University of Southampton

The relationship between associated stimuli and drug use:

The role of attentional bias

Lisa Frankland

Submitted in partial fulfilment of the

Doctorate in Clinical Psychology

Faculty of Social Science

Department of Psychology

July 2001

16199 Words

Abstract

Stimuli in the environment can become associated with drug use and act as cues to maintain drug-taking. A number of psychological theories have proposed mechanisms through which cues may influence behaviour. These are discussed in the literature review and the conclusion is drawn that research is needed that tests the specific predictions made by the currently influential models. The investigation of cognitive biases that influence response to cues is highlighted as one area that could be investigated by psychologists in the field.

An attentional bias towards drug-related stimuli has been shown in dependent subjects across substances. The empirical paper investigates the nature of this bias. The results suggest a significant attentional bias for drug related information in opiate dependent subjects when the stimuli were presented for 200ms in a dot probe task. There was no evidence of an attentional bias when the stimuli were presented for 500 or 1500ms. It is suggested that 200ms reflects an automatic level of information processing which will guide behaviour without the engagement of strategic cognitive processes. This supports the incentive-sensitisation mechanism suggested by Robinson & Berridge (1993). The clinical implications of this are discussed in terms of cognitive behavioural interventions.

Acknowledgements

I would like to express my thanks to Professor Karin Mogg and Professor Brendan Bradley for their support and advise in designing and conducting this work. I would also like to thank Lesley Millner and Liz Frankland for their comments on earlier drafts. I would like to acknowledge staff at Bath Specialist Drug and Alcohol Service and Bristol Specialist Drugs Service for their assistance in finding willing participants and offering themselves as controls. Finally I would like to thank all the participants who gave up their time to take part.

The relationship between cues and drug use:

The role of attentional bias

Contents

Empirical paper

Attentional bias in opiate dependence:

Manipulation of stimulus duration

Appendices

Disclaimer Statement

I the undersigned confirm that the work that I have presented as my thesis is entirely my own work. Reference to, quotation from, and discussion of the work of any other person has been correctly acknowledged within the work and in accordance with University guidelines for production of a thesis.

Signed [redacted] ..... Date 23/07/01

Literature Review

The relationship between associated stimuli and drug use:

The role of attentional bias

Submitted to British Journal of Clinical Psychology (See Appendix 1)

Where notes to contributors for this journal have differed from guidelines for the production of the thesis, the instructions from the doctoral course in clinical psychology have been followed.

The relationship between associated stimuli and drug use:

The role of attentional bias

Lisa Frankland

Department of Psychology

University of Southampton

Key words:

Addiction, Opiate Dependence, Attentional Bias

Running Title:

Associated stimuli and drug use

Correspondence should be addressed to Lisa Frankland, c/o Department of Psychology, University of Southampton, Highfield, Southampton, SO17 1BJ, United Kingdom (Telephone: 02380-5952588; Fax: 02380-592588)

Abstract

Purpose

The objective of this review is to consider the relationship between environmental stimuli and drug use. It explores, through the examination of theory and research, the most useful ways to test the proposed psychological mechanisms put forward by theories of drug dependence.

Methods

Information for this review was obtained through searches on psychological databases. Further references were collected from review articles published in the past two years. Psychological theories were selected for review only if they proposed a mechanism through which cues may act to influence drug use.

Results

Through critical discussion of the theoretical models, the review draws out the question of how accessible drug seeking behaviour is for conscious cognitive reflection. Examination of the limitations of research techniques and the influential construct of craving leads to the suggestion that psychological research needs to focus on objective measures of cognitive bias. Attentional bias may be a useful indicator of the proposed mechanisms through which cues are said to influence drug use.

Conclusions

There is a need to empirically test the proposed theories against each other and to develop objective measures which can act as indicators of the theoretical mechanisms. Further research into attentional bias in addiction may inform understanding and have important clinical implications.

Introduction

Individuals who are dependent on a substance are reminded of it by stimuli in the environment that they associate with using e.g. objects, specific places or people. This is commonly recognised in the lay population, for example cigarette smokers may report habitually wanting a cigarette when they have a coffee, or after a meal. The connection between coffee and cigarettes occurs through repeated exposure of one stimulus, the cigarette, with another, the coffee, so that over time the two become associated. At a later point in time, the sight or smell of coffee may trigger thoughts and desire for cigarettes.

It is well known by psychologists working in the field of substance dependence, that stimuli in the environment can become associated with drug use, and act as cues to maintain drug-taking behaviour. The focus of this review is to critically discuss the proposed mechanisms for this process. Historically the main contributions to this understanding have been based on conditioning (Stewart, de Wit & Eikelboom, 1984; Wikler, 1948) and cognitive behavioural theories (Marlatt & Gordon, 1985; Tiffany, 1990). In the last decade, theories have combined knowledge of psychological processes with those in the fields of biological and neuroscience (Robinson & Berridge, 1993). There is no clear accepted model which outlines how drug cues affect individuals with opiate dependence. It is known not to be a simple linear relationship, as exposure to drug-associated cues does not consistently lead to drug use. It is most likely that idiosyncratic cues develop motivational significance, as they develop the potential to motivate behaviour towards drug seeking, through individual associations. The presentation of cues may interact with mood, thoughts and behaviours in different ways at different times. The aim for theories and research

in this area is to find out more about the mediating and moderating processes in order to make some reliable predictions, which may inform interventions.

Structure of the review

This review will critically discuss the relationship between the processing of drug-related stimuli and drug use. It is not possible within the scope of this review to analyse wider theories of drug use, although significant contributions from other areas will be drawn on with reference to psychological research and theory. The discourse will support the proposition that objective empirical research must be undertaken to test the specific predictions made by currently influential models. Suggestions of how to clarify understanding of cognitive mechanisms underlying the dependence will be made, and the clinical implications of this will be discussed. The background is set with a review of the relevant theoretical models.

Theoretical review

The process of drug-related stimuli acting as cues to drug seeking and ingestion, in dependent users, has been explicit within the research literature since the writing of Wikler (1948). The recognition that cues may have a role in the maintenance of drug use and relapse following abstinence has led to many theories about the mechanisms underlying this relationship, (e.g., Wikler ,1948; Stewart, de Wit & Eikelboom, 1984; Tiffany ,1990; Robinson & Berridge,1993). A critical review of psychological theories will follow. The first section will critically discuss models based on conditioning theory. It will outline models which emphasis the role of negative reinforcement (Wikler, 1948) and positive reinforcement (Stewart, de Wit & Eikleboom, 1984) as the primary mechanism linking cues and drug use. It will go on

to discuss theories that integrate principles of reinforcement with neurodevelopment (Sculteis & Koob, 1996; Robinson & Berridge 1993). The second section will review the contribution of cognitive and social learning theories (Marlatt & Gordon 1985; Tiffany 1990).

Conditioned withdrawal (Wikler, 1948)

Wikler (1948) first proposed that addiction developed because of the propensity of drugs to remove unpleasant withdrawal symptoms through negative reinforcement. Stimuli present at the time of drug use were said to become associated, through conditioning, with the experience of withdrawal. Environmental stimuli, over time and with repeated associations, then develop the potential to trigger a withdrawal-like reaction when they are presented alone. Through this process of associative conditioning, Wikler (1948) suggested that the stimuli themselves acquire motivational significance. This means that as the stimuli trigger a withdrawal like reaction, behaviour is motivated towards drug taking in order to eliminate the negative effects of the withdrawal experience. Ingestion of the drugs eliminates the withdrawal symptoms and the behaviour is negatively reinforced.

There has been extensive work in humans and animals to show that withdrawal reactions can be conditioned to drug-related stimuli. Laboratory experiments have shown that, after as few as seven pairings, a conditioned stimuli of peppermint odour, present when subjects are experiencing withdrawal, has shown the ability to induce withdrawal symptoms when presented alone (O'Brien, O'Brien & Mintz, 1975; O'Brien, Testa, O'Brien, Brady & Wells, 1977). It has also been shown that subjects have experienced withdrawal effects in response to videotapes of drug use. Decreases in skin temperature and skin resistance were evident along with self-

reported withdrawal and craving when subjects watched video tapes of previous drug-taking environments (Childress, McLellan & O'Brien 1986 a,b; Ternes, O'Brien, Grabowski, Wellerstein & Jordon-Hayes, 1980). These studies did not use control groups or control stimuli so the results must be interpreted with caution. Although these experiments do not conclusively show evidence for drug conditioning, they do offer an explanation for the withdrawal experience that drug users sometimes report when returning to previous drug-using environments (O'Brien, Childress, Ehrman & Robbins, 1998).

Wikler (1948) proposed that the withdrawal reaction was a key mechanism in drug use. It is unlikely that withdrawal is a fundamental motivating factor across all substances but it may have a role in explaining one aspect of responses to drug cues under specific conditions. This early work is limited by its inability to explain the acquisition of drug dependence. It does not explain why people use drugs before they experience withdrawal, or develop a dependence syndrome. It is known that physical dependence is not necessary for opiates to be sought and injected (Beach, 1957; Deneau, Yanagita & Seevers, 1969; Schwartz & Marchok, 1976; Woods & Schuster, 1971), so a mechanism other than withdrawal must be motivating behaviour at this point. A unitary explanation of drug withdrawal maintaining drug use is inadequate because there is no single defining withdrawal syndrome across addiction, (Stewart et al., 1984; Wise 1988; Wise & Bozarth, 1987) and withdrawal symptoms are very different for different drugs (Schulteis & Koob, 1996). Some drugs that do not produce strong withdrawal syndromes, such as psycho-stimulants, can be highly addictive (Robinson & Berridge, 2000), and some drugs produce tolerance and withdrawal but not compulsive use, e.g. some tricyclic anti-depressants, anticholinergics and kappa opioid agonists (Robinson & Berridge, 2000). Further

evidence against this model has been shown in experimental work with animals where rats self-administered low doses of opiates to restricted brain sites (Bozarth & Wise, 1981) which are known to be independent of those involved with withdrawal reactions (Bozarth & Wise, 1983; Wei, 1981). The accumulation of this research suggests that there is more than withdrawal motivating this behaviour.

As an all-encompassing model of drug dependence the theory of conditioned withdrawal is limited, but in combination with other mechanisms it may have a role in explaining why some dependent users continue to take drugs. The principle of withdrawal motivating drug use is more useful when combined with the principles of positive reinforcement and knowledge of brain development. Psychopharmacological research investigates how the brain can develop over repeated exposure to drugs and change neurological and affective responses to the same dose. The process of the brain changing over time is termed neuroadaptation. This is referred to in homeostatic models which will be discussed next.

Homeostatic models

The conditioned opponent process theory (Siegel, 1989) is an adaptation of the opponent process theory (Solomon & Corbitt, 1979). It is based on the premise that the body has a homeostatic drive to return to a neutral state. This means that if something has a positive effect on the body, then the body will produce a negative effect to cancel it out in order to return to a neutral state. The conditioned opponent process model (Siegel, 1989) postulates that many reinforcers, such as drugs, which produce positive affective processes in the body, stimulate a negative affective process because the body's natural response is to seek homeostasis. This means that on immediate presentation, the individual experiences positive effects of the drug,

e.g. euphoria, but the body works to maintain a neutral state so as drug levels deplete, the individual experiences the negative effects of withdrawal. Homeostatic models acknowledge that positive reinforcement occurs due to the immediate effects of the drug, but they also predict that a negative emotional state, similar to withdrawal, will occur in response to drug cues over time. The motivational properties of the drug are defined as its ability to produce positive affect and its ability to decrease negative affect and somatic symptoms (Schulteis & Koob, 1996). These ideas have been extended with the integration of neuroadaptation to explain the biochemical mechanisms responsible for the homeostatic response (Koob & Bloom, 1988; Koob, Markou, Weiss & Schulteis, 1993; Koob, Stinus, Le Moal & Bloom, 1989). Although different drugs have distinct pharmacological profiles, opiates, psychostimulants and alcohol all share positive reinforcing properties (Koob, 1992). Upon acute administration, the neural substrates for the positive reinforcing actions appear to involve common reward circuitry in the central nervous system. Alterations in brain reward circuitry expressed as emotional motivational signs of withdrawal, may be common elements of dependence across drugs, but physiological signs of withdrawal do not necessarily reflect the affective neuroadaptations (Schulteis & Koob, 1996). Opiate withdrawal creates a negative affective state, which acts as a dysregulator of motivational homeostasis. The individual therefore seeks to return to homeostasis through seeking more opiates and is negatively reinforced in the process. There is now considerable evidence that neuroadaptation to repeated drug administration results in the emergence of negative reinforcement processes as additional motivational factors in the transition to and maintenance of compulsive use (Schulteis & Koob, 1996). Alleviation of withdrawal may not be the sole motivational force in continuing drug use as was suggested by the early model

(Wikler, 1948). It appears most likely that the motivating factors for the continuation of compulsive drug use are a combination of withdrawal and memory of the pleasurable effects of drugs (Schulteis & Koob 1996). The desire for further drug use may be characterised either by a deficit state (withdrawal or conditioned withdrawal), or a sensitisation of the reward system to stimuli that predict drug effects (e.g. conditioned reinforcement), or both Koob (1996). Homeostatic models acknowledge the potential of positive reinforcement as a mechanism in motivating drug-seeking behaviours. The ability of drug-related stimuli to become conditionally associated with the positive effect of drugs was outlined comprehensively by Stewart, de Wit & Eikelboom (1984).

Drug like effects (Stewart, deWit & Eikelboom 1984)

While drug-related external cues can elicit drug-withdrawal or drug-like effects, Stewart, de Wit & Eikelboom (1984) suggest that it is the drug-like effects that are most significant in motivating further drug use. They suggest that the negative reinforcing properties of drugs, the withdrawals, are not significant in maintaining or reinstating drug-taking behaviour. In this model, drug use is explained as being due to the positive reinforcement of taking substances that have pleasurable effects. This model suggests that having drugs in the system activates motivational systems in the brain and increases the probability of drug-related thoughts (Stewart et al., 1984). Environmental stimuli which over time become conditioned to the positive effects of drugs, activate the central neural state similar to that elicited by drugs. Through paired association the cues develop the potential to elicit drug-related thoughts and increase the salience of other drug-related stimuli. The individual is primed to reinstate drug use by the activation of the same motivational state as is triggered by

exposure to the drug itself. Ingestion of the drug is also suggested to prime further drug use, through activation of the central motivational positive reinforcement system. Drug-users learn through positive reinforcement to expect a pleasurable affective state when drugs are ingested and this motivates behaviour towards drug use. This has been shown in experimental conditions in which priming injections of drugs increase incentive motivation for drug-seeking behaviour (Davis & Smith, 1987; Geber & Stretch, 1975; de Wit & Stewart, 1981,1983). When drugs are present in the body, the individual is motivated to seek further drug use. This suggests that the presence of the drug in the body activates the motivational appetite which reinstates drug seeking (Niaura, 2000).

