subjective reports of stress (Kalman, 2002). Some studies have found no effects of nicotine on physical and subjective indices of stress (Knott, Hart & Lusk-Mikkelsen, 1998). Other studies have reported that nicotine reduced negative affect (Balfour & Ridley, 2000; Gilbert, Dibb, Plath & Hiyane, 2000), yet another set of studies report that nicotine increases negative affect (e.g. increased dysphoria) (Rose, Behm, Westman & Johnson, 2000). There is also a group of studies within the literature that highlight disparity between self-reports of calmness after use and physiological indices which indicate increased physiological arousal (see Gilbert, 1979; Parrott, 1998 for summary). The ambiguous nature of these findings has triggered a number of studies aimed at exploring whether particular variables moderate nicotine's subjective effects.

Several studies have examined the relationship between nicotine dose and its impact upon affective variables. Again, there have been mixed findings of its effect. High nicotine dose has been associated with greater reductions in tension, and increases in subjective pleasantness (Ague, 1973) and in euphoric

sensations (Pomerleau & Pomerleau, 1992). However, increasing nicotine dose has also been found to produce greater sedation and dysphoria (Jones, Garrett & Griffiths, 1999), more unpleasant sensations (Gilbert, Meliska, Williams & Jenson, 1992), and fewer pleasant sensations (Gilbert et al., 1992).

A number of studies have investigated whether the stressors may mediate the effect of nicotine on affect. Hatch, Bierner & Fisher (1983) used a social stress manipulation to induce negative affect. Participants were given cigarettes which had either a high or low dose of nicotine to smoke. Self-reported anxiety did not reduce within either nicotine dose group. Gilbert, Estes and Welser (1997) used a noise-stress manipulation and also found no effect of smoking upon mood regardless of nicotine dose. In another study, where negative affect was manipulated via a musical mood induction procedure, smoking did not significantly alter mood state (Willner & Jones, 1996). However, another study reported that cigarettes high in nicotine led to smaller increases in self-reported anxiety after exposure to a stressful movie (Gilbert, Robinson, Chainberline & Spielberger, 1989). It is unclear why these studies have produced mixed results. Further research is required to clarify this issue.

Other variables have been suggested to mediate the relationship between smoking and affect. Several studies have found an association between reduction of negative affect and expectancies about ability of cigarettes to reduce negative affect. For example, anxious mood was induced in a group of smokers who then smoked either nicotine cigarettes or cigarettes which did not contain nicotine. Nicotine cigarettes produced greater subjective

reports of anxiety-reduction than the placebo cigarettes (Juliano & Brandon, 2002). However, there was no significant reduction in physical symptoms of withdrawal. Juliano and Brandon (2002) also manipulated instructional set (e.g. whether participants thought they were smoking nicotine cigarettes or not) and found that anxiety reduction was only reported among those participants who believed they were receiving nicotine cigarettes. This effect included those given instructions that the cigarette contained nicotine even when it did not. Thus, it was the expectations that the individual held about the cigarette that appeared to reduce anxiety. Expectations individuals hold about the ability of cigarettes to reduce negative affect has been found to be linked to drug-use (Cohen et al., 2002; Marlatt & Rohsenow, 1980), the magnitude of nicotine dependence, the severity of withdrawal symptoms, and treatment outcome (Copeland et al., 1995; Wetter et al., 1994); see Brandon, Juliano and Copeland (1999) for a review.

Another variable which is proposed to moderate the relationship between smoking and affect is distraction. Two studies have found that smoking reduced anxiety only when paired with a distracter (Kassel & Shiffman, 1997; Kassel & Unrod, 2000). The effects of nicotine dose, high and low trait anxiety and the presence of a distracter were examined in a group of smokers. The results showed that the greatest reduction in negative affect occurred within the group who were high in trait anxiety and who smoked the high nicotine cigarettes in the presence of a distracter (Kassel & Unrod, 2000).

The literature regarding the effect of smoking upon negative affect is ambiguous. One should be cautious about making inferences as the effect of several potential moderating variables requires further clarity (e.g. nicotine dose, type of stressor, distraction and expectancies). In addition, this section of the literature is particularly troubled by methodological complications. There are several important methodological factors which should be considered when making inferences from the literature concerning the relationship between negative affect and smoking. Before examining these in detail, it is first helpful to consider the effect of giving up smoking on negative affect?

What is the effect of quitting on negative affect?

Subjective stress and negative affect have been found to initially increase subsequent to nicotine cessation, but these are reported to diminish over time to levels lower than observed prior to quitting (Shiffman et al., 1997; Gilbert et al. 2000; West & Hajek, 1997; Hughes, 1992). Carey, Kalra, Carey, Halperin, Richards (1993) also found a significant reduction in self-rated distress in a group of individuals who successfully quit smoking. This effect has been shown to increase over time (Parrott, 1995). Furthermore smokers who cannot maintain abstinence generally continue to experience high levels of stress and negative affect over time (Covey, Glassman & Stetner, 90; Cohen & Lichtenstein 1990).

One study failed to find this stress-reduction effect after cessation (Gilbert et al., 1998). However this study assessed people one month after

quitting and allowed participants in the quit group to smoke up to 10 cigarettes within this period. Parrott (1995) and West and Hajek (1997) suggest that this time period may be too short to find an effect (previous studies report an initial increase of negative affect and then a reduction) and question whether it is wise to include subjects who lapsed in their sample. A lapse is likely to re-establish dependency and corresponding affective distress, leading to increased levels of stress (Parrott, 2000). In fact, in a prospective study which used palm top computers to assess antecedents to relapse, a lapse increased negative affect, whereas a temptation episode (urge with no use) did not (Shiffman et al., 1997). Furthermore, relative to temptation, a lapse resulted in significant reduction in self-efficacy and increased feelings of guilt and discouragement. This is possibly akin to what Marlatt and Gordon (1985) would term the abstinence-violation effect.

In summary, the research reviewed here suggests a complex relationship between smoking and negative affect. A number of studies have indicated a relationship between negative affect and smoking. However, our understanding of the underlying mechanisms which maintain this relationship is still vague. As yet, many of the mechanisms suggested in current theoretical models of addictions have not been empirically tested (e.g. whether negative affect inflates the incentive value of drug cues as proposed by Baker et al. (2004b), and thereby increases the likelihood of smoking behaviour). There is also a lack of detailed predictions regarding the effect of smoking on negative affect and how it might contribute to the maintenance of nicotine use. Clearly,

many addicted individuals believe that smoking reduces negative affect, but several theorists have debated whether smoking actually has stress-dampening effects (Parrot, 1999; Kassel, 2000; Kassel et al., 2003). Given the equivocal nature of some of the research findings, it is helpful to consider some methodological issues which seem important to take into account in future research.

## Methodological considerations

The following methodological factors will now be discussed: varying definitions of key variables, identifying sources of negative affect, choice of adequate control groups, measurement effects, effects of nicotine dosing and the effects of selective drop out rates.

### *Varying definitions of key variables*

Comparisons between studies are complicated by differing definitions of the key variables of interest (e.g. negative affect, dependence, abstinence). Examples of definitional problems might be how to define negative affect (e.g. should negative affect, anxiety and dysphoria be treated as equivalent); what is a "stressor" (e.g. is a stressful social situation similar to watching an aversive movie) and nicotine dependence (e.g. how do studies define dependence; by the nature of physical withdrawal symptoms, affective withdrawal symptoms or by a combination of both?).

*Identifying sources of negative affect*

Kassel et al. (2003) suggest that pharmacological effects of nicotine make it difficult to identify the sources of negative affect, as certain emotional states are characteristic of nicotine withdrawal. It, essentially, is very difficult to separate deprivation-based negative affect and stress-induced affect. There is debate as to whether this is in fact an important point. Baker et al. (2004b) suggest that there is a generalisation between negative affect associated with drug withdrawal and negative affect associated with exposure to stressors, both lead to craving and drug-taking behaviour. Baker et al. (2004b) posit that it is the negative affect per se, which is the primary motivational agent, not the stressor or withdrawal. However, other authors believe that it is theoretically helpful to differentiate these states (Kassel et al., 2003).

*Choice of adequate control groups*

Several studies have attempted to examine the effects of withdrawal and stressors on variables, such as negative affect and urge to smoke, by comparing various groups of nicotine users under differing conditions (e.g. deprived smokers, minimally deprived smokers, previous smokers, occasional smokers and non-smokers). Each comparison group has advantages and disadvantages (Kalman, 2002). Each group has the ability to control for additional confounding variables (e.g. pharmacological tolerance and the reinforcing effects of the actual smoking behaviour i.e. lighting a cigarette and raising it to ones mouth) (Kalman, 2002). The particular control group used is of

further methodological importance as the literature suggests that smokers and non-smokers differ on measures of negative affect. Individual differences in sensitivity to negative affect (e.g. e.g. trait anxiety, or neuroticism) are another potential confounding variable which also merits control. Another issue in studies using stress manipulation or mood induction procedures to manipulate negative affect is whether neutral mood or positive mood should be used as the main comparison condition for negative mood conditions. Baker et al. (2004b) suggest that neutral mood states are the optimum comparison condition, as positive affect, although proposed to be less important than negative affective states, do have a role in prompting drug-use and craving (Baker et al. 2004a).