Research in both animals and humans has shown the powerful positively reinforcing effects of drugs, which could maintain drug use. Studies using rodents and primates have demonstrated that previously neutral stimuli paired repeatedly with either contingent (self-administered) or non-contingent (experimenter administered) drugs, will acquire reinforcing properties as measured by their ability to induce operant responding (Davis & Smith, 1987; Goldberg, 1976). In humans a positive affective state induced by heroin and measured by a subjective reported 'high' can be conditioned to environmental stimuli (Childress, Ehrman, Rohsenow, Robbins & O'Brien, 1992; O'Brien, Greenstein, Ternes, McLellan & Grabowski, 1979). The drug-taking environment, paraphernalia and procedures associated with drug taking can be conditioned to the positive reinforcing or euphoric effects across categories of drugs, e.g. cocaine (Childress, McLellan, Ehrman & O'Brien 1988), and heroin (Meyer & Mirin, 1979), so that the subjects report drug-like effects on exposure to the previously neutral stimulus. This conditioning process is also evident in studies which have shown that dependent users can experience pleasure when

injecting inert substances (Levine, 1974), and report an opiate like euphoria from injecting saline in semi-naturalistic conditions (O'Brien, Chaddock, Woody & Greenstein 1974).

As an overall explanation of drug use this model is limited because there is no clear relationship between the euphoric effects of drugs and their potential to develop dependence. Subjective states are often poorly correlated with drug taking (Robinson & Berridge, 2000). Over time drug taking may increase with dependence but the pleasure induced is not reported to increase (Robinson & Berridge, 1993). It has been noted that conditioned euphoria is much less common than conditioned craving or withdrawal-like effects (O'Brien, Childress, McLellan & Ehrman, 1992), and drug taking can be maintained in the absence of positive effects (Fischman & Foltin, 1992; Lamb, Preston, Schindler, Meisch, Davis, Katz, Henningfield & Goldberg, 1991). The pleasurable effects of drugs and the motivation to continue drug use often occur together, indicating a correlational relationship, but this is not evidence of a causal relationship (Robinson & Berridge, 2000). Conditioned euphoria alone is an inadequate explanation to explain maintained drug use in the presence of negative as well as positive effects.

Limitations of Conditioning Theories

Although both negative affective states (withdrawal) and positive affective states have the potential to become associated with drug stimuli through the process of conditioning, the motivational significance of drug cues can not be assumed to be unidirectional. Each of these mechanisms of positive and negative reinforcement may have a potential role in explaining the link between cues and drug use but neither of these processes is able to fully account for the process. The ability of drug-

related cues to cause physiological responses, self-reported craving and withdrawal is well established (O'Brien et al., 1998). It is more difficult to establish either that such responses result from a past history of conditioning or that they motivate continued drug use (O'Brien et al., 1998). This has been tested through attempts to extinguish the conditioned relationship by systematic gradual exposure to drug-associated cues without the possibility of pharmacological reinforcement (O'Brien et al., 1974; O'Brien, Greenstein, Ternes, McLellan & Grabowski 1980). In an attempt at extinction a group of detoxified long-term drug free opiate users, were asked to perform unreinforced self-injections under laboratory conditions. This was ineffective and many subjects experienced increased withdrawal and craving and found the injections so unpleasant they had to pull out (O'Brien et al., 1974; O'Brien et al., 1980). These researchers later used videotaped presentations of drug use which produced significant reductions in reported withdrawal and craving but showed no evidence for an effect of extinction on treatment (Childress et al., 1986a). Similar results were found with subjects on methadone (Childress, McLellan & O'Brien, 1984; Childress et al., 1986b). Treatment showed increases in self-reported withdrawal and craving in response to opiate cues and subsequent reductions in craving response during extinction but there was no specific effect of extinction treatment on outcome (Childress, et al., 1984; Childress, et al., 1986b). Similarly results in other studies have shown a reduction in reactivity but no differences in clinical outcome for drug use (Dawe, Powell, Richards, Gossop, Marks, Strang & Gray, 1993; Powell , Gray & Bradley, 1993). Extinction programmes, which are the clinical interventions generated from conditioning theories, have not yet been shown to be effective in reducing drug use. This suggests that because the application of

extinction has not yet worked, there may be mechanisms in addition to conditioning which affect the relationship between cues and drug use.

The limitations of each of the positive and negative reinforcement models highlight the difficulties of attempting to explain reactions to drug cues and drug use in a simplistic monistic model. Each of the monistic models has empirical evidence for and against it, which give confusing and conflicting results. This is reflective of the complex interactional relationships between cues, drugs and drug effects (Drummond, 2000), which as yet have not been organised into a clear model.

Conditioning theory alone is inadequate in explaining the link between drug-related stimuli and drug use. There is a need to look at other knowledge, which can be integrated to give further insight. This has been happening with the integration of neurobiological knowledge (Robinson and Berridge, 1993), and the development of cognitive theories (Marlatt & Gordon, 1985; Tiffany, 1990). These theories will now be reviewed.

Incentive-sensitization theory (Robinson & Berridge, 1993)

The incentive-sensitisation model of drug dependence posits that all potentially addictive drugs share the ability to alter brain organisation. They all act on the brain systems involved in the process of incentive motivation and reward: the mesolimbic dopamine system, which is responsible for drug seeking and behaviour. Through the process of neuroadaptation, the brain reward systems become hypersensitive (sensitised) to drugs and drug associated stimuli. The brain systems that are sensitised do not mediate the pleasurable or euphoric effects of drugs (drug-liking), but instead mediate a sub-component of reward that is termed incentive salience (drug-wanting), (Robinson & Berridge, 1993, 2000, 2001). Through this system

drugs are attributed salience. Salience attribution transforms the drugs into stimuli, which 'grab attention', become attractive and 'wanted' and guide behaviour towards drug seeking. At this point drugs have motivational salience above all other stimuli in the environment and guide behaviour towards the incentive of drug taking over all other actions. This theory suggests that drug 'wanting' may not be conscious so drug use may occur without conscious awareness (Robinson & Berridge, 2001). It predicts that over time a bias in attention orientation toward drug-associated stimuli develops. The stimuli grab attention, become attractive and wanted, and guide behaviour without conscious awareness toward drug seeking and ingestion (Robinson & Berridge, 2001).

Robinson & Berridge (1993) propose that individual susceptibility to sensitisation varies according to genetic, hormonal, and experiential factors. These factors, which render people susceptible to sensitisation, are what contribute to the individual variation in susceptibility to drug dependence and sensitisation can also be modulated by environment (Robinson & Berridge, 2000). The expression of sensitisation is context specific so neural sensitisation would be expected to be strongest in contexts in which the drugs have often been previously used. The interaction between the neural sensitisation system and associative learning means that associated stimuli become powerful incentives themselves, and acquire the ability to activate the system independent of the administration of the drug. Also in familiar environments, unless there are high doses administered, the familiar context and lack of cues to drug administration may preclude the development of sensitisation at low doses (Robinson & Berridge, 2000). Robinson & Berridge (1993) suggest that an indicator of susceptibility to sensitisation, and therefore to addiction may be the degree to which attention is drawn to drug-related stimuli. As this model

proposes that there are individual differences in the degree of activation of the system, measures of attentional bias have been suggested to be an indication of the activation of the incentive-sensitisation system (Lubman, Peters, Mogg, Bradley & Deakin, 2000).

This theory proposes some interesting hypotheses, which are as yet unproven. The brain physiology involved in sensitisation needs to be identified and ways of testing the hypothesised processes need to be developed. As yet the research in humans is limited. It is unclear whether the research on animals will generalise or be transferable. There is a need to develop ways of testing the theory in humans. As previously mentioned, cognitive biases in attending to and processing dependence-related material have been suggested to be indicators of activation of the incentive sensitisation system (Lubman et al., 2000). Further research is needed to investigate whether attentional bias is an accurate index of activation of the incentive-sensitisation system. If it is, then measures of attentional bias can be used to test predictions of the theory. This will be discussed further under future directions of research. Cognitive theories of responses to drug cues will be critically discussed next.

Cognitive social learning theory (CSLT) (Marlatt & Gordon, 1985)

This influential theory has informed cognitive interventions, which assist people in avoiding relapse. Based on Social Learning Theory (Bandura, 1977), it states that in a 'high risk situation', in which the individual is faced with cues that are likely to trigger them into thinking about taking drugs, they have the choice whether or not to use them. This model states that the likelihood of their responding to the cues depends on an interaction between their expectations about their own efficacy and

the consequences of using. Efficacy expectations are based on how much confidence they have in their own ability to resist using (Bandura, 1977). Outcome expectations are based on what they believe will be the consequences of using or not using. These may be positive e.g. positive mood, relief of pain, or negative e.g. lowered mood or breakdown in relationships. CSLT has been extended with acknowledgement that cognitive biases may be present and affect the outcome when making these decisions (Beck, Wright, Newman & Liese, 1993). One example of these cognitive mechanisms is attentional bias in which attention is selectively drawn to drug-associated stimuli over all other stimuli in the environment (Beck et al., 1993). It is proposed that therapeutic techniques to discuss the cognitive and behavioural processes that guide drug use will enable the therapist and client to determine the biases that the individual may be applying to the situation. Intervention is aimed at cognitive restructuring of the beliefs about substance use and at developing cognitive and behavioural strategies to overcome the biases. The strength of this model is its clinical applicability.

Marlatt & Gordon (1985) purport that inflated 'positive outcome expectancies' and minimised negative expectancies along with poor self-efficacy leads to further drug use. In support of this proposition, cross-sectional and prospective research has shown that alcohol consumption varied as a function of positive belief biases. Beliefs that consumption would enhance social and personal functioning have been shown to be associated with increased instances of drinking (Christiansen, Smith, Roehling & Goldman, 1989; Jones & McMahon, 1996; Sher, Wood, Wood & Raskin, 1996; Stacy, Newcomb & Bentler, 1991). Bias expectancies have also been shown to have predictive validity regarding abstinence and relapse in nicotine smokers (Brandon & Baker, 1992), marijuana and cocaine users (Schafer &

Brown, 1991). Similarly in a study of four groups of teenagers, positive and negative outcome expectancies had discriminant validity in predicting drug use behaviours and vulnerability to the use of illegal drugs (McCusker, Roberts, Douthwaite & Williams, 1995).

A limitation of this model is that it is not able to account for the loss of control or the desynchrony between cognitive intentions and ongoing behaviour (McCusker, 2001). One of the defining features of addictive behaviour is the continuation of the behaviour despite consciously expressed intentions not to. Researchers of cognitive biases have suggested that the processes, which guide drug use behaviour, are outside conscious awareness and volitional control (McCusker & Gettings, 1997; McCusker, Leung & Armstrong 1999). If this is so then it is questionable whether the information related to drug-seeking behaviour is immediately accessible to reflect on in everyday situations pertaining to the addiction (Leung & McCusker, 1999). Cognitive social learning theory relies on subjective self-report measures and conscious cognitive reflection of behaviours related to substance use. It assumes that effortful cognitive strategies can govern drug-seeking behaviour. This is contradictory to the incentive-sensitisation model (Robinson & Berridge, 1993) which suggests that behaviour is determined primarily by preconscious processing and is not immediately available to conscious cognitive processing. An understanding which suggests that drug-using behaviour is governed by behaviour at an automatic level of processing gives a clearer understanding why there is desynchrony between intentions to abstain and behaviour that maintains drug seeking and consumption (McCusker, 2001).

Research on cognitive biases in addiction has tested the proposals of CSLT. It has been shown that the proposed 'positive outcome expectancies' and 'minimised

negative expectancies' are not consistently present in dependent users. Although smokers were shown to endorse more positive expectancies when compared to non-smokers, analysis within the groups of smokers showed that they endorse just as many negative as positive outcome expectancies (Leung & McCusker, 1999; Litz, Payne & Colletti, 1987). This is inconsistent with the expectation that dependent drug-users would show a positive bias for outcome expectancies. Similarly in alcohol users (Curran, 1999) and in repetitive drug users (McCusker et al., 1995), there is evidence of a negative bias in the outcome expectancies of substance use. Drug users have been shown to be as aware that the negative effects of their using behaviour outweigh the positives (McCusker, Leung & Armstrong, 1999; Plant & Plant, 1992), yet the behaviour is maintained. McCusker (2001) suggests that this implies that cognitive biases that govern dependence and motivate behaviour, occur at automatic, implicit and preconscious levels of awareness and are outside of volitional control (McCusker & Gettings, 1997; McCusker, Leung & Armstrong, 1999). If this were true they would be less available for conscious reflection as is expected in CSLT, and this may explain why the behaviour continues despite expressed desires to stop and awareness of the negative outcomes of the behaviour.

This model is strong in its applicability to clinical practice but it may be enhanced by integration with research that indicates the nature of the cognitive biases that may affect decisions about drug use. If, as is suggested, by Robinson & Berridge (1993), and McCusker (2001), behaviour is governed by automatic, preconscious processing, then the model and interventions need to be adapted to incorporate this. The cognitive processing model (Tiffany, 1990) accounts for this in part by integrating knowledge from general cognitive psychology to explain how drug

seeking and consumption may be guided by automatic processing and conscious strategic processing in different circumstances.

Cognitive processing model (Tiffany, 1990)

This cognitive model of drug use is based on cognitive psychology's understanding of automacity (Logan, 1988; Posner & Snyder, 1975; Shiffrin & Schneider, 1977). It proposes that drug seeking and drug consumption behaviours have the attributes of automatised behaviour. That means that they are stereotyped, highly stimulus bound, lack control and require little cognitive effort. Over time drug seeking and consumption behaviour, because it is highly learned and repeated frequently, becomes a stereotypical automatic sequence. Because it involves little cognitive effort it does not interfere with other automatised behaviour. This automatic sequence can be triggered by stimuli that have become associated with it, and due to the nature of the sequence it is difficult prevent when triggered and extremely difficult to interrupt. Cognitive regulation of automatised performance seems to occur outside of conscious awareness. Tiffany (1990) proposes that drug taking in dependent users can be viewed as an example of the kinds of behaviours one would expect with any highly automatised skill. Only when the sequence is interrupted by an unexpected incident does the process involve effortful, conscious cognitive processing in order to overcome the interruption. This understanding of drug use behaviour, enables theorists to draw on literature from the cognitive sciences to investigate the cognitive control of automatised behaviour. This model suggests that at times drug-seeking behaviour requires little cognitive effort or attention as it is governed by automatic processes. At other times when the sequence is interrupted the drug-seeking will consume attention, occupy thoughts and interfere with other

goal orientated behaviours. Tiffany (1990) does not make any specific prediction about attentional bias, but this theory needs to be tested by looking at the occupation of attention by drug-related stimuli at different times in the drug sequence. This theory explains how drug-seeking behaviour can become automatic and engages little conscious thought, but it does not explain why the habitual nature of drug-seeking behaviour should be viewed as a key factor in the acquisition and maintenance of drug use. This theory needs to be systematically evaluated to investigate the nature of the proposed characteristics and whether they do apply to behaviours associated with dependence.

Summary of theoretical review

This theoretical review has critically discussed the proposed mechanisms through which stimuli associated with drug use can come to elicit drug seeking behaviour and consumption. The models differ in how they propose that this occurs e.g. negatively or positively reinforced conditioning, eliciting activation of an incentive sensitisation system, or triggering automatic behaviour patterns. One question that has emerged is how available drug seeking behaviours are to conscious cognitive reflection, and how well current interventions access the mechanism that motivates drug use. A systematic approach is now needed to investigate specific aspects of these theories in order to obtain a clearer understanding of the potential role of associated stimuli in drug taking (McCusker, 2001). Research in this area has advanced in the last two decades, yet the theories have yet to be tested against each other (Drummond, 2001). Future directions for research will now be discussed, but first the application of the theories to clinical practice will be critically reviewed.