*Measurement effects*

Measurement effects also limit research. Several different measures are used within studies to assess affect, dependency and craving. Firstly, there is considerable debate within the literature regarding concepts such as affect and craving. In addition, experimenter effects may bias many of these measures as they predominantly rely upon subjective self-reports and may be influenced by experimental expectancies (Julliano & Brandon, 2002). Additionally, baseline measures of affect (Sharpe & Gilbert, 1998) and withdrawal (McChargue & Collins, 1998) have been found to vary considerably within individuals at different points of time. There is the potential for repeated measurement, in the absence of a stable baseline, to confound the interpretation of an experimental effect (Gilbert & McClernon, 2000). Studies also vary in the precise nature of

the dependent variables and the measures used to test hypotheses. Thus, limiting the ability to make comparisons between studies.

*Effects of nicotine dosing*

There are also several methodological issues to consider in studies examining the effect of nicotine dose. For example, what doses should be given? What is the route of administration and who controls this? Nicotine dosing is a complex issue, as attempts to manage potential problems have resulted in subsequent confounding variables (Kalman, 2002). For example, the use of cigarettes low in nicotine have been criticised as individuals may actually take more frequent and larger puffs, and in effect, obtain similar doses to those given high-nicotine cigarettes (Kalman, 2002). However, controlling the dose via intravenous injection or nicotine patch administration also has its own problems. It may control for reinforcement effects; however, what is the ecological validity of this means of administration (Kalman, 2002)? In addition, individuals vary in their ability to metabolise nicotine in the body (Benowitz & Jacob, 1997; Gourlay & Benowitz, 1997; Rose, Behm, Westman & Coleman, 1999).

*Effect of selective drop out rates from treatment studies*

Within studies which investigate the relationship between relapse, smoking cessation, and negative affect, it is important to acknowledge that the results reported often do not show the whole picture. Treatment studies

frequently exclude those individuals who do not remain completely abstinent from the results that are published (Gilbert & McClernon, 2000). This selective drop out rate can be as high as 85 to 92 percent of the original sample (Gilbert & McClernon, 2000) and poses problems for the ecological validity of many of the reported effects as the evidence suggests that in everyday life the propensity to relapse is high, especially when experiencing negative affect (Glassman et al., 1990; Hall et al., 1993; Shiffman, 1989; Hodgins et al., 1995).

Summary of empirical evidence

In summary, despite the methodological limitations mentioned above, several studies indicate a relationship between smoking and negative affect. There is also an association between smoking and several socioeconomic and environmental variables which may contribute to psychological distress, such as poor social support, low socio-economic status and negative life events. However, there is still ambiguity surrounding the causal nature of these relationships.

The empirical evidence does, however, suggest that negative affect can directly prime craving and smoking (e.g. Payne et al., 1991; Brandon et al., 1996). Furthermore, it has been associated with relapse and unsuccessful attempts to quit (e.g. Glassman et al., 1990; Hall et al., 1993; Covey, 1999). However, there is a lack of research testing the underlying mechanisms proposed by various theoretical models (e.g. the role of negative affect in

increasing the incentive salience of drug cues, as suggested by Baker et al., 2004b).

## Moving research on

Within the smoking and affect literature there are many interesting areas for further research. Clarifying the nature of the relationship between smoking and negative affect may be beneficial in developing smoking treatment and prevention initiatives. For example, a prospective study on a cohort of children (an especially at risk group for smoking initiation), which includes a comprehensive assessment and long-term follow up of their personality characteristics, mental health state, exposure to socioeconomic variables, experience of stressful life events, social support networks, and nicotine use might shed some light on the factors which predict use and those which do not.

It would be helpful to carry out experimental studies to test predictions from recent models of smoking. For example, there are some conflicting theoretical assumptions regarding the degree to which negative affect is an important motivational factor. Baker et al. (2004b) suggest that negative affect is motivationally superior to other variables whereas other theorists have questioned its overall importance (e.g. Tiffany, 1990).

Specifically, it would be important to examine in what way might the level of negative affect influence the processing of drug cues? Baker et al. (2004b) hypothesise that high levels of negative affect will increase the incentive

salience of drug cues, which will make them more attention-grabbing, and increase the probability of drug-taking behaviour. Research in the field of addiction has already found that drug cues grab addicted individuals attention (Field et al., 2004; Mogg et al., 2003; Bradley et al., 2003; Waters et al., 2003). However, no study has so far tested the hypothesis that negative affect will increase the ability of drug cues to capture attention.

Clearly, the role of several variables, which may influence the relationship between smoking and negative affect, requires further scrutiny (e.g. the effect of distraction, the type of stressor, nicotine dose, beliefs and expectancies). Research needs to focus upon both the effect of each variable and, additionally the interaction between variables.

Empirically testing hypotheses from recent theories of smoking behaviour may aid the development of more effective treatments for nicotine dependence, as different variables may moderate drug-use. Addicted individuals may in fact need to employ different therapeutic techniques to combat drug-use at differing levels of affective distress. This review next highlights several clinical implications which merit consideration.

## Linking theory and research to practice

Smoking cessation treatments can broadly be defined as pharmacological and psychosocial in nature. The main pharmacological interventions for smoking

cessation are Nicotine Replacement Therapy (NRT) and Bupropion. NRT works by replacing nicotine from cigarettes by another means of administration (e.g. nicotine gum, nicotine patch) whilst gradually reducing the nicotine dose. Burpropion is an anti-depressant that is thought to work upon chemical pathways in the brain. Both NRT and Burpropion are effective treatments for nicotine use (National Institute of Clinical Excellence (NICE), 2002). There is some evidence that when used in combination their efficacy is further improved (NICE, 2002). In contrast with treatments for other substance use problems, such as alcohol abuse, psychosocial treatments for smokers are reported to be under utilised (First & Tasman, 2004). Possibly because of the high number of smokers and the limited number of professionals trained to assist individuals to intervene in this way. Psychosocial treatments typically include psycho-education of the health risks associated with smoking, counselling which utilises motivational enhancement techniques and the use of cognitive behavioural strategies such as self-monitoring, recognising triggers for drug-use, and relapse prevention strategies. Despite the limited access to this type of intervention psychosocial treatments are reported to be effective one-year post treatment and increase in efficacy when combined with pharmacological interventions such as NRT (First & Tasman, 2004). The treatment of choice for smoking cessation appears to be one which integrates both pharmacological and psychosocial components (First & Tasman, 2004).

Many of the treatments developed to date appear to focus upon preventing actual drug-use, but possibly do not adequately address the

underlying mechanisms, such as increased experience of affective distress and drug-use as a means of mood regulation in smokers. Perhaps treatments need to focus more upon assisting addicted individuals to regulate affective distress through other means than smoking (for example, cigarettes are presumed to reinforce smoking behaviour by the reduction of negative affect caused by withdrawal, so other means of dealing with withdrawal based negative affect seem likely to help the individual to abstain). The teaching of emotional regulation skills (e.g. mindfulness, cognitive and behavioural self-monitoring and distraction) would appear to be beneficial.

However, the timing of interventions may need to be planned carefully and validated through scientifically reliable means. A recent study evaluating the use of cognitive-behavioural therapy for depression and smoking cessation actually resulted in an increased risk of relapse (Kahler et al. 2002). This may be explained by the short follow-up period and the intensive nature of this treatment (eight two-hour sessions over six weeks). However, perhaps individuals need to have effective emotional coping skills in place before an intervention to quit smoking begins.

## Conclusions

The aims of this paper were to examine the growing body of literature indicating that negative affect is an important motivational factor in the maintenance of nicotine dependence. Current theories and models of addiction have been

reviewed in terms of the empirical evidence and clinical implications and further areas of research have been identified.

Much research so far has focused upon identifying the presence of a relationship between affective distress and the maintenance of nicotine use. Research should now centre upon exploring the underlying mechanisms which moderate and maintain this relationship. Enhanced understanding of the mechanisms underlying smoking will undoubtedly shape more effective smoking treatments and prevention initiatives which are extremely important given the high physical and psychological cost for individuals and the significant burden to society.

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UNIVERSITY OF SOUTHAMPTON

# Induced negative affect increases initial orienting towards smoking cues in smokers

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## Abstract

**Aims:** To investigate the effect of a negative mood induction procedure (MIP) on biases in orienting of attention to smoking-related cues in cigarette smokers.

**Design:** Smokers and non-smokers participated in two sessions (negative MIP, neutral MIP). Their eye movements and reaction times were recorded during a visual probe task, which presented smoking-related and non-smoking pictures for 2000ms. The task was performed after they had undergone either the negative or neutral MIP.