Linking theory to practice

An important factor in any theory is its application to clinical practice. The approach that has most influence in the clinical field at the current time is CSLT. It proposes that cognitive techniques can be applied to enable individuals to mobilise coping strategies when they are most at risk of using. What has come out of the theoretical review is the suggestion that cognitive biases, which influence behaviour, may not be immediately available to conscious processing. Both Tiffany (1990) and Robinson and Berridge (1993) have suggested, through different mechanisms, that behaviour leading to consumption is primarily guided at an automatic level of processing. The implication for interventions may be that they will be less effective if attempts are not made to interrupt the automatic process (Tiffany, 1990), or bring into conscious awareness the triggers for the incentive-sensitisation system (Robinson & Berridge, 1993). In order to improve on the clinical application it is necessary to test the theories which suggest that drug seeking is governed by automatic processing. If it is found to be the case, then clinical interventions need to be adapted to take account of this.

Limitations of current research

One weakness of research in this area so far has been the focus on subjective measures, which do not have a strong theoretical basis. This is a complex area with many confounding factors that make it difficult to draw conclusions. In order to suggest future directions for research, the difficulties will be outlined first.

The complexity of procedures and cues

There is a lack of standardised procedures for investigating reactions to cues. There are so many different research designs and measurement tools that results can not be generalised across studies and this has made meta-analysis difficult (Carter & Tiffany, 1999). The complexity of the area has been added to by the variety of items that can be viewed as cues. Drummond (2000) outlines the types of characteristics that may affect the relationship between the cue and subsequent drug seeking or consumption. These are:

1- Exteroceptive characteristics, examples of which are sight, smell, and taste, which will vary and hold different motivational significance for different individuals.

2- Interoceptive characteristics, such as mood, cognition and priming all of which are idiosyncratic and vary both within and between individuals.

3-Temporal relationships, for example cues proximal to ingestion may be more salient and therefore produce greater reactivity.

4- Cue relationships, in which the context of cues will influence salience and reactivity, e.g. cluster of cues may be necessary to activate drug seeking and consumption. Each of the cues individually may be a necessary part of the mechanism but not sufficient to instigate behaviour when presented on their own.

Limitations of measurement techniques

In the last decade the complex relationship between drug cues and drug effects has been explored through laboratory simulation. The reactions of dependent users to presented drug cues have been observed and measured in attempts to understand the mediating and moderating factors and the important variables in the relationship between cues and drug use. This area of research has been termed cue reactivity, and

the variety of possible reactions to cues have been organised into the cue-reactivity paradigm (Drummond, Tiffany, Glautier & Remington, 1995), which presents a framework for investigating drug cue relationships. There have been three standard approaches to measuring cue reactions. They are subjective self-report, physiological reactivity and behavioural reactions. Each of these approaches to measurement has limitations and they will be discussed next.

1. Symbolic expressive reactivity refers to the individual's subjective responses to the presented cues, taken as measures of craving, anxiety and pleasure. The subjective self-reported feelings of the dependent users are difficult to compare between studies due to the lack of standardised measurement used (Sayette, Shiffman, Tiffany, Niaura, Martin & Shadel, 2000). Cross study comparison is further hindered by the subjective nature of reporting on these measures (Tiffany, Carter & Singleton, 2000) and the range of definitions and interpretations of the term craving (Drummond, Litten, Lowman & Hunt, 2000). The complications of relying on the construct of craving will be discussed separately.
2. Physiological reactivity is the body's biological response to cues, assessed with measures such as heart rate and skin conductance. This does offer an objective measure but there are difficulties due to the idiosyncratic nature of physiological reactions to substances. Psycho-physiological equipment is used to take readings e.g. of heart rate and skin conductance, but some difficulties have arisen due to variable and unpredictable reactions to substances. For example the direct effects of heroin on heart rate, are complex (Legarda, Bradley & Sartory, 1990), and vary according to dose and tolerance (Fennessy & Ortiz, 1968; Meyer & Mirin, 1979; Volavka, Levine, Feldstein & Fink, 1974). Dependence on the drug and tolerance must therefore be controlled for when relying on these measures. As

the response patterns of individuals vary according to their history and metabolism, it is difficult to investigate physiological effects and draw conclusions that can be generalised to a wider population. This is a promising objective measurement approach because it offers a variety of objective measures, which are being developed. It needs to be investigated within a wider body of knowledge that is able to make better predictions on how the individual will react to known variables.

3. Behavioural reactivity refers to the individual actions directed at drug seeking or consumption. This involves the extent to which cues lead to subsequent drug seeking behaviour and consumption. This area has often been investigated through retrospective analysis of instances of relapse. The main difficulty with this is the reliance on conscious cognitive reflection of the motivating mechanisms at the time of drug seeking or after the event. It is questionable whether such self report strategies reflect the cognitive processes that motivate ongoing behaviour (Feldman & Lynch, 1988; Nisbett & Wilson, 1977). At the time at which subjects are responding to the questions about their behaviour, the memories and processes that were motivating them previously may no longer be accessible in memory (Cooney, Gillespie, Baker & Kaplan, 1987). Accessibility to information in memory is highly cue dependent and in particular situations an accumulation of cues may trigger automatic behaviours that are difficult to reflect on at a later stage in a different situation, in the absence of cues and the previous emotional state, (Cooney, et al., 1987). McCusker (2001) argues that in retrospective reporting the individual is engaged in a 'cued-judgement' exercise (Leung & McCusker, 1999) in which their choices are confounded by dissonance reactions based on socially held facts, rather than personally motivating

propositions. It may be that after the event drug users report what they think they should say, or what they know rather than accessing the motivations for using drugs which had been active at the time. If, as is suggested by Robinson & Berridge (1993), behaviour is governed by activation of the incentive-sensitisation system, then the processes guiding behaviour may be less accessible to retrospective reporting. At that point in time, when they are expected to give an answer, socially held facts about drug use may be more accessible.

The limitations of the measurement techniques, the lack of standardised procedure and the complex nature of potential cues all highlight why it has been so difficult for research to gain a clear understanding in this area. Added to this is the construct of craving, which is referred to throughout the literature. Historically craving has been viewed as a crucial mediating variable between cues and drug use. In lay terms it makes intuitive sense to understanding craving within a linear model, in which cues, trigger craving, which leads to drug use. Research has however been unable to support this understanding of craving, and as a subjective measure it is vulnerable to the limitations which were previously discussed. The difficulties with the construct of craving as a mediator between cues and drug use will be outlined next to clarify why it should not be relied on as a central tenet in current investigations.

The limitations of 'craving'

Craving is a useful lay term, for drug users to describe the physical and psychological discomfort elicited by cues and associated with withdrawal (Drummond et al., 2000). As a result, the professional community has held on to the assumption that craving is responsible for compulsive drug use despite the lack of evidence (Tiffany & Carter, 1998). There is no clear agreed definition of craving

across disciplines, no reliable and valid measure of the construct, and no evidence, which indicates that it will predict drug use or relapse. Research which has used craving as a key component in linking cues and drug use has, as a result, been of weak design and inconclusive. Craving is not a consistent feature of either drug use or relapse (Tiffany, 1990) and there remains a difference of opinion between researchers as to whether it is a necessary, but not sufficient, component of drug use (Drummond, 2000), or a redundant epiphenomenon, neither necessary nor sufficient (Marlatt, 1978). In order for the construct of craving to be of use, it must be theoretically based and backed by empirical research.

The term craving is used in many different ways. Some clinical researchers view it as a conscious cognitive process measured by self-report. Some psychological theories refer to craving as a preconscious state (e.g. Robinson & Berridge, 1993). Neuroscientists investigate what they term craving using neuroimaging (Weinstein, Feldtkeller, Malizia, Wilson, Bailey & Nutt, 1998). Some cognitive theorists view it as reflecting retrieval from memory of a learned desire to satisfy a biological or psychological need (Abrams, 2000). Given this wide variation of uses, it is questionable whether researchers are measuring different aspects of the same phenomenon, or different phenomenon (Drummond et al., 2000).The lack of clear definition has led to a complex and at times confusing array of research which makes very different claims about craving and its usefulness in investigating links between cues and drug use.

It is no longer assumed that craving, as a conscious cognitive process, needs to be present for drug use to occur (Drummond, 2000; Robinson & Berridge 1993, 2000; Tiffany, 1990). Research has highlighted that there is no evidence for a direct causal relationship between craving and relapse after abstinence (Drummond, 2001).

Despite this, interest remains because the presentation of drug-related stimuli to drug dependent individuals reliably produces cue-specific increases in reported craving (Tiffany & Carter, 1998). In extensive laboratory based investigations with cigarette smokers, the presentation of smoking related stimuli, through imagery or in-vivo, has consistently produced elevated craving scores relative to control conditions (Burton & Tiffany, 1997; Cepeda-Benito & Tiffany, 1996; Elash, Tiffany & Vrana, 1995; Maude-Griffin & Tiffany, 1996; Tiffany & Drobes, 1990; Tiffany & Hakenwerth, 1991). This effect has been shown across substances. A meta-analysis of research using this method of cue reactivity, showed consistent increases in craving in response to drug-related material over control conditions in alcohol, heroin and cocaine dependent subjects (Carter & Tiffany, 1999). The results of these studies show that there is a correlation between the presentation of drug-related stimuli and self-reported craving. It does not however indicate a causal relationship. Research, which has attempted to investigate more directly the relationship between craving and drug use, has been unable to report a robust causal relationship (Tiffany & Carter 1998). A review of laboratory based studies which have asked subjects to rate craving and given them the opportunity to consume drugs have not shown a strong relationship between ratings of craving and use of the drugs (Tiffany 1990). If craving were responsible for drug use then correlations between the measures should be high. Tiffany & Carter (1998) in their review, report them to be non-significant or modest. In those that were significant, correlations between craving and drug use measures generally accounted for less than 25 percent of the shared variance between the two variables. Similarly in retrospective analyses of relapse episodes, the results do not indicate a strong relationship between craving and drug use. Research has shown that people rarely spontaneously cite craving as their reason for returning to

drug use, (Bradley, Phillips, Green & Gossop, 1989; Littman, Stapleton & Oppenheim, 1983; Ludwig, 1989; Marlatt & Gordon, 1980; Wallace, 1989). Similarly in one study which specifically asked addicts to identify relapse precipitants, only 7 percent of 300 subjects identified craving as the primary reason (Miller & Gold 1994). Further evidence for the independence of the two variables, craving and drug use is shown in interventions which have reduced craving without attenuating drug use (Fischman, Foltin, Nestdt & Pearlson 1990), and conversely drug use behaviours, which have changed with no effect on craving ratings (Dawe, Gerada, Russell & Gray, 1995; Gross & Sitzer, 1989; Lamb et al., 1991; Nemeth-Coslett & Henningfield, 1986; Nil, Buzzi & Bättig, 1984).

Given the current lack of clarity surrounding the construct of craving, this discourse purports that it may be necessary to consider a different approach. One approach would be to investigate further the construct of craving and set about defining and distinguishing the different types and circumstances of craving. In order to clarify the usefulness of the construct, Drummond et al. (2000) state that there is a need to determine the predictive power of craving, They describe four possibilities for the relationship between craving and relapse.

1-That they are completely unconnected random events. This is unlikely.

2-That craving is predictive of relapse but as yet measures are inadequate. In this case there is a need to develop better measures.

3- That craving is only predictive of relapse under certain circumstances. In this case there is a need to determine the circumstances.

4 – That the subjective experience of craving is not predictive of relapse, but autonomic and behavioural correlates or the mediators and moderators of craving are predictive. One could suggest that if this is the case then the construct is redundant.

Research would be better focussed on directly studying drug-using behaviours and more objective measures, which give a stronger base for research.

Objective measures such as autonomic reactivity and cognitive biases have been shown to relate to subjective craving in response to drug cues, in alcohol use (Glautier & Drummond, 1994), and in opiates, cocaine and nicotine (Carter & Tiffany, 1999). As long as this lack of clarity surrounds the construct of craving it may be more appropriate to use objective measures that are independent of craving to measure cue-reactivity and the likely predictors of continued use or relapse.

In order to gain a clearer understanding of the relationship between cues and drug use, it appears beneficial to set aside the construct of craving as a central issue. Instead empirical research, based on objective measures can be designed to investigate the relationship between cues and drug use. This approach is pertinent given the emphasis of some influential theories on the automatic nature of drug using behaviours (Robinson & Berridge, 1993; Tiffany, 1990). It has been suggested that craving is an epiphenomenon used post-hoc by drug users to explain drug use after an unconscious behaviour has been carried out (Tiffany, 1990). If this is the case, then the investigation of the construct of craving may be obscuring the underlying mechanism. The only way this will be determined is if more empirical research tests out the predictions made by Tiffany (1990) and Robinson & Berridge (1993), who suggest that craving is not available to conscious cognitive reflection. Prior to further investigation of this construct it is necessary to investigate empirically and possibly eliminate these suggested mechanisms.

There is no clear definition of craving and as a valid and reliable predictor of drug use, it lacks empirical support. To move this area forward, two approaches are possible. Either to unpack the construct of craving and come to a clearer

understanding of what substance users mean by their experience, or as this discourse purports, to work with more objective means, that directly access the relationship between drug associated cues and drug use. This discourse purports that the focus of research should be empirical testing of specific predictions from the currently influential theories. This will give a clearer understanding of the mechanism that may mediate responses to drug cues. When this is understood, research can be more clearly directed at the subjective experience of the individual within the context of this new information. At this point it should become clearer whether craving is either a limited linguistic concept or a clearly defined entity for which further reliable and valid measures can be designed.

Taking Research Forward

This review suggests that more objective measures are needed to investigate the area. Emerging cue reactivity measures such as startle reflexes, neuroimaging, cognitive processing (e.g. modified Stroop task), expressive behaviour, reinforcement paradigms and drug self-administration paradigms may prove to be of greater predictive power than subjective or autonomic measures (Sayette et al., 2000). A systematic top down approach is now needed in which specific predictions generated from current theories are tested to determine if these alternative measures will be useful indicators of the theoretically proposed mechanisms which link drug cues and drug use. Research now needs to focus on the empirical objective measures, e.g. startle reflexes, neuroimaging and cognitive processing that can be used to compare and contrast the proposed models, e.g. incentive-sensitisation model (Robinson & Berridge, 1993, information processing (Tiffany, 1990) and CSLT (Marlatt & Gordon 1985). This wide range of measures requires the specialist skills within a

multi-disciplinary approach. The role of psychology could be to focus on cognitive processing and the further investigation of cognitive biases.

Cognitive Biases

A specific model of cognitive biases in addiction has not yet been formed (McCusker, 2001). The desynchrony of self-reported intentions being so different to ongoing addictive behaviour is more understandable if cognitive biases mediating behaviour are seen to be at an automatic level of information processing. The individual is understood to express conscious intentions not to use substances, but behaviour may be driven by less accessible processes. This has been suggested in two of the currently influential theories, the incentive-sensitisation model (Robinson & Berridge, 1993), and the cognitive processing model (Tiffany, 1990). Research is needed to investigate the nature of the information processing which governs drug-use behaviour. Experimental methods that test the cognitive processes based on behavioural responses to addiction related cues (McCusker, 2001), appear to be the most effective way of measuring cognitive bias in this area. Measures of cognitive bias directly access propositions and processes that motivate ongoing behaviour, are more demand free in the absence of direct enquiry, and access aspects of cognition which are not accessible to introspective accounting (Stacey, 1997).