**Participants:** Twelve smokers and thirteen non-smokers completed both test sessions.

**Measurements:** Direction of initial shift in gaze and duration of gaze and response times to probes were measured whilst participants completed the visual probe task.

**Findings:** In smokers, induced negative mood significantly increased self-reported urge to smoke and was associated with increased initial orienting towards smoking cues, relative to a neutral mood condition (i.e. a significant effect of MIP on the direction of smokers' initial shift of gaze). There was no significant effect of the MIP on initial orienting to smoking cues in non-smokers. Measures of maintained attention were not significantly affected by induced negative mood (i.e. an absence of significant effect of MIP on duration of initial fixation and the RT bias scores assessing attentional processing of pictures at 2000 ms offset). However, there was some evidence of a bias in maintained

attention to smoking cues which was affected by smoking status (i.e. whether the participant was a smoker or non-smoker), rather than mood state.

**Conclusions:** These results suggest that induced negative mood increases drug craving and biases in initial orienting to smoking cues in smokers, but not in the maintenance of attention on smoking cues. The findings are discussed in terms of current theoretical models of drug motivation and affective processing.

## Introduction

Smoking kills in the region of 120,000 people in the United Kingdom a year (Department of Health, 1998). Approximately 25 percent of adults in the United Kingdom smoke despite awareness of the significant health risks (Coulthard, Farrell, Singleton & Meltzer, 2002). There is much debate about why individuals continue to smoke (e.g. appetitive effects of nicotine, habitual behaviour). Many addicted individuals describe coping with negative affect as key reason for continued drug-use (Wetter et al., 1994).

Baker, Piper, McCarthy, Majeskie and Fiore (2004) suggest a reformulated model of addiction motivation: namely, an affective processing model of negative reinforcement. This model predicts that avoidance and escape of negative affect is the primary motivation for drug-use. Thus, negative affect which is elicited by withdrawal states or by exposure to a stressor (e.g. interpersonal conflict, work pressures, negative self-referential thoughts) will increase drug cravings and drug-taking behaviours. Baker et al. (2004) propose a number of underlying information processing systems involved in the production and maintenance of drug-taking behaviour. When negative affect is high (e.g. when drugs are absent or when there are significant stressors), biases in selective attention and response selection occur. Drug cues are proposed to have increased incentive value, whereas alternate non-drug cues have decreased incentive value. Baker et al. (2004) suggest that such processes may operate outside of conscious awareness (e.g. are automatic and occur in the early states of stimulus processing) as negative affect may affect the hedonic evaluation of a cue (i.e. its perceived

ability to relieve distress). It is proposed that through repeated withdrawal/drug taking cycles, individuals learn introceptively that drug use rapidly reduces distress, thus, drug cues have increased salience and lead to drug seeking-behaviour and drug-use. According to incentive models, stimuli which have increased incentive salience are highly attractive, become wanted and will grab attention (Robinson & Berridge, 1993; 2000). Thus, according to Baker et al. (2004), negative affect should increase the capacity of drug cues to capture an addicted individual's attention. This appears an important theoretical issue as it may provide a rationale for greater emphasis to be placed upon affective elements in smoking cessation programmes, such as providing training in coping with negative mood states. What evidence exists for the predictions made by Baker et al. (2004)?

*Is negative affect an important issue in smoking?*

Research suggests that, within the smoking population, negative affect may be associated with higher rates of nicotine use (Breslau, Kilbey & Andreski, 1991; 1994; Dierker, Avenevoli, Merikangas, Flaherty & Stolar, 2001; Johnson et al., 2000), increased risk of becoming dependent upon nicotine (Hirschman, Leventhal & Glynn, 1984; Koval, Pederson, Mills, McGrady & Carvajal, 2000; Olrlando, Ellickson & Jinnett, 2001; Siqueira, Diab, Bodian & Rolnitzky, 2000) and increased likelihood of relapse (Glassman et al. 1990; Hall, Munoz, Reus & Sees, 1993; Shiffman, 1989).

As noted in a recent review by Baker et al. (2004), several studies have also highlighted that smoking is more commonly found in individuals

meeting criteria for several psychiatric disorders such as anxiety, depression, and psychosis (Hughes, Hatsukami, Mitchell & Dahlgren, 1986; John, Meyer, Rumpf, Hapke, 2004; Degenhardt & Hall, 2001; Coulthard et al., 2002, Glass, 1990). Smokers are additionally more likely to experience co-morbid diagnostic conditions (Coulthard et al., 2002, Degenhardt & Hall, 2001) and higher traits of neuroticism and psychoticism compared to non-smokers (Canals, Blade & Domenech, 1997; Gilbert & Gilbert, 1995; Pritchard & Kay, 1993; McChargue, Cohen, & Cook, 2004). However, a limitation of this research is that, although it points to an association between negative affect and smoking, it does not clarify the nature of the relationship.

*Can negative affect increase craving and prompt drug-use?*

There are a number of studies which have directly manipulated affect and drug use in the laboratory. Payne, Schare, Levis and Colletti (1991) used a learned helpless task to manipulate negative affect. Smokers in the negative affect condition reported greater desire to smoke than controls. This was evidenced by both a higher puff rate and a longer duration of puff when given the opportunity to smoke (Payne et al., 1991). Similar increases in cravings have been found using a comparable manipulation (Brandon, Wetter & Baker, 1996). Social stressors have also been used successfully to manipulate negative affect in smokers and, in studies using such stressors, social anxiety ratings were found to be associated with significantly greater urge to smoke (Juliano & Brandon, 2002; Niaura, Shadel, Britt & Abrams, 2002).

*Does negative affect increase attentional biases to drug cues?*

Baker et al. (2004) hypothesise that high levels of negative affect will increase the incentive salience of drug cues, which should in turn, make them more attention-grabbing, and increase the probability of drug-taking behaviour. Research in the field of addiction has already found that drug cues grab the attention of addicted individuals (e.g. Field, Mogg & Bradley, 2004a; Mogg, Bradley, Field & De Houwer, 2003; Bradley, Mogg, Wright & Field, 2003; Waters, Shiffman, Bradley & Mogg, 2003). No study has so far tested whether negative affect increases the ability of drug cues to capture attention.

Thus, this study tested whether negative affect increases selective attention to drug cues (i.e. whether smokers in a negative mood state demonstrate increased attentional bias to smoking-related cues relative to neutral cues). Several studies have used the visual probe task to investigate attentional biases to drug cues in addictions (Field et al., 2004a; Field, Mogg, Zetteler & Bradley, 2004b; Bradley et al., 2003). This task is favoured over other tasks (e.g. modified Stroop task) as it provides a more direct measure of where visual attention is deployed in the spatial array. In this task, participants are presented with two pictures (e.g. a smoking-related and control picture) concurrently, one on each side of the visual array. The pictures are presented for a specified time period after which a probe replaces the picture on one side of the visual display. Individuals are required to respond to the probe as quickly as possible by pressing a button.

Since individuals are typically faster to respond to probes which appear in an attended spatial location relative to an unattended location (Posner, Snyder & Davidson, 1980), the allocation of attention to the pictures can therefore be deduced from the response times (RTs) to the probes which replace them. The visual probe task can therefore be used to assess attentional bias to different types of pictures presented within the task.

La Berge (1995) suggests that there are separate processes involved in the allocation of attention. A distinction is proposed to exist between processes involved in the initial shifting of attention towards a stimulus and those involved in the maintenance of attention. Several studies have recorded eye movements whilst participants undergo the visual probe task (Field, Mogg & Bradley, 2005; Field et al., 2004a; Mogg et al., 2003). Eye movement measures have the advantage of indexing more sensitively the distinct aspects of attentional orienting (e.g. initial shifting versus maintenance of gaze). It offers an explicit measure of visual orienting, which is reported to be ecologically valid as individuals commonly look at stimuli which attract their attention (Jonides, 1981; Kowler, 1995). Research in the field of addiction suggests that biases in attention operate in the maintenance of attention (i.e. increased duration of gaze towards drug-related cues), with no consistent evidence existing for attentional processing of drug cues at a preconscious level or in early initial orienting towards drug-cues (see Field, Mogg & Bradley, in press, for review).

One aim of this present study was to explore the extent to which negative affect in smokers' influences different facets of attentional processing of smoking-related cues (e.g. the direction and duration of eye

movements towards smoking-related cues). It was hypothesised that smokers who have undergone a negative mood induction procedure (MIP) would show an enhanced bias for smoking cues in all components of attentional processes. That is, smokers would be more likely to direct their gaze towards and hold their gaze for longer on smoking related cues, in comparison with smokers who have undergone a neutral mood induction procedure. In addition, induced negative mood in smokers should be associated with a greater attentional bias for smoking cues as shown by the RT data from the visual probe task (i.e. faster RTs to probes replacing smoking pictures than control pictures). The study also included a control group of non-smokers as it was predicted that the effects of the negative mood on attentional bias for smoking cues would be specific to smokers. It was also predicted that the negative MIP would increase self-reported urge to smoke in smokers.