Clinical importance of investigating attentional bias

The clinical importance of determining the mechanism through which responses to drug cues occurs is crucial to offering specialist interventions. The absorption and allocation of attention by drug-related stimuli has been suggested to determine post-treatment drinking when faced with a high-risk situation (Marlatt & Gordon, 1985;

Rohsenow, Monti, Rubonis, Sirota, Niaura, Colby, Wunschel & Abrams 1994). For example, Sayette, Monti, Rohsenow, Gulliver, Colby, Sirota, Niaura, & Abrams, (1994) tested the cognitive strategies of alcoholic subjects. They determined that the presence of cues may disrupt alcoholic subjects' skill in refusing a drink (Sayette et al., 1994). If attentional resources are automatically drawn to drug-related cues then fewer resources are available for coping with the offer of a drink (Sayette et al., 1994). Further investigation of attentional bias is needed to determine if it predicts susceptibility to relapse (Monti, Rohsenow & Hutchinson, 2000) and to help develop strategies to overcome the effects of having attentional resources taken by drug-related material.

The conscious strategic allocation of attention towards potential cues has been suggested to assist dependent users in preventing relapse. Rohsenow et al. (1994) asked participants to rate, by self-report, how much their attention was drawn to alcoholic drinks. They found that those participants who reported more attention to the alcohol drank significantly less at follow-up. Monti et al. (2000) suggest that this is consistent with the information processing model (Tiffany 1990) and CSLT (Marlatt & Gordon 1985), which suggests that if effortful cognitive strategies are engaged to allocate attention then it allows for increased mobilisation of coping skills (Rohsenow et al., 1994). Tiffany (1990) proposes that when an obstacle interrupts the automatic sequence of drug-seeking behaviour, then effortful cognitive strategies are engaged to obtain the substance. It is at this point, when drug-seeking is engaging strategic cognitive processing that it is most accessible to the use of cognitive techniques described by Beck et al. (1993) to prevent ongoing drug-use. Further investigation of attentional bias in addiction may determine at what stage of processing the biases are most active. Robinson & Berridge (1993) suggest that bias

in attention may reflect the activation of the incentive-sensitisation system, therefore measures of attentional bias could be important in determining susceptibility to initial dependence and to relapse after abstinence (Lubman et al., 2000). Evidence of an attentional bias for drug-related stimuli has been found in dependent users of opiates, alcohol and nicotine (Gross, Jarvik, & Rosenblatt, 1993; Johnsen, Laberg, Cox, Vaksdal & Hugdahl, 1994; Lubman et al., 2000; Stetter, Ackermann, Bizer, Straube & Mann, 1995). The most robust of these research studies in terms of drawing conclusions about attentional bias, was the study by Lubman et al. (2000). They used a dot probe task in which drug-related and neutral stimuli were presented side by side on a computer screen to opiate dependent and control subjects. They found that opiate dependent subjects were faster at responding to probes that replaced drug-related pictures, showing that their attention had been orientated to these pictures over the others. This process was seen to reflect an attentional bias for drug-related stimuli which was not evident in the control subjects. Further research is needed to determine whether this bias is evident across levels of processing e.g. at an automatic level, and when more effortful cognitive processing is used. This may give some indication, which of the theories that has been discussed is the most accurate representation of the cognitive processing biases in drug users. It would also be useful to determine whether this bias is evident across all substances or whether it is more prominent in some than others. It may be that activation of attentional bias is different in abstinent and currently using individuals. It may be that sensitisation to incentive stimuli remains despite abstinence (Robinson & Berridge 1993) in which case attentional bias may have the potential to predict subjects who would be more susceptible to relapse.

Conclusions

Drug-related stimuli are acknowledged to have a role in the maintenance of and relapse to drug use. The mechanism through which they affect behaviour is unclear. A number of theories have suggested explanations for this process. Research in this area needs to find measures that can be used as indicators of the underlying processes, which have been proposed in the prominent theories. Investigation of the incentive-sensitisation model (Robinson & Berridge 1993) and the implications for cognitive clinical interventions may be helped by the investigation of cognitive biases. Research has shown that drug dependent subjects have an attentional bias for drug-related stimuli but as yet it is unclear whether that bias is consistent across levels of cognitive processing and across the time course of an addiction. Further work in this area is required to increase understanding and inform clinical interventions.

References

Abrams, D.B. (2000). Transdisciplinary concepts and measures of craving: commentary and future directions, Addiction, 95, 2, S237-S246.

Bandura, A. (1977). Self-efficacy: towards a unifying theory of behaviour change, Psychological Review, 84, 191-215.

Beach, H.D. (1957). Morphine addiction in rats. Canadian Journal of Psychology, 11, 104-112.

Beck, A.T., Wright, F.D., Newman, C.F. & Liese, B.S. (1993). Cognitive Therapy of Substance Abuse. New York: Guilford Press.

Bozarth, M.A. & Wise, R.A. (1981). Intracranial self-administration of morphine into the ventral tegmental area in rats. Life Science, 28, 551-555.

Bozarth, M.A. & Wise, R.A.(1983). Dissociation of the rewarding and physical dependence-producing properties of morphine. In L.S. Harris (Ed), Problems of drug dependence. pp. 171-177. Rockville, MD: National Institute of Drug Abuse.

Bradley, B.P., Phillips, G., Green, L. & Gossop, M. (1989). Circumstances surrounding the initial lapse to opiate use following detoxification. British Journal of Psychiatry, 154, 354-359.

Brandon, T.H. & Baker, T.B. (1992). The smoking consequences questionnaire: the subjective utility of smoking in college students, Psychological Assessment: A. Journal of Consulting and Clinical Psychology, 3, 484-491.

Burton, S. & Tifffany, S.T. (1997). The effect of alcohol consumption on craving to smoke. Addiction, 92, 15-26.

Carter, B.L. & Tiffany, S.T. (1999). Meta-analysis of cue-reactivity in addiction research, Addiction, 94, 327-340.

Cepeda-Benito, A. & Tiffany, S.T. (1996). The use of a dual-task procedure for the assessment of cognitive effort associated with smoking urges. Psychopharmacology, 127, 155-163.

Childress, A.R., McLellan, A.T., Ehrman, R. & O'Brien, C.P. (1988). Classically conditioned responses in opioid and cocaine dependence : a role in relapse, In B.A. Ray (Ed) Learning Factors in Substance Abuse, DHHS pulbication no. ADM-88-1576, pp. 25-43, Rockville, MD: Alcohol, Drug Abuse and Mental Health Administration.

Childress, A.R., McLellan, A.T. & O'Brien, C.P. (1984). Measurement and extinction of conditioned withdrawal-like responses in opiate dependent patients. NIDA Research Monograph, 49, pp. 212-219. Washington DC: US Government Printing Office.

Childress, A.R., McLellan, A.T. & O'Brien, C.P. (1986a). Abstinent opiate abusers exhibit conditioned craving, conditioned withdrawal and reduction in both through extinction, British Journal of Addiction, 81, 644-660.

Childress, A.R., McLellan, A.T., & O'Brien, C.P. (1986b). Conditioned responses in a methadone population: a comparison of laboratory, clinic and natural setting, Journal of Substance Abuse Treatment, 3, 173-179.

Childress, A.R., Ehrman, R., Rohsenow, D.J., Robbins, S.J. & O'Brien, C.P. (1992) Classically conditioned factors in drug dependence. In J.H. Lowinson, P. Ruiz, R.B. Millman & J.G. Langrod, (Eds), Substance Abuse: A Comprehensive Textbook, pp.56-69 Baltimore: Williams and Wilkins.

Christiansen, B.A., Smith, G.T., Roehling, P.V. & Goldman, M.S. (1989). Using alcohol expectancies to predict adolescent drinking behaviour at one year. Journal of Consulting and Clinical Psychology, 57, 93-99.

Cooney, N., Gillespie, R.A., Baker, L.H. & Kaplan, R.F. (1987). Cognitive changes after alcohol cue exposure. Journal of Consulting and Clinical Psychology, 55, 150-155.

Curran, D. (1999). Evidence of memory biases for positive alcohol expectancies in problem drinkers. Paper presented at the Annual Conference of The British Psychological Society, Belfast, UK.

Davis, W.M., & Smith, S.G. (1987). Conditioned reinforcement as a measure of the rewarding properties of drugs. In M.A. Bozarth, (Ed), Methods in Assessing Reinforcing Properties of Abused Drugs, pp. 199-209. New York: Springer-Verlag.

Dawe, S., Gerada, C., Russell, M.A.H. & Gray, J.A. (1995). Nicotine intake in smokers increases following a dose of haloperidol. Psychopharmacology, 117, 110-115.

Dawe, S., Powell, J., Richards, D., Gossop, M., Marks, I., Strang, J. & Gray, J.A. (1993). Does post-withdrawal cue exposure improve outcome in opiate addiction? A controlled trial. Addiction, 88, 1233-1245

Deneau, G.A., Yanagita, T. & Seevers, M.H. (1969). Self-administration of psychoactive substances by the monkey: A measurement of psychological dependence. Psychopharmacologia, 16, 30-48.

de Wit, H. & Stewart, J. (1981). Reinstatement of cocaine-reinforced responding in the rat. Psychopharmacology, 75, 134-143.

de Wit, H. & Stewart, J. (1983). Drug reinstatement of heroin-reinforced responding in the rat. Psychopharmacology, 79, 29-31.

Drummond, D.C. (2000). What does cue-reactivity have to offer clinical research? Addiction, 95, S129-S144.

Drummond, D.C. (2001). Theories of drug craving, ancient and modern, Addiction, 96, 33-46.

Drummond, D.C., Litten, R.Z., Lowman, C. & Hunt, W.A. (2000). Craving research: future directions. Addiction, 95, S247-S255.

Drummond, D.C., Tiffany, S., Glautier, S.P. & Remington, B. (1995). Addictive Behaviour: cue exposure theory and practice. Chichester: John Wiley & Sons.

Elash, C.A., Tiffany, S.T. & Vrana, S.R. (1995). Manipulation of smoking urges and affect through a brief-imagery procedure: Self-report, psychophysiological and startle probe responses. Experimental and Clinical Psychopharmacology, 3, 156-162.

Feldman, J.M. & Lynch, J.G. (1988). Self-generated validity and other effects of measurement on belief, attitude, intention and behaviour, Journal of Applied Psychology, 73, 421-435.

Fennessy, M.R. & Ortiz, A. (1968). The behavioural and cardiovascular actions of intravenously administered morphine in the conscious dog. European Journal of Pharmacology, 3, 177-185.

Fischman, M.W. & Foltin, R.W. (1992). Self-administration of cocaine by humans: a laboratory perspective, In: G.R. Bock & J.Whelan (Eds), Cocaine: scientific and social dimensions, CIBA Foundation Symposium no. 166, pp. 165-180. Chichester, UK: Wiley.

Fischman, M.W., Foltin, R.W., Nestdt, G. & Pearlson, G.D. (1990). Effects of desipramine maintenance on cocaine self adminstration by humans. Journal of Pharmacology and Experimental Therapies, 253, 760-770.

Gerber, G.J. & Stretch, R. (1975). Drug-induced reinstatement of extinguished self-administration behaviour in monkeys. Pharmacology, Biochemistry and

Behaviour, 3, 1055-1061.

Glautier, S., & Drummond, D.C. (1994). Alcohol dependence and cue reactivity. Journal of studies on Alcohol, 55, 224-229.

Goldberg, S.R. (1976). Stimuli associated with drug injections as events that control behaviour. Psychopharmacological Review, 27, 325-340.

Gross, T.M., Jarvik, M.E. & Rosenblatt, M.R. (1993). Nicotine abstinence produces content-specific Stroop interference. Psychopharmacology, 110, 333-336.

Gross, J. & Stitzer, M.L. (1989). Nicotine replacement: Ten-week effects on tobacco withdrawal symptoms. Psychopharmacology, 98, 334-341.

Johnsen, B.H., Laberg, J.C., Cox, W.M., Vaksdal, A. & Hugdahl, K. (1994). Alcoholic subjects' attentional bias in the processing of alcohol-related words. Psychology of Addictive Behvaiours, 8, 111-115.

Jones, B. & McMahon, J. (1996). A comparison of positive and negative alcohol expectancy and value and their multiplicative composite as predictors of post-treatment abstinence survivorship. Addiction, 91, 89-99.

Koob, G.F. (1992). Drugs of abuse: Anatomy, pharmacology and function of reward pathways. Trends in Pharmacological Science, 13, 177-184.

Koob, G.F. (1996). Drug addiction: The yin and yang of hedonic homeostasis. Neuron, 16, 893-896.

Koob, G.F. & Bloom, F.E. (1988). Cellular and molecular mechanisms of drug dependence. Science, 242, 715-723.

Koob, G.F., Markou, A., Weiss, F. & Schulteis, G. (1993). Opponent process and drug dependence: neurobiological mechanisms. Seminars of neuroscience, 5, 351-358.

Koob, G.F., Stinus, L., Le Moal, M. & Bloom, F.E. (1989). Opponent process theory of motivation: neurobiological evidence from studies of opiate dependence. Neuroscience and Biobehaviour Reviews, 13, 135-140.

Lamb, R.J., Preston, K.L., Schindler, C.W., Meisch, R.A., Davis, F., Katz, J.L., Henningfield, J.E. & Goldberg, S.R. (1991). The reinforcing and subjective effects of morphine in post-addicts: A dose-response study. Journal of Pharmacology and Experimental Therapies, 259, 1165-1173.

Legarda, J.J., Bradley, B.P. & Sartory, G. (1990). Effects of drug-related cues in current and former opiate users. Journal of Psychophysiology, 4, 25-31.

Leung, K.S. & McCusker, C.G. (1999). Accessibility and availability of smoking-related associations in smokers. Addiction Research, 7, 213-226.

Levine, D.G. (1974). Needle freaks: Compulsive self-injections by drug users. American Journal of Psychiatry, 131, 297-300.

Littman, G.K., Stapleton, J. & Oppenheim, A.N. (1983). Situations related to alcoholism relapse. British Journal of Addiction, 78, 381-389.

Litz, B.T., Payne, T.J. & Colletti, G. (1987). Schematic processing of smoking information by smokers and never-smokers. Cognitive Therapy and Research, 11, 301-313.

Logan, G.D. (1988). Toward an instance theory of automatisation. Psychological Review, 95, 492-527.

Lubman, D.I., Peters, L.A., Mogg, K., Bradley, B.P. & Deakin, J.F.W. (2000). Attentional bias for drug cues in opiate dependence, Psychological Medicine, 30, 169-175.

Ludwig, A. (1989). The mystery of craving. Alcohol Health Research World, 11, 12-17.

Marlatt, G.A. (1978). Relapse prevention: theoretical rationale and overview of the model, In G,A, Marlatt & J. Gordon (Eds), Relapse Prevention: maintenance strategies for addictive behaviours, pp. 3-70. New York: Guilford Press.

Marlatt, G.A. & Gordon, J.R. (1980). Determinants of relapse: Implications for the maintenance of behavior change. In P.O. Davidson & S.M. Davidson (Eds), Behavioral medicine: changing health life-styles. pp. 410-452. New York: Brunner/Mazel.

Marlatt, G.A. & Gordon, J.R. (1985). Relapse Prevention: maintenance strategies for addictive behaviours, New York: Guilford

Maude-Griffin, P.M. & Tiffany, S.T. (1996). Production of smoking urges through imagery: The impact of affect and smoking abstinence. Experimental and Clinical Psychopharmacology, 4, 198-208.

Meyer, R.E. & Mirin, S.M. (1979). The Heroin Stimulus: implications for a theory of addiction. New York: Plenum.

McCusker, C.G. (2001). Cognitive biases and addiction: an evolution in theory and method. Addiction, 96, 47-56.

McCusker, C.G. & Gettings, B. (1997). Automaticity of cognitive biases in addictive behaviours: further evidence with gamblers. British Journal of Clinical Psychology, 36, 543-554.

McCusker, C.G., Leung, K.S. & Armstrong, A. (1999). The return of the "unconscious" in psychology: examples from addiction research. Paper presented at the Annual Conference of The British Psychological Society, Belfast, UK.

McCusker, C.G., Roberts, G., Douthwaite, J. & Williams, E. (1995). Teenagers and illicit drug use-expanding the "user vs. non-user" dichotomy. Journal of

Community and Applied Social Psychology, 5, 221-241.

Meyer, R.E. & Mirin, S.M. (1979). The heroin stimulus. New York: Plenus Press.

Miller, N.S. & Gold, M.S. (1994). Dissociation of 'conscious desire' (craving) from and relapse in alcohol and cocaine dependence. Annals of Clinical Psychiatry, 6, 99-106.

Monti, P.M., Rohsenow, D.J., & Hutchinson, K.E. (2000). Toward bridging the gap between biological, psychobiological and psychosocial models of alcohol craving. Addiction, 95, S229-S236.

Nemeth-Coslett, R. & Henningfield, J.E. (1986). Effects of nicotine chewing gum on cigarette smoking and subjective and physiologic effects, Clinical and Pharmacological Therapies, 39, 625-630.

Niaura, R. (2000). Cognitive social learning and related perspectives on drug craving. Addiction, 95, S155-S163.

Nil, R., Buzzi, R. & Bättig, K. (1984). Effects of a single doses of alcohol and caffeine on cigarette smoke puffing behavior. Pharmacology, Biochemistry and Behavior, 20, 583-590.

Nisbett, R.E. & Wilson, T.D. (1977). Telling more than we can know: Verbal reports on mental processes. Psychological Review, 84, 231-259.

O'Brien, C.P., Chaddock, B., Woody, G. & Greenstein, R. (1974). Systematic extinction of narcotic drug use using narcotic antagonists. Psychosomatic Medicine, 36, 458.

O'Brien, C.P., Childress, A.R., Ehrman, R. & Robbins, S.J. (1998). Conditioning factors in drug abuse: can they explain compulsion? Journal of Psychopharmacology, 12 (1), 15-22.

O'Brien, C.P., Childress, A.R., McLellan, A.T. & Ehrman, R. (1992). A learning

model of addiction. In C.P. O'Brien & J.H. Jaffe (Eds.), Addictive States, pp.157-177. New York: Raven Press.

O'Brien, C.P., Greenstein, R., Ternes, J., McLellan, T. & Grabowski, J. (1979). Unreinforced self-injections: effects of rituals and outcome in heroin addicts. NIDA Research Monograph, 27, 275-281.

O'Brien, C.P., Greenstein, R., Ternes, J., McLellan, A.T., Grabowski, J. (1980) Unreinforced self injections: effects on rituals and outcome in heroin addicts. NIDA Research Monograph 27, pp.275-281, Washington, DC: US Government Printing Office.

O'Brien, C.P., O'Brien, T.J. & Mintz, J. (1975) Conditioning of narcotic abstinence symptoms in human subjects. Drug Alcohol Dependence, 1, 115-123.

O'Brien, C.P., Testa, T., O'Brien, T.J., Brady, J.P. & Wells, B. (1977). Conditioned narcotic withdrawal in humans. Science, 195, 1000-1001.

Plant, M. & Plant, M. (1992). Risk-takers. London: Tavistock:Routledge

Posner, M.I. & Snyder, C.R.R. (1975). Attention and cognitive control. In R.L. Solso (Ed), Information processing and cognition: The Loyala symposium. pp. 55-85. New York: John Wiley.

Powell, J., Gray, J. & Bradley, B.(1993) Subjective craving for opiates: Evaluation of a cue exposure protocol for use with detoxified opiate addicts. British Journal of Clinical Psychology, 32, 39-53.

Robinson, T.E., & Berridge, K.C. (1993). The neutral basis of drug craving: an incentive-sensitisation theory of addiction. Brain Research Review, 18, 247-291.

Robinson, T.E., & Berridge, K.C. (2000). The psychology and neurobiology of addiction: an incentive-sensitization view. Addiction, 95, 2, S91-S117.

Robinson, T.E., & Berridge, K.C. (2001). Incentive-sensitization and addiction. Addiction, 96, 103-114.

Rohsenow, D.J., Monti, P.M., Rubonis, A.V., Sirota, A.D., Niaura, R.S., Colby, S.M., Wunschel, S.M. & Abrams, D.B. (1994). Cue-reactivity as a predictor of drinking among male alcoholics. Journal of Consulting and Clinical Psychology, 62, 620-626.

Sayette, M.A., Monti, P.M., Rohsenow, D.J., Gulliver, S.B., Colby, S.M., Sirota, A.D., Niaura, R. & Abrams, D.B. (1994). The effects of cue exposure on reaction time in male alcoholics, Journal of Studies on Alcohol, 55, 629-633.

Sayette, M.A., Shiffman, S., Tiffany, S.T., Niaura, R.S., Martin, C.S. & Shadel, W.G. (2000). The measurement of drug craving. Addiction, 95, S189-S210.

Schafer, J. & Brown, S.A. (1991). Marijuana and cocaine effect expectancies and drug use patterns. Journal of Consulting and Clinical Psychology, 59, 558-565.

Shiffrin, R.M. & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. Psychological Review, 84, 127-190.

Schulteis, G. & Koob, G.F. (1996). Reinforcement processes in opiate addiction: A homeostatic model. Neurochemical Research, 21, 1437-1454.

Schwartz, A.S. & Marchok, P.L. (1976). Persistence of relapse of morphine-seeking behaviour in rats: The relative role of certain biological variables. Psychopharmacology, 47, 149-152.

Sher, K.J., Wood, M.D., Wood, P.K. & Raskin, G. (1996). Alcohol outcome expectancies and alcohol use: a latent variable cross-lagged panel study. Journal of Abnormal Psychology, 105, 561-574.

Siegel, S. (1989). Pharmacological conditioning and drug effects, In A.J. Goudie & M.W. Emmett-Oglesby (Eds), Psychoactive Drugs: Tolerance and Sensitisation, pp. 115-180, Clifton, HJ: Humana Press.

Solomon, R.L. & Corbit, J.D. (1979). Opponent process theory of acquired movement: I. Temporal dynamics of affect. Psychological Review, 81, 119-145.

Stacy, A.W. (1997). Memory activation and expectancy as prospective predictors of alcohol and marijuana use. Journal of Abnormal Psychology, 106, 61-73.

Stacy, A.W., Newcomb, M.D. & Bentler, P.M. (1991). Cognitive motivation and problem drug use: a 9-year longitudinal study. Journal of Abnormal Psychology, 100, 502-515.

Stetter, F., Ackermann, K., Bizer, A., Straube, E.R. & Mann, K. (1995). Effects of disease-related cues in alcoholic inpatients: Results of a controlled 'Alcohol Stroop' study. Alcoholism: Clinical and Experimental Research, 19, 593-599.

Stewart, J., de Wit, H. & Eikelboom, R. (1984). Role of unconditioned and conditioned drug effects in the self-administration of opiates and stimulants, Psychological Review, 91, 251-268.

Ternes, J.W., O'Brien, C.P., Grabowski, J., Wellerstein, H., Jordon-Hayes, J. (1980) Conditioned drug responses to naturalistic stimuli: problems of drug dependence. NIDA Research Monograph 27, pp. 282-288 Washington DC: US Government Printing Office,

Tiffany, S.T. (1990). A cognitive model of drug urges and drug-use behaviour: role of automatic and nonautomatic processes. Psychological Review 97, 147-168.

Tiffany, S.T. & Carter, B.L. (1998). Is craving the source of compulsive drug use? Journal of Psychopharmacology, 12, 1, 23-30.

Tiffany, S.T., Carter, B.L. & Singleton, E.G. (2000) Challenges in the manipulation, assessment and interpretation of craving relevant variables, Addiciton, 95, S177-S187.

Tiffany, S.T. & Drobes, D.J. (1990). Imagery and smoking urges: the manipulation of affective content. Addictive Behaviour, 15, 531-539.

Tiffany, S.T. & Hakenewerth, D.M. (1991). The production of smoking urges through an imagery manipulation: Psychophysiological and verbal manifestations. Addictive Behaviour, 16, 389-400.

Volavka, L., Levine, R., Feldstein, S. & Fink, M. (1974). Short-term effects of heroin in man. Is EEG related to behaviour. Archives of General Psychiatry, 30, 677-681.

Wallace, B.C. (1989). Psychological and environmental determinants of relapse in crack cocaine smokers. Journal of Substance Abuse Treatment, 6, 95-106.

Wei, E.T. (1981). Enkephalin analogues and physical dependence. Journal of Experimental Pharmacology and Therapeutics, 216, 12-18.

Weinstein, A., Feldtkeller, B., Malizia, A., Wilson, S., Bailey, J. & Nutt, D.J. (1998). Integrating the cognitive and physiological aspects of craving, Journal of Psychopharmacology, 12, 31-38.

Wikler, M.D. (1948). Recent progress in research on the neurophysiologic basis of morphine addiction. American Journal of Psychiatry, 105, 329-338.

Wise, R.A. (1988). The neurobiology of craving: Implications for the understanding and treatment of addiction. Journal of Abnormal Psychology, 97, 118-132.

Wise, R.A. & Bozarth, M.A. (1987). A psychomotor stimulant theory of addiction. Psychological Review, 94, 469-492.

Woods, J.H. & Schuster, C.R. (1971). Opiates as reinforcing stimuli. In T. Thompson

& R. Pickens (Eds), Stimulus properties of drugs. New York: Appleton-Century-Crofts.

Empirical Paper

Attentional bias in opiate dependence:

Manipulation of stimulus duration

Submitted to British Journal of Clinical Psychology (See Appendix 1)

Where notes to contributors for this journal have differed from guidelines for the production of the thesis, the instructions from the doctoral course in clinical psychology have been followed.

Attentional Bias in Opiate Dependence:

Manipulation of Stimulus Duration

Lisa Frankland

Department of Psychology

University of Southampton

Key words:

Addiction, Opiate Dependence, Attentional Bias

Running Title:

Attentional bias in opiate dependence

Correspondence should be addressed to Lisa Frankland, c/o Department of Psychology, University of Southampton, Highfield, Southampton, SO17 1BJ, United Kingdom

(Telephone: 02380-5952588; Fax: 02380-592588)

Abstract

Objectives

Attentional bias in opiate dependence was investigated to examine whether it is active at a relatively automatic speed of processing or whether it was evident at a speed that allowed time for the use of more strategic cognitive processing.

Design

A mixed design was used to explore differences between the experimental group, participants dependent on opiates (n=19), and a group of matched controls (n=20).

Methods

Participants were tested on two tasks, a dot probe task with three within-subject factors, (exposure lengths of 200ms, 500ms and 1500ms), and a modified Stroop task with two within-subject factors (Drug-related words and control words). The varied exposure lengths measured different levels of processing of the attentional bias.

Results

The opiate dependent group showed a significant attentional bias for drug-related information presented at 200ms. There was no evidence of an attentional bias at 500 or 1500ms. The interaction between the groups and word cards was not significant.

Conclusions

An attentional bias was found at 200ms in the dot probe task. This is suggested to be indicative of an automatic bias in attentional processing which may reflect activation of the incentive-sensitisation mechanism proposed by Robinson & Berridge (1993). These results however must be interpreted with caution as differences were found between the groups on measures of affect , verbal ability and age. Suggestions are made as to how this may be overcome in future research.

Introduction

People's attention is drawn to things in the environment which attract or concern them. Over time, a bias in cognitive processing may develop which guides attention to these stimuli over all others. This is known in psychological literature as an attentional bias. In the field of addiction, with repeated use of a substance and the development of dependence, attention is selectively drawn to things associated with that substance. This attentional bias has been shown to be evident in individuals dependent on nicotine, opiates, and alcohol (Gross, Jarvik & Rosenblatt, 1993; Johnsen, Laberg, Cox, Vaksdal & Hugdahl, 1994; Lubman, Peters, Mogg, Bradley & Deakin, 2000; Sharma, Albery & Cook 2001; Stetter, Ackermann, Bizer, Straube & Mann, 1995).

A number of psychological theories of drug dependence have suggested mechanisms through which this attentional bias may act on drug-relevant stimuli present at the time of consumption. Initially models which theorised about the role of stimuli in continued drug use were based on simple conceptualisations of conditioning theory. They proposed that stimuli became associated with drug effects through negative (Wikler, 1948), and positive reinforcement (Stewart, de Wit & Eikelboom, 1984). Attempts to extinguish the conditioning and prevent further drug use have been largely unsuccessful (Niaura, Abrams, Shadel, Rohsenow, Monti & Sirota, 1999). This indicates that the process of conditioning is not the single motivating factor in the relationship between drug-related cues and continued drug use or relapse. Theories have since advanced to integrate knowledge from neurobiology (e.g. Robinson & Berridge, 1993), and cognitive psychology (e.g. Marlatt & Gordon, 1985; Tiffany, 1990). A common theme that emerges from these

theories is the importance attributed to attention being drawn to drug-related stimuli over all other environmental stimuli.

With the activation of an attentional bias, stimuli associated with the substance are more likely to be noticed in the environment. These stimuli become more salient and consume processing resources. The individual is at increased risk of intrusive thoughts and becomes preoccupied with the drug of dependence. This may make it more difficult for the individual to engage in non-drug related activities and is likely to increase subjective desire for the drug and urges to engage in drug taking. This has implications for clinical interventions aimed at helping the dependent individual to cut down or stop their drug use.

The psychological model that has most influenced current clinical practice is cognitive social learning theory (CSLT) (Marlatt & Gordon, 1985). This model proposes that in high-risk situations in which cues are likely to trigger drug use, the dependent individual has the choice whether or not to use. It states that the likelihood of their using drugs depends on an interaction between their expectations about self-efficacy and the consequences of using. If they have positive expectations about the effects of using and a low self-efficacy then they are likely to use. Within this model the decisional process is said to be affected by cognitive biases (Beck, Wright, Newman & Liese, 1993). Attentional bias is one example of this. Beck et al. (1993) state that as attention is drawn to things in the environment that remind the users of their dependence, cognitive techniques such as thought suppression and distraction must be employed to counteract this effect.