## Method

*Design*

The study employed a mixed design. There were two groups of participants within the study; smokers and non-smokers. Each participant attended two sessions where they underwent one of two mood induction procedures (negative and neutral mood conditions were counterbalanced between groups over the sessions). Each session consisted of two tasks, the attentional task for which there were three dependent variables (direction and duration of initial eye movement and reaction times to detect probes)

and the craving measures. The independent variables differed for each of these tasks and are therefore described in more detail below.

For the eye movement data from the attentional task, the study employed a mixed design. The between-groups independent variable was group (smokers; non-smokers). When the dependent variable was the bias in duration of initial eye movements (i.e. proportion of trials where participants looked first at the smoking picture, rather than the control picture), there was one within-subject variable of MIP (negative; neutral). When the dependent variable was the duration of initial fixation, there were two within-subject independent variables which were mood induction procedure: MIP (negative; neutral) and picture type (smoking; non-smoking).

For the reaction time data from the attentional task, the study employed a mixed design. The between-groups independent variable was group (smokers; non-smokers). The within-subject independent variable was mood induction procedure: MIP (negative; neutral). The dependent variable was the attentional bias score, which was difference in mean RTs to probes replacing smoking pictures versus mean RTs to probes replacing control pictures (see Bradley et al., 2003).

For the craving measures, the study employed a mixed design with group (smokers; non-smokers) as the between subjects independent variable and MIP (negative; neutral) and time (time one, time two, time three) as the within-subjects independent variables. The dependent variables were the Questionnaire of Smoking Urges- Brief form (QSU; Cox, Tiffany, & Christen, 2001) and a 0-10 rating scale of urge to smoke.

*Participants*

Students and staff at the University of Southampton were recruited via poster advertisements and an online experimental booking system. 38 participants were recruited in the study. Participants were screened for depression through the administration of the BDI-II (Beck, Steer & Brown, 1996). Those scoring above the cut off of 10 were excluded to avoid exposing dysphoric individuals to a depressed MIP (Bradley, Mogg & Lee, 1997). Eight participants' BDI-II scores were higher than the suggested cut off and were excluded from participating in the MIP and experimental analyses. There was also incomplete data for five participants. Two participants did not return to complete the second session and technical problems with the equipment meant that data was incomplete for a further three participants. Thus, 25 participants completed both sessions of the study.

There were 12 smokers (9 females, 3 males, with a mean age of 24.1 years) who were regular smokers and reported smoking at least five cigarettes per day (this cut off criteria has been used to determine inclusion into other studies e.g. Herbert, Foulds & Fife-Schaw, 2001; Lerman et al. 1996) and 13 non-smokers (11 females, 2 males, with a mean age of 25.2 years) who reported never having been regular smokers (e.g. smoked no more than five cigarettes in their life time). Additional selection criteria were that participants spoke fluent English and had visual acuity within the normal range (as per Mogg et al. 2003). These criteria were used to ensure participants could comprehend the task instructions and visualise the stimuli

used in the task. The study was conducted in accordance with the University of Southampton, School of Psychology Ethics Committee.

*Materials and equipment*

*Mood induction procedure*

The mood induction procedure consisted of both a musical and recall task. Prokofiev's 'Russia under the Mongol Yoke' played at half speed was used to induce a negative mood. This piece of music has successfully been used to induce negative mood sates (Clarke & Teasdale, 1985; Willner & Jones, 1996). Kraftwerk's 'Pocket Calculator' was used to induce a neutral mood. This piece of music has successfully been used to induce neutral mood sates (Clarke & Teasdale, 1985; Sutton, Teasdale & Broadbent, 1988; Bradley, Mogg & Lee, 1997). The recall task (described later) has been successfully used to manipulate negative and neutral mood states (Bradley et al. 1997).

Musical mood induction paradigms have been used successfully to manipulate negative affect in smokers (Willner & Jones, 1996). Combination mood induction procedures are cited by Westerman, Spies, Stahl & Hesse (1996) to be one of the most effective ways of inducing negative mood states.

*Experimental tasks: equipment*

The tasks were presented on a 333 MHz Pentium II PC, with 15" monitor, attached to a parallel-port, two-button response box and standard

keyboard. Participants horizontal eye movements were recorded while they completed the visual probe task using a computerized eye tracking system (Pan/Tilt optics system, Model 504, Applied Science Laboratories, Bedford, MA, USA), which uses infra-red beams directed at the eye. The eye movement software was run on a 333 MHz Pentium Celeron PC. The visual probe task was run using Inquisit software (Inquisit 1.33, 2002. Seatle, WA: Millisecond Software).

*Pictorial stimuli*

The pictorial stimuli consisted of 16 photographs of smoking scenes. Sixteen control pictures were matched as closely as possible for content with each smoking-related picture (e.g. presence of person, scene), but did not contain any smoking-related cues. The pictorial stimuli were similar to those used in previous research (e.g. Bradley et al., 2003; Healy, 2004, unpublished doctoral thesis). Eight additional pairs of photographs containing images of neutral household stimuli were also used (for use in practice and buffer trials).

*Questionnaires*

*Beck Depression Inventory-II (BDI-II) (Beck, Steer & Brown, 1996).*

The BDI-II is a 36-item commonly used depression scale. There is solid psychometric support for the BDI-II (e.g. Storch, Roberti & Roth, 2004). The

BDI-II was used to screen participants for dysphoria in order to avoid exposing at-risk participants to a negative mood induction procedure.

*Fagerström Test for Nicotine Dependence (FTND) (Heatherton, Kozlowski, Frecker & Fagerström, 1991)*

This six-item self-report scale was used to measure nicotine dependence. It is a revision of the Fagerström Tolerance Questionnaire (FTQ; Fagerström, 1978) and was designed to improve its reliability and validity. Heatherton et al. (1991) report good internal consistency (α=.61) and predictive validity with carbon monoxide levels, a biochemical measure of nicotine dependence ($r^2$=28.4).

*Questionnaire of Smoking Urges (QSU-Brief) (Cox, Tiffany & Christen, 2001)*

This is a ten-item version of the Questionnaire of Smoking Urges (QSU; Tiffany & Drobes, 1991) which measures urge to smoke. Cox et al. (2001) report good internal consistency ($\alpha$=.97) of this measure in the laboratory, across smokers at differing stages of drug use, and it is highly correlated ($r$=0.51, $p$<0.001) with the global craving score of the original 32-item measure. Cox et al. (2001) report two factors within the scale. Factor one reflects urge to smoke in the context of positive reinforcement whereas factor two represents urge to smoke in the context of negative reinforcement

from negative affect. This scale was used in this present study to measure urge to smoke at various points during each test session.

*Smoking habit and history questionnaire (see appendix 1)*

This is a seven-item unpublished questionnaire which was used to collect demographic and smoking history information (e.g. level of use, how long they have been smoking, previous attempts to quit, number of cigarettes in the last six hours).

*State Trait Anxiety Inventory Y1 (State) & Y2 (Trait) (STAI: Speilberger, Gorsuch, Lushene, Vagg & Jacobs, 1983)*

This is a well known 40-item self-report questionnaire measuring state and trait anxiety (20 items for each scale). Speilberger et al. (1983) report good internal consistency of the scale (male, trait and state anxiety respectively K-$R_{20}$=.90, .91, female, trait and state anxiety respectively K-$R_{20}$=.91, .93) in college students and good concurrent validity with the IPAT Anxiety Scale ($r$=.75). This measure was used to establish baseline trait and state anxiety ratings for smokers and non-smokers and to assess any session differences between state reports of anxiety.

*Rating scales assessing mood and urge to smoke*

Four rating scales were used to assess "Sad", "Happy", and "Anxious" mood states and the strength of participants "urge to smoke". The rating scales were used to check for variation in mood (e.g. in order to evaluate the MIP) and urge state during the experimental sessions. Each scale consisted of 11 numbers and contained 5 anchor points (i.e. 0 (not at all), 2 (slightly), 5 (moderately), 8 (strongly), 10 (extremely)). Participants were instructed to indicate "how you feel right now" (see appendix 2).

*Procedure*

Ethics approval was first obtained from the School of Psychology Ethics Committee (see appendix 3 for confirmation of ethical approval). Participants were asked not to smoke for one hour prior to each experimental session. This criterion was used to avoid ceiling (very high levels of craving) and flooring (satiation from high nicotine use) effects of deprivation upon craving and attentional bias (see Field et al. 2004a). Testing took place in a small, dimly lit room. A brief introduction was given before participants completed the depression screening questionnaire (BDI-II; Beck et al. 1996) and the smoking habit and history questionnaire. Participants who had a BDI-II of ten or less were invited to take part in the study. Once informed consent was provided (see appendix 4 for an example consent form) participants were allocated to the MIP condition (negative or neutral) in a counterbalanced order within each group (smokers, non-smokers). Visual acuity was

measured using a Snellen chart to check that visual acuity was within normal limits. A sample of expired carbon monoxide (CO) was then taken from participants. Participants then completed some questionnaires (STAI state and trait measures, QSU-brief and the rating scales of mood and craving) to assess baseline mood and urge to smoke prior to administration of the mood induction procedure. The experimental procedure consisted of two parts, the mood induction procedure and the visual probe task.