During the last decade, researchers have begun to investigate the area of cognitive bias in addiction. A recent review of the research indicates that the allocation of attention during the sequence of drug seeking may not be open to

conscious cognitive reflection (McCusker, 2001). This is in line with the prediction of the incentive-sensitisation model (Robinson & Berridge, 1993) which suggests that attentional bias occurs at an automatic level of processing. If this is true then cognitive intervention, which relies on the recall of the motivations behind drug-seeking behaviour, may need to be adapted to take account of the automatic nature of the behaviour. Robinson and Berridge (1993,2000,2001) suggest that attentional bias is a reflection of the activation of their proposed incentive-sensitisation system. Their theory posits that drug-related stimuli are perceived as salient stimuli that are 'wanted' by the individual and demand attention. Drugs act on the brain within the mesolimbic dopamine system, which is involved in the process of incentive motivation and reward, and is responsible for drug seeking and behaviour. Through the process of neuroadaptation the brain reward system becomes hypersensitive (sensitised) to drugs and drug associated stimuli. The degree of activation of the system is the key mechanism that causes drugs to be perceived as attractive and directs behaviour towards drug consumption. The environment modulates sensitisation, so susceptibility varies according to genetic, hormonal and experiential factors. Expression of sensitisation is context specific so it would be expected to be strongest in contexts in which the drugs had previously been used. There is individual variation in the extent to which drug associated stimuli activate the system, grab attention and become 'wanted'. Measures of attentional bias would provide an index of this activation. This process is suggested to occur at an automatic level of processing of which the individual would not necessarily be consciously aware. Behaviour may therefore be automatically driven towards drug seeking and consumption with little engagement of strategic cognitive processing.

The automatic regulation of drug-seeking behaviour is suggested by the cognitive processing model (Tiffany, 1990). Tiffany suggests that over time drug seeking behaviours, because they are highly learned and repeated frequently, become a stereotypical automatic sequence. As performance of this sequence involves little cognitive effort it does not interfere with other automatised behaviour. The sequence is triggered by stimuli that have become associated with it and due to the nature of the sequence it is difficult to prevent when triggered, and extremely difficult to interrupt. This model suggests that cognitive regulation of drug seeking behaviour occurs largely outside conscious awareness unless an obstacle obstructs its completion. Only when the sequence is interrupted by an unexpected incident does the process involve effortful, strategic cognitive processing in order to continue with the goal of drug consumption.

Each of the models discussed proposes a mechanism in which stimuli associated with drug use capture attention and guide behaviour towards drug seeking and consumption. Robinson & Berridge (1993) suggest that the bias in attention is automatically activated, whereas the cognitive model (Beck et al. 1993) assumes that the bias is accessible at a more effortful, strategic level of cognitive processing. Tiffany (1990) suggests that a non-automatic attentional bias occurs under conditions of drug deprivation when the automatic sequence of drug-seeking has been interrupted.

There have been a number of studies in the field of addiction, which have shown the presence of an attentional bias toward stimuli associated with the dependence. Interference was shown on modified versions of the colour naming Stroop task, indicating an attentional bias in problem drinkers and nicotine smokers (Gross, Jarvik & Rosenblatt, 1993; Johnsen et al., 1994; Sharma et al., 2001; Stetter,

Ackermann, Bizer, Straube & Mann, 1995). The modified Stroop task requires participants to name the ink colour of words relevant to drug taking. The task is based on the assumption that attentional resources are occupied by the drug related word meaning and this interferes with the individual's ability to carry out the colour naming task. Similar results were found with dependent drinkers on a reaction time task. While engaged in the task, the drinkers were exposed to auditory stimuli. When auditory material was related to drinking, they found that there was an automatic diversion of cognitive resources from intentional cognitive activity to information related to their addiction (Sayette & Hufford, 1994; Sayette, Monti, Rohsenow, Gulliver, Colby, Sirota, Niaura & Abrams, 1994).

Although these tasks purport to measure attention, there is a possibility that interference on the modified Stroop task may occur at either the attentional or response stage of processing, (Lubman et al., 2000). The effect may be due to difference in the process of selecting and generating a response (MacLeod, 1991; Stirling, 1979) rather than the allocation of attention. Other research has used a dot probe task to overcome this difficulty. The probe detection task has been used previously to detect attentional bias in smokers (Bradley, Mogg, Wright & Bates, unpublished) and in opiate users (Lubman et al., 2000). In this task pairs of pictures, a drug-related stimulus and a matched control picture are presented side by side on a computer screen. They are replaced by a dot probe in the location of one of the pictures. The participant is then asked to indicate with a response box which side the probe was on. The computer records response times. As response times are faster for stimuli that appear in an attended rather than unattended visual field (Posner, Snyder, & Davidson, 1980) response time can be used to indicate which picture is being attended to. This task indicates whether attention is drawn to drug-related stimuli

over neutral stimuli as would be expected with an attentional bias. It is now well established that attentional bias does exist across populations of dependent drug users. The mechanism through which this occurs has not yet been determined. Current theories suggest different mechanisms, which need to be tested against one another.

Investigation of the time course of the attentional bias to drug stimuli, may show whether the bias is in initial automatic orientation to drug cues, as predicted by Robinson & Berridge (1993), or whether it takes effect through more strategic cognitive processing mechanisms. The study by Lubman et al. (2000) which showed attentional bias in opiate dependent participants presented the stimuli for 500ms which was not able to indicate the nature of the bias, as this stimulus duration is long enough to allow either automatic or strategic processing. It remains unclear whether the observed bias is mediated by automatic processes or by effortful strategic processes. Another study attempted to study the processing level of the bias by varying the presentation of word cues to opiate dependent participants (Franken, Kroon, Wiers & Jansen, 2000). This study used word presentations and asked participants to identify real words from nonsense words. They found an attentional bias for stimuli presented supraliminally when letter strings remained on the screen until participants responded. They found no attentional bias in a masked Stroop task, which presented words at 28ms, which is suggested to be below the perception threshold (Bradley, Mogg & Millar, 1996). Further research is needed in this area because there is a growing recognition that attentional bias should be assessed with stimuli as ecologically valid as possible, such as pictures rather than single word stimuli (Lubman et al., 2000).

The research that is reported in this article used a dot probe task to assess attentional bias in opiate dependence. The aim was to investigate, through the manipulation of stimulus duration, whether the attentional bias to drug cues is more prevalent at a relatively automatic level of processing, 200ms, or at a level that allows time for more strategic processing, 1500ms. Lubman et al. (2000) found attentional bias at 500ms but at this time presentation it is unclear what process is in action.

It was predicted that on the dot probe task, opiate dependent participants would have faster response times to dots that appear in the same position as drug associated stimuli because their attention would be drawn to these pictures. Variation of the stimuli presentation time would indicate whether the attentional bias is at an automatic level of processing or whether it engages more strategic cognitive processing. A modified Stroop task was included as secondary interest to allow for comparisons between measures of attentional bias. It was predicted that on the modified Stroop task, opiate dependent participants would be slower to name the words associated with drugs because they would have difficulty ignoring the drug-related information. The Stroop task is a relatively crude measure of cognitive interference and it cannot be interpreted as a clear reflection of an attentional bias. Colour-naming interference may arise, not only at the input stage but also at the response selection stage (MacLeod, 1991). Relying on interference by drug related information may not reflect orientation of attention to the stimulus, but may be due to distraction from task-irrelevant thoughts or increased arousal (MacLoed, Matthews & Tata, 1986). The task which is of primary interest therefore is the dot-probe which overcomes these difficulties.

Hypotheses

1. It was predicted from Robinson & Berridge (1993) that a significant bias for drug cues would be evident at 200ms in the opiate dependent group reflecting a bias at an automatic processing level.
2. An attentional bias was predicted for drug cues in the opiate dependent group at 500ms following the findings of Lubman et al. (2000).
3. If an attentional bias is mediated by effortful cognitive strategies, then it should be found in the longer stimulus exposure duration of 1500ms.
4. It was predicted on the modified Stroop task, that participants in the dependent group would take significantly longer to name the colours of drug related words than control words. This effect was not predicted in the control group. An interaction between group and word type was therefore predicted on colour-naming times.

Method

Design

A mixed design was used. Between groups comparison was made between the experimental group, participants dependent on opiates (n=19), and a group of matched controls (n=20). In the dot-probe task attentional bias scores were used as the dependent variable and within-group comparison was made across exposure duration. In the modified Stroop task participants were all presented with drug related and control stimuli enabling within group comparison.

Participants

Experimental Group: Participants were recruited from two community drug and alcohol teams within an area that covers two cities and some rural communities. This

group was recruited either by responding to written information left in waiting rooms, or they chose to take part having been given information by their key-worker. A copy of the information sheet can be found in appendix 3. The experimental group consisted of 19 participants, 4 females and 15 males. All participants met the ICD-10 criteria for opioid dependence (World Health Organisation, 1992) with a past history of intravenous heroin use. They were on a stable dose of methadone with no dependence on other illicit or prescribed medication. People with a history of major psychiatric illness, significant head injury or significant physical health problems were not included. Four participants who completed the research were excluded on the grounds that they primarily smoked heroin and had injected less than five times. All of these participants reported a dislike of injecting practices and this may have affected their response to injecting related stimuli for these reasons.

Control Group: All clinical staff across the two drug and alcohol teams were given written information on the research (appendix 4). The 20 staff who volunteered to take part formed the control group. It consisted of 11 females and 9 males, all of whom had experience of working in addiction and were familiar with drug related material. As in Lubman et al. (2000) this control group was used to match for knowledge of drug paraphernalia and associated language. Matching for familiarity is important because unfamiliarity with such material might elicit an attentional bias in the control group due to the novelty and potentially shocking nature of the stimuli.

Materials

Questionnaire Measures

All participants were asked to complete measures of affect and verbal ability as follows:

Hospital Anxiety and Depression Scale (HAD), (Zigmond & Snaith, 1983), This was used to measure anxiety and depression across the two groups. The HAD was chosen because it has good face validity and is quick and easy to administer. It has good psychometric properties based on medical out-patients, (Zigmond & Snaith, 1983), although it has not been standardised specifically on out-patient drug users.

Speilberger State Trait Anxiety Inventory, (STAI), (Speilberger, Gorsuch, Lushene, Vagg, & Jacobs,1983). State measures of anxiety were taken to determine if either has an effect on attentional bias. The STAI has been used extensively as a psychological research tool (Speilberger, 1989) and has been shown across studies to have good psychometric properties across various populations (Speilberger, 1989).

Mill Hill Vocabulary Scale (alternate version) (Raven, 1965) A shortened version of the synonyms section of the Mill Hill vocabulary scale was used to match the groups for current verbal ability. A copy of this test can be found in appendix 5. This alternate item version of the multiple-choice scale was chosen for ease and speed of administration. As this test has not been standardised it was included primarily as a comparison between groups.

Participants in the opiate dependent group were also asked to fill out questionnaires on their drug use. They were administered as follows.

Use of opiates, A series of questions on current and past drug use and route of administration (appendix 6) were given verbally. This set of questions was put together by the experimenter to obtain information for within group comparisons.

Severity of Dependence Scale, (SDS), (Gossop, Darke, Griffiths, Hando, Powis, Hall & Strang, 1995). This is a short and easily administered scale, which measures psychological components of dependence (appendix 7). The psychometric properties of the scale have been shown to be good across classes of drugs, including opiates, in Britain and Australia (Gossop et al., 1995). It was used to determine if dependence is correlated with attentional bias.

Short Opiate Withdrawal Scale (SOWS), Gossop (1990) Items on this scale reflect symptoms of recognised physiological opiate withdrawal (appendix 8). It was used to determine if any of the participants were in physical discomfort. According to the cognitive processing model (Tiffany, 1990) a state of acute withdrawal would represent an interruption in the sequence of drug-seeking and may engage a different cognitive processing system.

Craving Measure, A single item likert scale was used to measure craving before and after the experimental tasks (appendix 9). Although single item measures are limited in their measurement of this complex phenomenon (Abrams, 2000), the function of the questionnaire in this experiment was to assess whether the experiment had affected participants desire to use. Single item measures have been shown to be highly reliable across repeated administration (Monti, Rohsenow & Hutchinson, 2000), and strong relationships have been found between single item ratings and other theoretically relevant variables (Rohsenow, Monti, Abrams, Rubonis, Niaura, Sirota & Colby, 1992).

Experimental Tasks

Two experimental paradigms were used to measure attentional bias, a modified Stroop colour-naming task, and a dot probe detection task.

The modified Stroop task: Colour Naming: Participants were presented with three cards on which words were written in four columns, with 24 words per column. The words appeared in four different colours, red, blue, yellow and green. The practice card listed names of animals. The words used were: *goat, donkey, horse, badger, elephant, zebra, hedgehog, chicken, eagle, otter, cat, and sparrow.* An experimental card used words associated with illicit opiate use. The words used were: *syringe, dealer, craving, gouch, works, needle, methadone, heroin, gear, fix, score, and smack.* A control card listed household words matched for length and frequency with the drug-related words. The words used were: *cellar, furniture, lounge, divan, wok, kettle, suite, clock, cupboard, cutlery, sofa, teacup.* The participant was asked to ignore the content of the word and name the colour in which the word was printed. A copy of the instructions can be found in appendix 10.

The dot probe task: A sub-set of the pictorial stimuli prepared by Lubman et al. (2000) was used. This consisted of 14 drug related pictures showing drug related paraphernalia and scenes of an addict 'cooking up' and injecting heroin, and a set of matched control pictures of isolated items from a children's building game and an unidentified person building a model railway. Six drug-related and matched control pictures were eliminated from the original set in order to keep the task under 15 minutes long. Seven pairs of pictures of neutral household stimuli were used on filler trials. All the photographs were taken in the same room, from similar perspectives and resized to the same dimensions (100mm length x 90mm high), using a computer and scanner. The pictures were presented on a 13-inch computer monitor using an Opus Pentium 75 computer. MEL2 software (Micro Experimental Laboratory, version 2; Schneider, 1995). A parallel port two button MEL box was used to record responses.

Procedure

Ethical approval was obtained from the appropriate ethical bodies. A copy of this can be found in Appendix 2. Participants met with the investigator for one session, which took up to 45 minutes. They were introduced to the study and any questions regarding the information sheet were answered. Written consent was taken (appendix 11). All participants were asked to fill out the following questionnaires in the order given here: age and gender, Hospital Anxiety and Depression Scale, State Trait Anxiety Inventory, Mill Hill Vocabulary Scale (synonyms section, appendix 5). The experimental group were also asked questions verbally on their past and current drug use (appendix 6), and then completed the measures of dependence, (SDS, appendix 7) withdrawal (SOWS, appendix 8) and craving (single item, appendix 9). Following the questionnaires, participants were asked to complete the two experimental tests of attentional bias, a modified Stroop colour-naming task and a dot probe task.

Stroop Task: Prior to administration of this task participants were asked if they were colour blind. No colour blindness was reported. The Stroop task was administered using standardised instructions for all participants. A copy of these can be found in appendices 10. Participants were asked to ignore the content of the words and name the colour of each word as quickly as possible without making mistakes. The practice card was administered first and participants were asked to complete the first column of words only. This first card was not timed. For the experimental and control cards, participants were required to complete the whole card, going down the list as quickly as possible, taking care not to make mistakes. The order of presentation of the experimental and control cards was counterbalanced to control for presentation effects. Half the participants completed the drug-related word card first and half completed the control card first. The time taken to complete the

experimental and control cards and the number of errors made were recorded in minutes and seconds for each participant using a stopwatch.