*Mood induction procedure:* Participants undergoing the negative MIP were instructed to get in to a miserable or sad mood by recalling unhappy memories from their past whilst listening to a sad piece of music (seven minute duration). Participants in the neutral condition were instructed to get into a neutral mood by recalling routine journeys from the past whilst listening to neutral music (seven minute duration). Immediately after the MIP, participants were asked to complete the rating scales and QSU-brief about how they were feeling "right now".

*Visual probe task:* Participants sat at a desk approximately one meter away from the monitor. The eye tracking sensors were situated on the desk in front of the participant, below the right eye. The equipment was calibrated by displaying the numbers one to nine on the screen in a three by three array (with number one at the top left of the screen and nine at the bottom right). The direction of participants' gaze was recorded whilst asking them to look at each number in turn.

In the visual probe task, each trial began with a central fixation cross shown for 1000 ms, which was replaced by a pair of pictures (smoking-related or control), side by side, for 2000 ms. Immediately after the offset of the picture pair, a probe was presented in the position of one of the preceding pictures, until the participant gave a manual response. The probe was a small arrow pointing up or down. Participants were instructed to press one of two response buttons to indicate the identity of the probe. Participants were instructed to look at the fixation cross at the start of each trial. Eye movement data were recorded during each trial, starting immediately before the onset of the fixation cross and terminating immediately after the participant had made a response.

The task consisted of 8 practice, 2 buffer, and 128 critical trials. During the critical trials, each of the 16 smoking-control picture pairs was presented 8 times. Each smoking-related picture appeared four times on the left side and four times on the right side of the screen. The size of each picture was125 mm wide and each picture was 100 mm high. The distance between the pictures was 60 mm. The probe appeared in the location of either the smoking-related or smoking-control picture with equal frequency and there were an equal number of trials with each probe type. Trials were presented in a new random order for each participant. Immediately after completing the visual probe task participants completed the rating scales (to check that there was no adverse effect of the MIP prior to the session end), QSU-brief and FTND. Participants who had undergone the negative MIP were offered the opportunity to undergo a positive MIP (listening to cheerful

music). The experimenter then arranged the time and date of the second session.

The second session was designed to be as close in content as the first session in order to control for session effects. A sample of expired CO was taken from participants. Participants then completed some questionnaires (STAI state measures, QSU-brief and rating scales of mood and craving) to assess baseline mood and urge to smoke prior to administration of the MIP. Participants underwent the alternate MIP from the first session (as per counterbalanced order) and then completed the visual probe task as per session one. At the end of the second session participants were debriefed and either awarded course credits or ten pounds sterling for their participation.

*Preparation of eye movement data*

The Eyenal Data Analyses Program (Applied Science Laboratories, Bedford, MA, USA) was used to analyses the data. The direction of gaze, measured in degrees, was measured once every 17 ms. A fixation was defined as a stable eye movement (i.e. gaze remained within one visual degree for 100ms or more) to a particular position on the screen. This allowed the direction and duration of the fixation to be measured. Fixations were classed as being directed at the left or the right pictures if they were more than one degree wide of the central fixation position (the position which had been occupied by the fixation cross before the picture onset) on the horizontal plane.

Eye movement (EM) data were analysed for critical trials (e.g. where smoking-related and control pictures were presented). Initial fixations were calculated as long as the following three criteria were met: (a) participants were fixated on the central region before picture onset, (b) the initial eye movement occurred at least 100 ms after the picture onset (according to Fischer and Webster (1983) fixations made before this time are likely to reflect anticipatory eye movements), and (c) the fixation was towards a picture location as opposed to remaining in the central location.

## Results

### *Mood and smoking measures*

*Group characteristics:* The two groups (smoker; non-smokers) did not differ in age, $t(23)= 0.31$, $p>.05$, trait anxiety, $t(23)= 0.04$, $p>.05$, state anxiety at the start of the negative MIP session, $t(23)= 0.60$, $p>.05$, or state anxiety at the start of the neutral MIP session, $t(22)= 2.16$, $p>.05$. Smokers scored higher on the BDI-II than non-smokers, $t(23)= 2.36$, $p<.05$. See Table 1 for means.

Table 1

Group Characteristics

| | Smokers (n=12) | | Non-smokers (n=13) | |
|---|---|---|---|---|
| | M | SD | M | SD |
| Age | 25.17 | 7.171 | 24.08 | 10.15 |
| Trait anxiety | 36.5 | 6.26 | 33.31 | 7.95 |
| State anxiety | | | | |
| Pre neg MIP | 30.75 | 6.23 | 32.23 | 6.15 |
| Pre neu MIP | 29.58 | 11.69 | 30.5 | 8.88 |
| Expired CO | | | | |
| Pre neg MIP | 14.17 | 8.8 | 1.92 | 1.98 |
| Pre neu MIP | 14.83 | 11.64 | .92 | .29 |
| BDI-II | 6.58 | 2.78 | 3.62 | 3.46 |

A mixed design analysis of variance (ANOVA) of CO scores was carried out with group (smoker, non-smoker) and session (negative MIP, neutral MIP) as independent variables. A significant main effect of group was found, $F(1,22) = 20.28$, $p<.01$. Smokers had higher CO scores than non-smokers across both sessions (see Table 1). There were no other significant results. CO and rating scale measures were missing for one participant from the neutral MIP session. See Table 2 for summary of smoking measures for smokers (e.g. daily cigarette intake, FTND scores, etc.).

Table 2

Smokers Mean Scores on Measures of Smoking

| | *M* | *SD* | *Min/Max* |
|---|---|---|---|
| QSU-brief (assessed prior to MIP) | 26.03 | 10.29 | 10/42 |
| Urge to smoke ratings (assessed prior to MIP) | 2.87 | 2.18 | 0/8 |
| Daily cigarette intake | 12.67 | 4.22 | 5/20 |
| Years smoked | 8.07 | 6.49 | 1/23 |
| Quit attempts | 2.07 | 1.94 | 0/7 |
| FTND | 2.50 | 1.24 | 1/6 |

n =12

*Effect of MIP on mood:* A 2 x 2 x 3 mixed design ANOVA was carried out on each rating scale measure of mood to assess the effectiveness of the MIP on mood state at three points during the experimental session, with group (smoker, non-smoker), MIP (negative, neutral) and time (Time 1: pre MIP, Time 2: = post MIP, Time 3: after visual probe task) as independent variables. For sad mood, there were significant main effects of MIP ($F(1,22) = 48.71$, $p < .01$) and time ($F(1, 22) = 24.94$, $p < .01$), and a significant MIP x time interaction ($F(1, 22) = 42.04$, $p<.01$). This interaction was not affected by group ($F<1$). Post hoc contrasts showed that sadness ratings (averaged across groups) were higher following the negative than neutral MIP (Time 2: $t(23) = 7.49$, $p<.01$; Time 3: $t(23) = 3.44$, $p<.05$) (see Figure 1).

A parallel interaction effect of MIP x time occurred on happy mood ratings ($F(2, 22) = 8.11$, $p<.05$) which was not influenced by group ($F<1$).

Happy ratings (averaged across groups) were significantly lower after the negative compared to the neutral MIP (Time 2: $t(23) = 3.56$, $p<.05$), but did not significantly differ at other times (See Figure 2).

A significant group x time interaction ($F(2,22) = 4.27$, $p<.05$ ) was found for anxious mood ratings. This was not significantly affected by the MIP ($F<1$). Post hoc contrasts showed a significant main effect of time on anxious mood ratings in non-smokers, as anxiety ratings tended to reduce during the session ($F(2,22) = 3.24$, $p<.05$; see Figure 3), whereas the effect of time on anxiety ratings was not significant in smokers.

In conclusion, the mood manipulation was effective as participants reported more sadness and reduced happiness after the negative MIP compared to the neutral MIP. The significant differences in sad mood were maintained over the visual probe task. The effectiveness of the MIP did not differ significantly between the smoking and non-smoking groups.

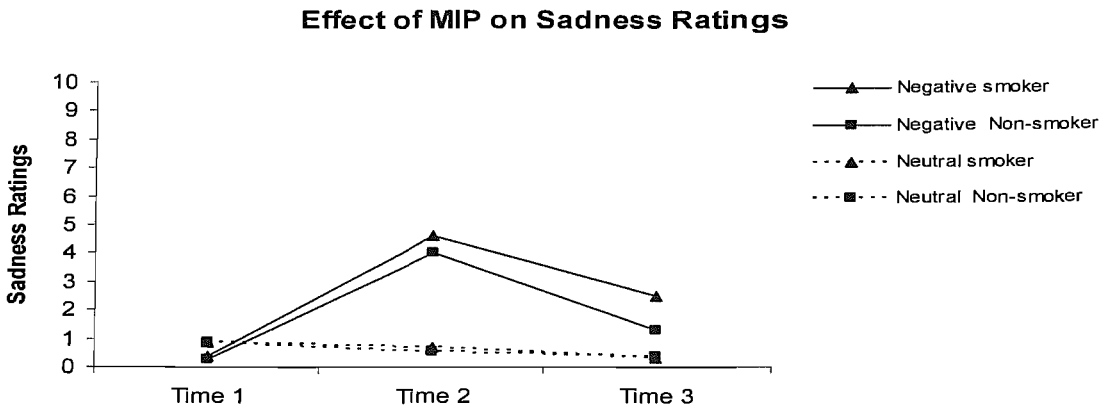


Figure 1: Mean ratings of sad mood for group and MIP conditions (Time1 = pre MIP; Time 2 = after MIP; Time 3 = after visual probe task).

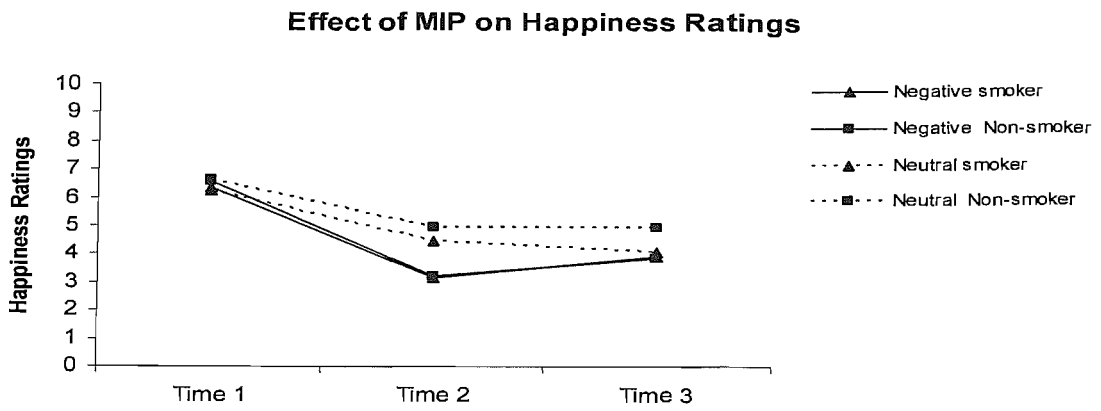


Figure 2: Mean ratings of happy mood for group and MIP conditions (Time1 = pre MIP; Time 2 = after MIP; Time 3 = after visual probe task).

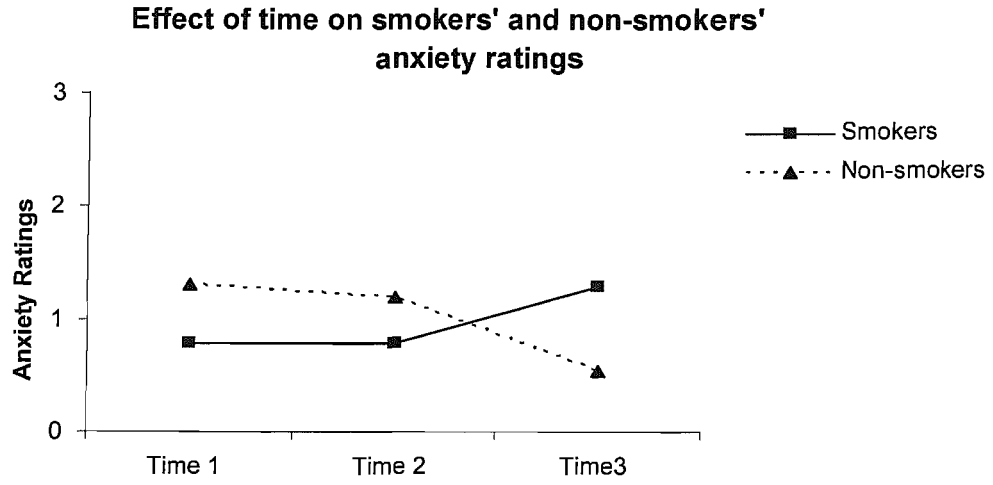


Figure 3: Smokers' and non-smokers mean ratings of anxious mood (Time1 = pre MIP; Time 2 = after MIP; Time 3 = after visual probe task).

*Post-experimental mood check.* There were no significant differences between the negative and neutral MIP group's final mood rating measures which were taken after the last batch of questionnaires in each session, suggesting that there were no lasting effects of the MIP.

*Effect of MIP on urge to smoke:* QSU-brief and urge to smoke ratings were rated zero by non-smokers throughout both MIP sessions, so were not included in the analyses. QSU-brief urge ratings from smokers were entered into a 2 x 3 ANOVA with MIP (negative, neutral) and Time (1-3) as independent variables. There was a significant effect of Time ($F(2,10) = 8.12$, $p<.05$) and a significant MIP x Time interaction ($F(2,10) = 7.36$, $p<.05$). Post hoc contrasts comparing the two MIP conditions showed that there was no significant difference in urge to smoke before the MIP (Time 1: $t(11) = 0.64$, $p>.05$) and at the end of the session (Time 3: $t(11) = 1.71$, $p>.05$). However, smokers' urge ratings were significantly higher immediately after the negative MIP than the neutral MIP (Time 2: $t(11) = 7.46$, $p<.01$); see Figure 4.

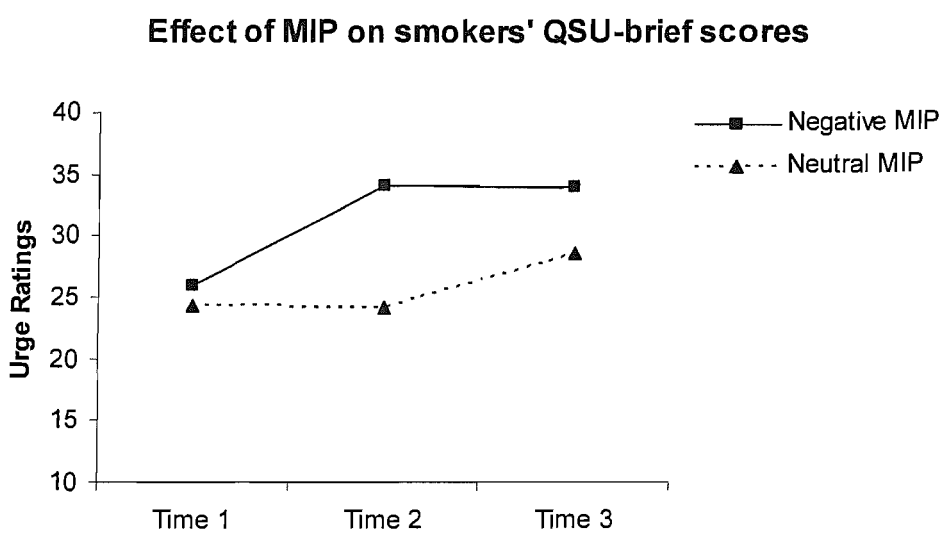


Figure 4: Smokers mean QSU-brief scores for MIP conditions (Time1 = pre MIP; Time 2 = after MIP; Time 3 = after visual probe task).

Urge to smoke ratings (assessed by the 0-10 rating scale) were entered in to a 2 x 3 ANOVA, with MIP (negative, neutral) and Time (1-3) as

independent variables. This showed only a significant main effect of Time ($F(2, 10) = 5.3$, $p<.05$), which indicated that urge tended to increase during the course of the session (see Figure 5). Post hoc contrasts showed that urge ratings were significantly higher at Time 3, relative to Time 1 ($p<.05$). The effect of Time was not significantly influenced by the MIP ($F(2,22) = 1.48$ , $p= .24$ ).

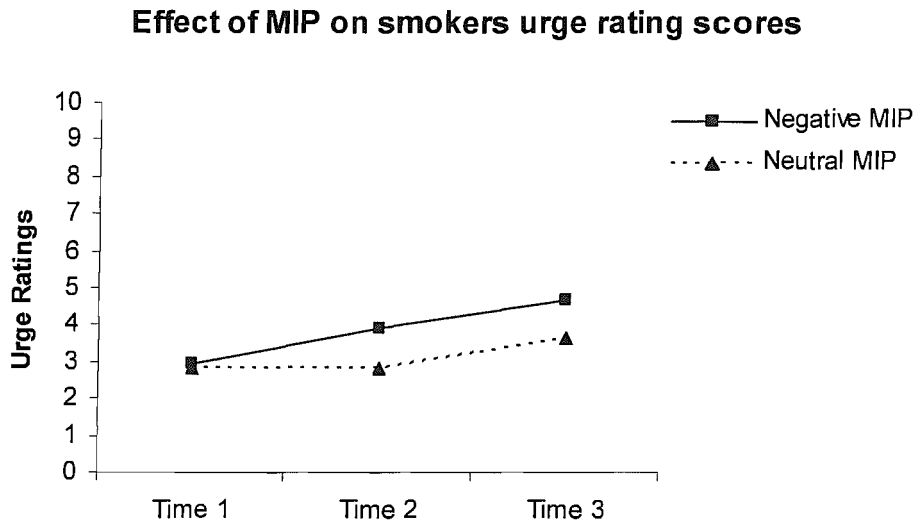


Figure 5: Smokers mean urge rating scores (as measure 0-10 rating scale) for MIP conditions (Time1 = pre MIP; Time 2 = after MIP; Time 3 = after visual probe task).