Dot Probe Task: Participants were presented with 12 practice trials followed by 252 main trials, 168 critical trials and 84 filler pairs. At the start of each trial, participants were asked to focus on a central fixation cross in the centre of the screen for 1000ms. Next a pair of pictures were presented. The length of presentation of the pictures was varied between 200ms, 500ms, and 1500ms. After each presentation a dot probe replaced one of the pictures. Participants were instructed to press response buttons as quickly as possible to indicate whether the probe appeared on the left or right of the screen. Each pair of pictures was presented twelve times, once in each of the four conditions resulting from the combination of picture location (left or right), and probe location (left or right), at each of the three time intervals. Order of the trials and length of time of presentation was fully randomised. Response accuracy and latencies were recorded automatically by the computer.

After completion of the dot probe task, a second measure of craving was taken from the opiate group. At this point any questions were discussed. Participants were informed how to access the results and contact the experimenter if any further questions arose.

Results

Group Characteristics

Table I shows descriptive statistics and t-tests comparing the two groups in age and on measures of affect and verbal ability. There were no significant differences between the two groups in terms of age. The opiate dependent group had significantly higher scores on measures of state and trait anxiety and depression and

had significantly lower scores on the Mill Hill Vocabulary scale. There were more males in the opiate group and more females in the control group. The male: female ratios in each group were 15:4 and 9:11 respectively. Chi Squared analysis revealed a significant association between gender and group, $\chi 2=4.74$; $df = 1$; $p < 0.05$.

Insert Table 1 about here

Drug use demographics

The mean number of years that the dependent group had been using opiates was 12.1 (SD=9.1). The mean daily dose of oral methadone prescribed at the time of the study was 34.5mg (SD=15.9). The mean Severity of Dependence score, 11.6 (SD=2.8) was within one SD of the mean given for heroin users in Britain (8.7 (SD=4.0)), taken from sample 4 in the standardisation study (Gossop et al., 1995). The mean level of withdrawal, 5.9 (SD=6.9) out of a maximum of 30 on the SOWS, indicates a low level of discomfort in the 24 hours prior to testing. There was no significant difference between measures of craving before and after the experimental tasks ($t(18) = 0.36$, ns).

Dot Probe Task

Data from trials with errors were removed and reaction times less than 200ms and more than 3 S.D. above each participants mean, were excluded as outliers. There were no significant differences between the amount of data lost as errors or outliers in the two groups. The total data missing was 2.0 % for each group and the errors were 0.6 % for the control group and 0.6% for the opiate dependent group.

To analyse data from the dot probe task an attentional bias score was calculated for each participant's response times from trials with drug – neutral card pairs. This score was calculated by subtracting the mean response time when the probe was in the same position as the drug picture, from the mean response time when the probe was in the different position (Bradley, Mogg, Millar, Bonham-Carter, Fergusson, Jenkins & Parr, 1997). A positive attentional bias scores reflects faster response times to probes that replace drug pictures. It is assumed that the faster response time indicates that the individual was already attending to the spatial location of that picture. This would indicate that attention is drawn to drug related pictures over neutral stimuli.

Insert Table 2 about here

A bias score was calculated for each participant at each of the three exposure times, 200, 500 and 1500ms. A 2 x 3 mixed design analysis of variance (ANOVA) was carried out on the bias scores with group (control, opiate) as a between-subjects variable and exposure (200, 500, 1500) as a within-subjects variable. The results showed a significant effect of exposure ($F(2, 74) = 4.98$; $p < .01$) and a significant main effect of group ($F(1,37) = 5.93$; $p < 0.05$). The interaction of exposure by group showed a trend towards significance ($F(2, 74); = 2.52$; $p = 0.08$). Table 2 shows the mean bias scores at each of the exposure lengths for the two groups.

As clear predictions were made for each exposure condition, hypothesis-driven analyses were made comparing the two groups on the bias scores in each condition. The results show that there is a significant difference between the attentional bias scores of the opiate dependent and control group at the 200ms exposure duration,

($t(37) = 2.70$, $p < 0.05$) with the opiate group showing more vigilance for drug cues than the control group. There was no significant difference between the two groups at the exposure lengths of 500ms ($t(37) = 1.48$; $p = 0.14$) and 1500ms ($t(37) = 0.22$; $p = 0.82$).

Pearson's product moment correlation was used to examine the relationship between attentional bias scores and characteristics of age, affect, verbal ability, and demographics of drug use in the opiate dependent group. No relationship was found between attentional bias and any of these factors.

Insert Table 3 about here

Stroop Colour Naming Task

A 2 by 2 mixed design ANOVA was used to analyse mean colour naming times with group (opiate, control) as between subjects variables and word type (drug and control words) as within-subjects variables. The results indicate that there was a significant group main effect ($F(1,37) = 6.79$; $p < 0.05$), as the opiate group were generally slower than the control group at naming all words. There was also a significant word type effect ($F(1,37) = 9.01$; $p < 0.01$) as participants were generally slower in colour naming drug-related words than control words. The interaction however was not significant ($F(1,37) = 1.84$, $p = 0.18$). Table 3 shows the mean scores for each group in each condition on the modified Stroop task.

Discussion

The group main effect indicates that the opiate group is more vigilant for drug cues than the control group. The mean overall bias score for each group was 20.9ms and

7.8 ms respectively. The main effect of exposure indicates that across the whole sample, participants were more vigilant for drug cues at 200ms (mean bias = 25.3) than at 500ms (mean = 12.6) or 1500ms (mean = 4.7). Further investigation was led by the hypotheses. Hypothesis 1 predicted that a significant attentional bias for drug cues would be evident at 200ms in the opiate dependent group reflecting an attentional bias at an automatic level of processing. A significant difference between the attentional bias scores of the opiate and control groups was found at the exposure length of 200ms. This indicates that there is an attentional bias for drug cues in dependent individuals and that this bias may be occurring at a relatively automatic processing level. This finding was predicted from, and supports, the incentive-sensitisation theory (Robinson & Berridge, 1993) which suggests that activation of the proposed system by drug associated cues occurs at an automatic processing level.

The results of this study are not consistent with previous experimental work with opiate dependent subjects. Franken et al. (2000) suggested that they found an attentional bias for drug-related words presented at a speed that allowed time for the engagement of strategic processing. They found no evidence for a bias at a subliminal level of processing (28ms). In the work by Franken et al. (2000), individual words were used and subjects were asked to identify real words from a set of mixed and nonsense words. The use of words as drug-associated stimuli is less ecologically valid than the presentation of pictures, which depict drug-use stimuli. It is difficult therefore to draw conclusions from the work by Franken et al. (2000) without further studies to back it up. More work is needed using the more accurate measure of the dot probe task to determine the nature of the attentional bias at the automatic level of processing.

Hypothesis two predicted that the findings from previous research which used this material (Lubman et al., 2000) would be replicated. This was not supported, as there was no evidence of an attentional bias for drug-related cues in opiate dependent subjects at 500ms. More research is needed to clarify why this may be so. It must be taken into account that six of the original drug related pictures used in the study, were eliminated in order to keep the task under 15 minutes and prevent fatigue effects. Some of the pictures that were eliminated contained more explicit scenes of injecting, which included visible injecting sites with needles and blood. The explicit nature of these few pictures may have made them more powerful cues, and triggered them to grab attention in the previous study in a way that the less explicit pictures used here did not. This may indicate that at 500ms a bias in attention is present towards more explicit cues, which is not present for less extreme pictures of injecting practices and associated stimuli. Robinson & Berridge (1993) state that sensitisation is modulated by experiential factors and environment. The expression of sensitisation in the form of attentional bias may therefore be affected by the emotional valence of the stimuli and the power attributed to them. It may be that the pictures depicting the point of injection have a stronger association with the drug use because they are presented in closest proximity to the drug-effects. As associative learning interacts with the sensitisation system to attribute the incentive power to the stimuli, (Robinson & Berridge 1993), this may mean that the expression of sensitisation is greatest when presented with explicit stimuli. These stimuli may attract 'wanting' and motivate behaviour at exposure times which are longer than the 200ms exposure found in this experiment. It is unclear whether exposure time of 500ms engages automatic or strategic cognitive processing. It would be useful to repeat this

experiment with all the original pictures presented at the two exposure times of 200 and 500ms to determine if they do consistently show an attentional bias at 500ms.

Hypothesis three predicted that if attentional bias was mediated by effortful cognitive strategies, then it would be found in the longer exposure duration of 1500ms. An exposure length of 1500ms allows time for multiple shifts in gaze between the picture pairs so this time is more susceptible to strategic effects in selective processing. This study found no indication of an attentional bias at 1500ms.

Hypothesis four predicted an interaction between group and word type on colour-naming times in the modified Stroop task. It was expected that the opiate dependent group would take longer to name the colours of drug-related words than control words and that this effect would not be present in the control group. Results on the modified Stroop task indicate that the opiate dependent group were slower to name all words and that participants in both groups were slower at naming drug-related words than control words. The Stroop task is difficult to interpret as response times and errors can be confounded. The Stroop task was used here as a supplementary task and it is a relatively crude measure of attentional bias. The fact that the staff in the control group who had a specialist interest in addiction also took longer to name drug-related words, may be an indication that the modified Stroop task reflects an interference for words associated with the current concerns for the individual. It may be that the modified Stroop does not reflect the attentional mechanisms, which are relevant to the incentive-sensitisation model (Robinson & Berridge 1993). One of the aims of this study was to explore whether attentional bias could be seen to be an index of activation of the Incentive Sensitisation system (Robinson & Berridge, 1993). Word presentation of stimuli may not be as 'attention grabbing' as pictures of heroin related cues. It is less ecologically valid as drug users

are less likely to be exposed to words during their use of drugs, and associations are less likely to be formed.

Future work would benefit from the continued use of the dot probe task which is a more accurate measure of attentional bias. This study has suggested that attentional bias may be operating at an automatic level of processing in satiated opiate dependent subjects maintained on methadone. It would be interesting to determine whether this result is consistently found in dependent subjects who are using street heroin and still injecting. Similarly no work has yet been conducted with abstinent opiate addicts to determine whether an attentional bias toward drug-related stimuli is still active in the months and years following cessation. Robinson & Berridge (1993) predict that attentional bias may therefore act as an indicator for susceptibility to relapse. More extensive work using this paradigm could be used to determine this. The cognitive processing model (Tiffany 1990) may predict that more strategic cognitive processing would be engaged when the dependent users is abstinent from opiates therefore it would be interesting to see if a bias became evident at the longer exposure time.

Differences between the two groups were found on measures of affect. This is consistent with previous research, which has found that opiate dependent participants have higher rates of depression and anxiety compared to the general population (Darke, Swift & Hall, 1994; Ryan & White, 1996). This may be expected as social-economic deprivation is often associated with long-term drug abuse, and is identified as a significant risk factor for both anxiety and depression. It is possible however that the differences in mood between the two groups may have confounded the results. It may be that the drug related stimuli were more emotionally negative than the control pictures and triggered an automatic processing bias in the more anxious and

depressed subjects. The observed effect may therefore have been due to differences between the two groups in their sensitivity to emotionally laden stimuli caused by the differences in mood. Before it can be concluded that this study is evidence for an attentional bias to drug related material, this possibility needs to be eliminated. This could be done by conducting a study of the same design which uses emotionally negative stimuli devoid of drug-related content. If no effect is found in such a study then it could be suggested more conclusively that this study has shown an attentional bias to drug-related stimuli.

The groups also differed in age, gender ratio and the Mill Hill measure of current verbal ability. This is a limitation of the current research. When the Mill Hill scores were entered into the analysis as a co-variate however attentional bias was still significant at 200ms. The study would have been improved by increasing the period of data collection and matching the two groups for age and gender ratio. The difficulties of matching for verbal ability and affect are more complex given that a staff group were used as controls. As the use of opiates has a significant effect on mood (Darke, Swift & Hall, 1994; Ryan & White, 1996) and cognitive ability (Darke, Sims, McDonald & Wickes, 2000) it was not expected that the two groups would be matched for these characteristics. An alternative control group could have been used to match for this but the benefit of matching for knowledge of drug paraphernalia would then be lost.

In the context of other research (McCusker, 2001), empirical investigations appear to be indicating that attentional bias for drug cues is present at an automatic level of processing. The effectiveness of cognitive interventions in assisting dependent users to avoid relapse may be limited if it does not take into account the allocation of attentional resources. Self-regulation techniques may need to inform the

individual of the potential automatic allocation of attention. There may be a need to adapt the cognitive techniques used with dependent clients to take account of this bias in processing.

References

Abrams, D.B. (2000). Transdisciplinary concepts and measures of craving: commentary and future directions, Addiction, 95, 2, S237-S246.

Beck, A.T., Wright, F.D., Newman, C.F. & Liese, B.S. (1993). Cognitive Therapy of Substance Abuse. New York: Guilford Press.

Bradley, B.P., Mogg, K. & Millar, N. (1996). Implicit memory bias in clinical and non-clinical depression. Behaviour, Research and Therapy, 34, 856-879.

Bradley, B.P., Mogg, K. & Millar, N., Bonham-Carter, C., Fergusson, E., Jenkins, J. & Parr, M. (1997). Attentional bias for emotional faces. Cognition and Emotion, 11, 25-42.

Bradley, B.P., Mogg, K., Wright, T. & Bates, C. (in preparation). Attentional bias in drug dependence: vigilance for cigarette-related cues in smokers. (Paper available from authors, Department of psychology, University of Southampton).

Darke, S., Sims, J., McDonald, S. & Wickes, W. (2000). Cognitive impairment among methadone maintenance patients, Addiction, 95, 687-695.

Darke, S., Swift, W. & Hall, W. (1994). Prevalence, severity and correlates of psychological morbidity among methadone maintenance clients, Addiction, 89, 211-217.

Franken, I.H.A., Kroon, L.Y., Wiers, R.W. & Jansen, A. (2000). Selective cognitive processing of drug cues in heroin dependence. Journal of Psychopharmacology, 14, 395-400.

Gossop, M. (1990). The development of a short opiate withdrawal scale (SOWS). Addictive Behaviours, 15, 487-490.

Gossop, M., Darke, S., Griffiths, P., Hando, J., Powis, B., Hall, W. & Strang, J.

(1995). The Severity of Dependence Scale (SDS): psychometric properties of the SDS in English and Australian samples of heroin, cocaine and amphetamine users. Addiction 90, 607-614.

Gross, T.M., Jarvik, M.E. & Rosenblatt, M.R. (1993). Nicotine abstinence produces content-specific Stroop interference. Psychopharmacology, 110, 333-336.

Johnsen, B.H., Laberg, J.C., Cox, W.M., Vaksdal, A. & Hugdahl, K. (1994). Alcoholic subjects' attentional bias in the processing of alcohol-related words. Psychology of Addictive Behaviours, 8, 111-115.

Lubman, D.I., Peters, L.A., Mogg, K., Bradley, B.P. & Deakin, J.F.W. (2000). Attentional bias for drug cues in opiate dependence, Psychological Medicine, 30, 169-175.

MacLeod, C. M. (1991). Half a century of research on the Stroop effect: an integrative review. Psychological Bulletin 109, 163-203.