*Eye movement data: Direction of initial shift in gaze.*

Direction bias scores were calculated for each participant by expressing the number of trials when the EM was directed initially towards the smoking-related pictures as a proportion of the total number of trials in which an EM was made to smoking-related or control pictures (as described by Field et al. 2004). A direction bias value which is greater than 50% reflects a bias in orienting towards smoking-related pictures (50% denotes

no bias). EM-direction bias scores were entered into a 2 x 2 mixed design ANOVA, with group (smoker, non-smoker) and MIP (negative, neutral) as independent variables. There was a significant interaction of group x MIP ($F(1,22)= 5.92$, $p<.05$). There were no other significant results.

Post hoc contrasts were carried out to examine the group x MIP interaction. Bias scores were compared for the MIP conditions separately for each group. There was a significant effect of MIP on EM-direction bias scores in smokers ($t(11) = 2.27$, $p<.05$). Smokers showed greater initial orienting towards smoking cues in the negative than neutral MIP condition (see Figure 6). Smoker's bias scores were compared to a value of 50% (indicating no bias). A significant orienting bias was found for smoking cues after the negative MIP (55.5%; $t(11) = 2.71$, $p<.05$), but not after the neutral MIP (47.3%; $t(11) = 0.90$, $p>.05$). There was no significant effect of the MIP on bias scores for non-smokers ($t(11) = 0.91$, $p>.05$). The direction bias score of non-smokers was averaged across MIP sessions (53.2%) and was significantly greater than 50% ($t(11) = 4.63$, $p<.05$).

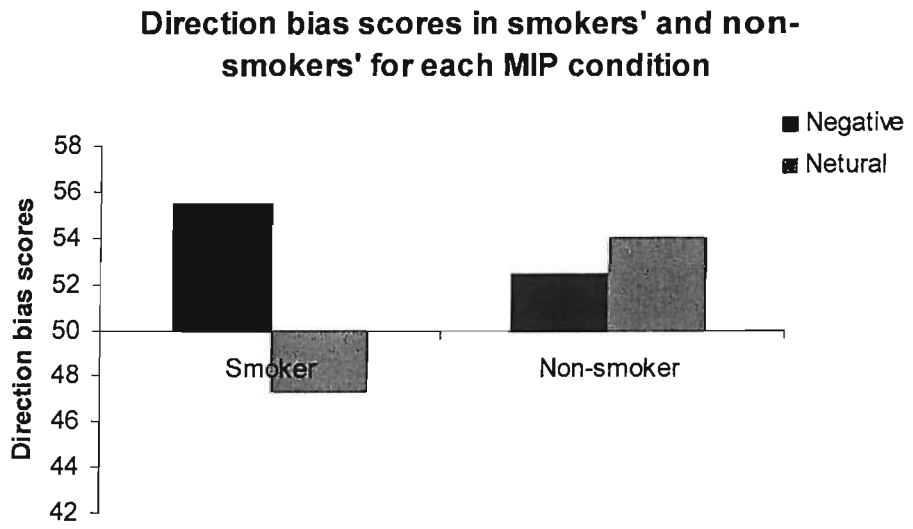


Figure 6: Smokers' and non-smokers' direction bias scores.

The earlier analyses of the mood data had shown that the smokers had higher depression scores than the non-smokers. Thus, the groups were matched on BDI-II scores to check that this was not confounding the comparison of smokers and non-smokers. Three non-smokers who had BDI-II scores of zero were omitted from the analyses (all smokers scored above zero on the BDI-II). The groups now no longer differed significantly in BDI-II scores ($t(19) = 1.52$, $p>.05$). The analyses were re-run, however, the results remained unchanged (i.e. a significant group x MIP interaction ($F(1,19) = 4.68$, $p<.05$).

*Eye movement data: Duration of first fixation*

EM duration data was entered into a 2 x 2 x 2 mixed design ANOVA, with group (smoker, non-smoker) as the between-subject variable, and MIP (negative, neutral) and picture type (smoking picture, control picture) as within-subject variables. Two participants were lost from these analyses due to missing data. There was a significant main effect of picture type ($F(1,20) = 10.04$, $p<.01$) indicating that participants generally looked at smoking pictures longer than control pictures (498ms vs. 443ms). There were no other significant results (e.g. group x MIP x picture type, $F(1,20) = 2.12$, $p=.16$).

Further analyses were carried out after matching the group on BDI-II scores. As described earlier, three non-smokers were omitted from the analyses. The pattern of results from the EM-duration data remained

unchanged (i.e. a significant effect of picture type on the duration of initial fixation, ($F(1,17) = 10.22$, $p<.05$, and no other significant results).

*Manual RT data: attentional bias at 2000 ms*

Data were excluded from filler trials, and from trials with errors. On critical trials RTs which were less than 200 ms, more than 1000 ms, and then more than 3 *sd* above each person's mean RT were excluded as outliers. Two participants (one smoker and one non-smoker) were excluded as they had excessive missing data due to error and outliers (>15%). Error and outlier rates for the remaining participants were 1.5% and 1.7%, respectively.

RT bias scores were calculated for each participant, by subtracting the mean RT when the probe replaced the smoking cue from the mean RT when the probe replaced the control cue (see Bradley et al. 2003). A positive value reflects an attentional bias to smoking cues. RT bias scores were entered into a 2 x 2 mixed design ANOVA, with group (smoker, non-smoker) and MIP (negative, neutral) as independent variables. There was a significant main effect of group on RT bias scores ($F(1,22) = 5.84$, $p<.05$). Smokers showed a greater bias for smoking cues compared with non-smokers (12.7 ms vs. -13.8 ms). However, this difference in RT bias between groups was not significantly affected by the MIP (i.e. MIP x Group: $F<1$).

After excluding the three non-smokers with BDI-II scores of zero (as described earlier), the pattern of results did not change; i.e. a significant main effect of group on RT bias scores ($F(1,19) = 5.90$, $p<.05$), with smokers

showing a greater attentional bias for smoking cues than non-smokers (12.7 vs. -17.2 ms).

## Discussion

The results of this study highlight that the MIP was effective in manipulating negative mood (evidenced by increased ratings of sadness in the negative mood condition). In line with Baker et al.'s (2004) prediction, induced negative affect significantly increased self-reported urge to smoke, when assessed by the QSU-brief. However, this was not evident when assessed using the 0-10 rating scale of urge to smoke.

The eye movement monitoring showed that induced negative affect in smokers was associated with increased initial orienting towards smoking cues, relative to the neutral mood condition (i.e. a significant effect of MIP on the direction of smokers' initial shift of gaze). There were no significant effects of mood on the initial orienting responses of non-smokers (i.e. no significant effect of the MIP on non-smokers' initial shift of gaze). Measures of maintained attention were not significantly affected by induced negative mood. That is, there was no significant effect of the MIP on the duration of initial fixations, or on the RT bias scores which assessed attentional processing of pictures (at 2000 ms offset).

However, as previously demonstrated in addiction research there was some evidence of a bias in maintained attention to smoking cues which was affected by smoking status (e.g. whether a smoker or non-smoker) rather than mood state. Thus, smokers had relatively quicker latencies to respond

to probes that replaced smoking cues than control cues (at 2000 ms) compared with non-smokers.

The main aims of this present study were to investigate whether negative affect increases attentional processing of smoking cues in smokers, and whether this would be apparent in different aspects of visual orienting (e.g. initial shifts vs. duration of gaze). These results provide preliminary support for Baker et al.'s (2004) prediction that negative affect influences very early stages of processing of drug-cues (indicating preconscious processes), as negative affect increased initial orienting of gaze in smokers relative to neutral mood. However, interestingly, negative mood did not significantly influence biases in the maintenance of attention (e.g. negative mood did not result in smokers' attention being held for longer on drug cues than non-drug cues). Baker et al. (2004) appear to suggest that controlled processing, although important, has less motivational impetus for drug use: "affective information is afforded priority in the stimulus evaluation or information processing cascade..... later stages of information processing incorporate non-affective information into processing and this may blunt or dilute the impact of the affective signal" (pg 39). Thus, they propose that the effects of negative affect on processing drug cues may occur relatively automatically and may not be primarily mediated by strategic processes. This may be one possible explanation for these present findings. However, it would be helpful for this issue to be clarified (i.e. what are the predictions regarding the role of negative affect in the maintenance of attention towards drug cues).

Again, in line with Baker et al.'s (2004) model, negative affect increased subjective reports of drug craving. However, as previously stated, this effect did not generalise across both measures of urge to smoke. One possible explanation for this could be that the QSU-brief is a more sensitive measure of craving than the 0-10 rating scale. It contains a greater number of items which have been psychometrically validated and shown to be a reliable index of urge state (Cox et al., 2001). Additionally, items on the QSU-brief tap a range of craving states; both desire to smoke and whether this desire is in the context of positive or negative reinforcement (Cox et al., 2001).

The results also demonstrated previously found attentional biases associated with smoker's status (i.e. smoking showed relatively faster reaction times to probes replacing smoking cues than non-smoking cues). Given that the probes appeared at picture offset, which was 200 ms after the pictures initially appeared (so there was considerable opportunity for attention to shift between the pictures); these results seem consistent with a bias in the maintenance of attention. These results suggest that smoking status and negative affect may influence the attentional processing of drug cues via different underlying mechanisms.

Interestingly, research into attentional biases in anxiety show a similar trend in results; biases are found in visual orienting (e.g. biases in initial shifts of gaze toward threat information) (Mogg, Millar & Bradley, 2000; Bradley, Mogg, White, Groom & de Bono, 1999). Further study of this issue has suggested that anxious individuals show a vigilance-avoidance pattern of attentional processing for threat stimuli (Hermans, Vansteenwegen &

Eelen, 1999; Mogg, Bradley, Miles & Dixon, 2004; Rohner, 2002). That is, they initially orient their attention towards salient stimuli, however then disengage attention away from these stimuli. This disengagement has been hypothesised to be due to an individuals desire to reduce subjective discomfort (Mogg & Bradley, 1998). Given that the current results suggest that negative affect in smokers is related to a bias in early orienting to motivationally salient stimuli, it would appear meaningful for future research to clarify whether smokers in negative affective states demonstrate a vigilance-avoidance pattern of processing drug-cues. For example, it may be helpful to present the drug cues for longer stimulus durations, as, for example, Rohner (2002) has only found avoidance at longer durations (2000-3000 ms).

*Limitations of current study and future directions*

One should exercise some caution when making inferences from this study due to the relatively small sample size. Unexpectedly, this study found an attentional bias towards smoking cues in non-smokers. This effect was not predicted and has not been shown in previous research. Given the lack of previous findings of this nature and the relatively small sample size it would appear possible that this may represent a chance effect which might not be replicable. It would be wise for future research to replicate this study with a larger sample, and a different set of participants (perhaps another type of substance use group such as alcohol users) to investigate whether these findings can be generalised. It is also important to bear in mind that this

current sample of smokers were not typically representative of the smoking population as the FTND indicated relatively low levels of nicotine dependence. It would also be prudent to replicate the study using a sample of smokers with a more dependent range of FTND scores.

According to Baker et al. (2004), negative affect will increase the incentive salience of drug cues and will be associated with both increased drug craving (as demonstrated within this study) and drug-taking behaviour. Incentive models predict that salient stimuli will not only grab attention but, in addition, be perceived as more attractive and elicit approach tendencies (Robinson & Berridge, 1993). Thus, it would be beneficial for further studies to assess the effect of negative affect upon measures of stimulus valence. For example, it may be interesting to examine the effect of mood on implicit measures of stimulus valence, such as the stimulus response compatibility task (Mogg et al., 2004). This measures behavioural response latencies towards and away from pictorial stimuli; faster latencies towards drug cues would reflect stronger approach tendencies and increased incentive salience of drug cues.

Mood induction procedures induce transient changes in mood. Perhaps, the absence of evidence that negative mood increases attentional biases in the maintenance of attention in this present study may be partly explained by the transient nature of the induced negative mood. Although there was still a significant difference in sad mood between the two MIP conditions after the attentional task, the effect of the MIP in significantly reducing happy mood was no longer evident, suggesting that the mood effect of the MIP was beginning to dissipate during the task. It would be

beneficial to clarify this issue by replicating this study with a sample of smokers, high and low in naturally occurring dysphoria that were matched on amount of nicotine use. The study found and a difference between smokers and non-smokers in self-reported anxiety within the study which was unaffected by the MIP but influenced by time. Non-smokers appeared to be able to acclimatise to the demands of the testing environment whereas smokers did not. One possible explanation for this is that smokers were more distracted by cigarette craving during the experiment and this may have interfered with their ability to acclimatise to the testing environment.

In line with incentive models of drug motivation, attentional bias for drug cues have been found to be associated with drug deprivation (Field et al., 2004a). Deprivation is proposed to further increase the incentive salience of drug cues (Robinson & Berridge, 1993; 1998). Further research might examine whether our current findings (e.g. negative mood increases an initial attentional bias for drug cues) hold up over varying levels of deprivation. According to Baker et al. (2004), deprivation increases withdrawal-induced negative affect which should further increase the ability of drug cues to attract attention.

In summary, this study provides preliminary evidence for Baker et al.'s (2004) hypotheses that negative affect increases the incentive value of drug cues, evidenced by increased attentional biases in initial orienting to drug cues. These present findings are in line with Baker et al.'s (2004) prediction that this bias in selective attention takes place in the early stages of stimulus processing. In addition, consistent with previous studies, a bias in the maintenance of attention was found, but this was related to smoking

status rather than mood state. These findings suggest that different underlying processes may mediate the relationship between attentional bias for drug-related cues, smoking status and negative mood states. Further research is required to clarify this issue.

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## List of appendices

**Smoking Habits and History Questionnaire**

1. Sex: M F

2. Age: _____years

3. How many years have you spent in education (e.g. primary, secondary and further education)?
_____years

4. On average how many cigarettes do you smoke

per day _____?
or
per week _____?

-For how long have you been smoking regularly? _____

-Do you smoke tobacco or cigarettes? _____

5. Have you ever attempted to give up smoking? Yes
No

-If so on how many occasions? _____

-For how long were you successful in abstaining? _____

-If you have successfully given up smoking when did you last smoke regularly? _____

6. How many cigarettes have you had in the last 6 hours?

_____

7. Approximately how many cigarettes have you consumed in your life time? _____

## 0-10 rating scale of mood and craving

Please circle a number to indicate how **anxious** you feel right now:

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| Not at all | | Slightly | | | Moderately | | | Strongly | | Extremely |

Please circle a number to indicate how **sad** you feel right now:

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| Not at all | | Slightly | | | Moderately | | | Strongly | | Extremely |

Please circle a number to indicate how **happy** you feel right now:

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| Not at all | | Slightly | | | Moderately | | | Strongly | | Extremely |

Please circle a number to indicate how strong your urge to smoke is right now:

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 |
|---|---|---|---|---|---|---|---|---|---|---|
| No urge at all to smoke | | Slightly | | | Moderately | | | Strongly | | Very strong urge to smoke |

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15 June 2005

Laura Hudson
Department of Clinical Psychology
University of Southampton
Highfield
Southampton SO17 1BJ

Dear Laura,

**Re: Mood and attention in smokers and non-smokers**

I am writing to confirm that the above titled ethics application was approved by the School of Psychology Ethics Committee on 9 December 2004.

Should you require any further information, please do not hesitate in contacting me on 023 8059 3995.

Please quote approval reference number CLIN/03/63.

Yours sincerely,

Kathryn Smith
Secretary to the Ethics Committee

University of Southampton

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**Mood and Attention in Smokers and Non-Smokers**

**Information sheet**

I am Laura Hudson a Trainee Clinical Psychologist. I am requesting your participation in a study regarding the role of mood and attention in smoking. This study will involve coming along to two separate sessions each lasting about 40 minutes which will be held about a week apart. You will be asked to complete some questionnaires that assess different mood states and your smoking history. You will be asked to recall some memories whilst listening to a piece of music. This is to help you get into a particular mood state (during one session you will be asked to get into a negative mood state). After this you will be shown some pictures. You will be asked to respond as quickly as possible to small arrows appearing on the screen, by pressing a response button. Your response times and eye movements will be measured while you do this task. You will also be asked to make some simple ratings of the pictures. Personal information will not be released to or viewed by anyone other than researchers involved in this project. Results of this study will not include your name or any other identifying characteristics.

Your participation is voluntary and you may withdraw your participation at any time. If you choose not to participate there will be no consequences to your grade or to your treatment as a student in the psychology department. If you have any questions please ask them now, or contact me Laura Hudson

at *lh702@soton.ac.uk.*

**Signature** **Date**

**Name** LAURA HUDSON

**Statement of Consent**

I _____have read the above informed consent form.

I understand that I may withdraw my consent and discontinue participation at any time without penalty or loss of benefit to myself. I understand that data collected as part of this research project will be treated confidentially, and that published results of this research project will maintain my confidentially. In signing this consent letter, I am not waiving my legal claims, rights, or remedies. A copy of this consent letter will be offered to me.

I give consent to participate in the above study. **Yes** **No**

**Signature** **Date**

**Name**

I understand that if I have questions about my rights as a participant in this research, or if I feel that I have been placed at risk, I can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ.

Phone: (023) 8059 3995.