MacLeod, C. M., Mathews, A. & Tata, P. (1986). Attentional bias in emotional disorders. Journal of Abnormal Psychology, 95, 15-20.

Marlatt, G.A. & Gordon, J.R. (1985). Relapse Prevention: maintenance strategies for addictive behaviours, New York: Guilford

McCusker, C.G. (2001). Cognitive biases and addiction: an evolution in theory and method. Addiction, 96, 47-56.

Monti, P.M., Rohsenow, D.J., & Hutchinson, K.E. (2000). Toward bridging the gap between biological, psychobiological and psychosocial models of alcohol craving. Addiction, 95, S229-S236.

Niaura, R, Abrams, D.B., Shadel, W.G., Rohsenow, D.J., Monti, P.M. & Sirota, A.D. (1999) Cue exposure treatment for smoking relapse prevention: a controlled clinical trial. Addiction 94, 685-695

Posner, M.I., Snyder, C.R. & Davidson, B.J. (1980). Attention and detection of signals. Journal of Experimental Psychology: General 109, 160-174.

Raven, J.C. (1965). Guide to the Mill Hill Vocabulary Scale with the Progressive Matrices Scale, London: H.K. Lewis & Co Ltd.

Robinson, T.E., & Berridge, K.E. (1993). The neutral basis of drug craving: an incentive-sensitisation theory of addiction. Brain Research Review, 18, 247-291.

Robinson, T.E., & Berridge, K.C. (2000). The psychology and neurobiology of addiction: an incentive-sensitization view. Addiction, 95, 2, S91-S117.

Robinson, T.E., & Berridge, K.C. (2001). Incentive-sensitization and addiction. Addiction, 96, 103-114.

Rohsenow, D.J., Monti, P.M., Abrams, D.B., Rubonis, A.V., Niaura, R.S., Sirota, A.D. & Colby, S.M. (1992). Cue-elicited urge to drink an salivation in alcoholics: relationship to individual differences, Advances in Behavior Research and Therapy, 14, 195-210.

Ryan, C.F. & White, J.M. (1996). Health status at entry to methadone maintenance treatment using the SF-36 health survey questionnaire.

Sayette, M.A. & Hufford, M.R. (1994). Effects of cue exposure and deprivation on cognitive resources in smokers. Journal of Abnormal Psychology, 103, 812-818.

Sayette, M.A., Monti, P.M., Rohsenow, D.J., Gulliver, S.B., Colby, S.M., Sirota, A.D., Niaura, R. & Abrams, D.B. (1994). The effects of cue exposure on reaction time in male alcoholics, Journal of Studies on Alcohol, 55, 629-633.

Schneider, W. (1995). MEL Professional User's Guide. Pittsburgh: Psychology Software Tools.

Sharma, D., Albery, I.P. & Cook, C. (2001). Selective attentional bias to alcohol related stimuli in problem drinkers and non-problem drinkers. Addiction, 96, 285-295.

Speilberger, C.D. (1989). State-Trait Anxiety Inventory: A comprehensive bibliography. (Second Edition). Palo Alto, California: Consulting Psychologists Press.

Speilberger, C.D., Gorsuch, R.L., Lushene, R., Vagg, P.R. & Jacobs, G.A. (1983). Manual for the State-Trait Anxiety Inventory for Adults. Palo Alto, California: Consulting Psychologists Press.

Stetter, F., Ackermann, K., Bizer, A., Straube, E.R. & Mann, K. (1995). Effects of disease-related cues in alcoholic inpatients: Results of a controlled 'Alcohol Stroop' study. Alcoholism: Clinical and Experimental Research, 19, 593-599.

Stewart, J., de Wit, H. & Eikelboom, R. (1984). Role of unconditioned and conditioned drug effects in the self-administration of opiates and stimulants, Psychological Review, 91, 251-268.

Stirling, N. (1979) Stroop interference: an input and an output phenomenon. Quarterly Journal of Experimental Psychology 31, 121-132.

Tiffany, S.T. (1990). A cognitive model of drug urges and drug-use behaviour: role of automatic and nonautomatic processes. Psychological Review 97, 147-168.

Wikler, M.D. (1948). Recent progress in research on the neurophysiologic basis of morphine addiction. American Journal of Psychiatry, 105, 329-338.

World Health Organisation (1992). The ICD-10 Classification of Mental and Behavioural Disorders. World Health Organisation: Geneva.

Zigmond, A.S. & Snaith, R.P. (1983). The Hospital Anxiety and Depression Scale. Acta Psychiatrica Scandinavia, 67, 361-370

Table 1 :

Group Characteristics: Mean scores, standard deviations and significance levels indicating the differences between the two groups.

	Opiate Dependent Mean (SD)	Control Mean (SD)	t value except where chi square indicated by *	p
Gender	4 female 15 male	11 female 9 male	*4.7	0.05
Age	33.3 (8.6)	35.0 (6.9)	0. 7	Ns
Mill Hill Vocab.	8.7 (2.9)	10.9 (2.1)	2.7	0.01
HAD Depression	8.7 (4.7)	1.7 (1.7)	6.1	0.00
HAD Anxiety	10.4 (4.5)	4.6 (2)	5.1	0.00
STAI State Anxiety	42.8 (12.4)	29.8 (7.1)	3.9	0.00
STAI Trait Depression	50.0 (13.5)	34.7 (8.2)	4.2	0.00
SDS	11.6 (2.8)			
SOWS	5.9 (6.9)			
Craving – Pre	2.7			
Craving - Post	2.6			

NB: Equal variance not assumed for t-tests on HAD and STAI scores.

Table 2:

Mean bias scores and standard deviations for each group at each exposure length.

	200ms	500ms	1500ms
Opiate Dependent	39.7 (39.7)	19.3 (25.3)	3.6 (35.2)
Control	11.6 (23.7)	6.2 (29.3)	5.7 (18.8)

Table 3 :

Mean colour naming times and standard deviations for each group (opiate, control) on the control and drug-related Stroop cards

	Drug related Words	Control Words
Opiate Dependent Group	83.5 (22.9)	76.6 (19.2)
Control Group	68.1 (12.1)	65. 5 (9.8)

Appendices

Appendix 1

Submission Guidelines for the British Journal of Clinical Psychology

British Journal of Clinical Psychology
Notes for Contributors

Four good copies of papers in line with the Journal Scope should be submitted to the Editor:
Professor Stephen Morley
The British Psychological Society,
St Andrews House,
48 Princess Road East,
Leicester,
LE1 7DR,
UK
The Journal encourages submissions in English from authors around the world.
Case studies are normally only published as Brief Reports. Papers are evaluated in terms of their theoretical importance, contributions to knowledge, relevance to the concerns of practising clinical psychologists, and readability. Papers generally appear in order of acceptance except for the priority given to Brief Reports and Comments.
The Editorial Board will reject papers which evidence discriminatory, unethical or unprofessional practices.
The Journal operates a policy of blind peer review. Papers will normally be scrutinized and commented on by at least two referees as well as by the Editor or an Associate Editor. The referees will not be made aware of the identity of the author. All information about authorship including personal acknowledgements and institutional affiliations should be confined to a removable front page and the text should be free of all such clues as identifiable self-citations ("In our earlier work..."). The paper's title should be repeated on the first page of the text.

Preparation of manuscripts

Publication is speeded by care in preparation. Contributions should be prepared in accordance with The British Psychological Society Style Guide available online, or in printed format from mail@bps.org.uk.
Contributions should be as concise as clarity permits, and illustrations kept as few as possible. Papers should not normally exceed 5000 words. A structured abstract of up to 250 words should be provided.
The title should indicate exactly but as briefly as possible the subject of the article, bearing in mind its use in abstracting and indexing schemes.
The Journal proposes to adopt structured abstracts. Articles containing original scientific research should include astructured abstract with the following headings and information:

- **Objectives:**
  State the primary objective of the paper and the major hypothesis tested (if appropriate).
- **Design:**
  Describe the design of the study and describe the principal reasoning for the procedures adopted.
- **Methods:**
  State the procedures used, including the selection and numbers of participants, the interventions or experimental manipulations, and the primary outcome measures.
- **Results:**
  State the main results of the study. Numerical data may be included but should be kept to a minimum.
- **Conclusions:**
  State the conclusions that can be drawn from the data provided, and their clinical implications (if appropriate).

Review articles should include an abstract which may be structured under the following headings:

- **Purpose:**
  State the primary objectives of the review.
- **Methods:**
  State the methods used to select studies for the review, the criteria for inclusion, and the way in which the material was analysed.
- **Results:**
  State the main results of the review.
- **Conclusions:**
  State the conclusions that can be drawn from the review, and their clinical implications if appropriate.

Authors please note: Revisions without a structured abstract will not be considered for publication.

1. Contributions should be typed in double spacing with wide margins and on only one side of each sheet. Sheets should be numbered. The top copy and three good copies should be submitted and a copy retained by the author.
2. Tables should be typed in double spacing on separate sheets. Each should have a self-explanatory title and should be comprehensible without reference to the text. They should be referred to in the text by arabic numerals. Data given should be checked for accuracy and must agree with mentions in the text.
3. Figures, i.e. diagrams graphs or other illustrations, should be on separate sheets, numbered sequentially "Fig. 1", etc., and each identified on the back with the author's name and the title of the paper . They should be carefully drawn, larger than their intended size, suitable for photographic reduction and clear when reduced in size. Captions should be listed on a separate sheet.
4. Bibliographical references in the text should quote the author's name and date of publication thus: MacGregor (1996). They should be listed alphabetically by author at the end of the article according to the following format:

   Herbert, M. (1993). Working with children and the Children Act (pp. 77-106). Leicester:The British Psychological Society.

   Smith, P. B., Peterson, M. F., & Misumi, J. (1994). Event management and work team effectiveness in Japan, Britain and the USA. Journal of Occupational and Organizational Psychology, 67, 33-44

   Particular care should be taken to ensure that references are accurate and complete.
5. SI units must be used for all measurements.
6. Participants in research should not be referred to as subjects; suitable alternative formulations will depend on the sample members.

Brief Reports and Comments are limited to two printed pages. These are subject to an accelerated review process to afford rapid publication of research studies, and theoretical, critical or review comments whose essential contribution can be made within a small space. They also include research studies whose importance or breadth of interest is insufficient to warrant publication as full articles, and case reports making a distinctive contribution to theory or method. Authors are encouraged to append an extended report to assist in the evaluation of the submission and to be made available to interested readers on request to the author. Figures and tables should be avoided. Title, author name and address for reprints and date of receipt are not included in the allowance.

Proofs are sent to the corresponding author for correction, but not for insertion of new or different material. They should be returned to the Journals Department within seven days of receipt. Fifty complimentary copies of each paper are sent to the corresponding author on request: further copies may be ordered on the form supplied with the proofs.

Copyright

Submission of a paper implies that it has not been published elsewhere and is not currently under consideration elsewhere.

Authors should consult the journal Editor concerning prior publication in any form or any language of all or part of their paper.

Authors are responsible for getting written permission to publish lengthy quotations, illustrations, etc., of which they do not own copyright.

To protect authors and journals against unauthorized reproduction of articles, The British Psychological Society requires copyright to be assigned to itself as publisher, on the express condition that authors may use their own material at any time without permission. On acceptance of a paper, authors will be

_____

requested to sign an appropriate assignment of copyright form.

Appendix 2

Ethical Approval from

University of Southampton

Avon and Western Wiltshire Mental Health Care Trust

of Southampton

Department of Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5000
Fax +44 (0)23 8059 4597
Email

3 November 2000

Lisa Frankland
Department of Clinical Psychology
University of Southampton
Highfield
Southampton SO17 1BJ

Dear Lisa,

Re: Application for Ethical Approval

I am writing to confirm you that your ethical application titled "An investigation into attentional bias for drug cues in opiate dependence: manipulation of stimulus duration", has been given approval by the department.

Should you require any further information, please do not hesitate in contacting me on (023) 80 593995.

Yours sincerely,

[redacted]

Kathryn Smith
Ethical Secretary

Royal United Hospital Bath NHS
NHS Trust

BATH LOCAL RESEARCH ETHICS COMMITTEE
Direct tel/fax: 01225 825725. e-mail: *research.ethics@ruh-bath.swest.nhs.uk*

Royal United Hospital
Combe Park
Bath
BA1 3NG

Tel: 01225 428331

11 December 2000

Ms L Frankland
30 Ashton Road
Ashton
Bristol
BA3 2EG

Dear Ms Frankland

BA128/00-01 ***(please quote this reference on all correspondence)***
An investigation into attentional bias for drug cues in opiate dependence: manipulation of stimulus duration

Thank you for your letter dated 25 November 2000 enclosing an information sheet for control participants 'November 2000 – version 2', together with amended versions of the Short Opiate Withdrawal Scale (SOWS), Severity of Dependence Scale (SDS) and past and current drug use (Use of Opiates). Following consideration by the original reviewers of your study, I can confirm that they are satisfied that you have addressed the concerns raised in Dr Taylor's letter to you dated 23 November 2000. I am therefore pleased to confirm that your study has full approval to proceed.

This Committee is organised and operates according to ICH/GCP and the applicable laws and regulations. Any changes or extensions to the protocol, or additional investigators should be notified to the Committee for approval. Serious and unexpected adverse events should also be notified to the meeting. May we remind you of the Data Protection Act 1984 and the need to conduct the trial in accordance with the Good Clinical Practice Guidelines.

The Committee is required to audit progress of research and to produce a yearly report to the Avon Health Authority and Department of Health. Investigators are therefore required to provide a brief yearly report and a short final report.

Yours sincerely

[redacted]

Anna Jenkins
Research Ethics Administrator

Appendix 3

Information Sheet for Opiate Dependent Participants

TAKING PART IN RESEARCH

INFORMATION FOR PATIENTS ABOUT THE STUDY

November 2000 – Version 2

Study Title
An investigation into attentional bias in people addicted to opiates.

What is the purpose of the study?
The study looks at the relationship between mood, drug taking and performance on a simple computer task.

Why have I been chosen?
People within the service who are currently using methadone are being asked to participate and you are one of the people that meet the criteria.

Who is organising the study?
The study is being organised by Lisa Frankland, Trainee Clinical Psychologist along with the University of Southampton, Bath Specialist Drug and Alcohol Service and Bristol Specialist Drugs Service.

What will happen to me if I take part?
If you chose to take part in the study you will be asked to attend one session, at the clinic you usually attend, for half an hour. Reasonable travel expenses will be paid if you attend.
There are a number of theories about opiate addiction, which make predictions about how drugs affect people's mood and thinking processes. This study intends to test these by asking you to fill out some simple questionnaires about your mood, past and current drug use, and level of withdrawal and craving. You will then be asked to complete some simple tasks on a computer, namely pressing a key in response to the presentation of different pictures, both drug-related and non drug-related. These pictures will have varied content and they are similar to pictures presented in magazines.
You will also be asked to complete a colour naming task which involves saying out loud the colour in which some words are printed, e.g. red, blue.
You are free to withdraw from this study at any stage without any penalty, or it in any way affecting your medical treatment.

Are there any disadvantages or advantages in taking part in this study?
The procedures of this research have been previously conducted in Manchester and Cambridge and there have been no reported disadvantages to taking part. This study does not involve any changes to your treatment and there are no clinical benefits to taking part

Confidentiality – who will know I am taking part in the study?
All information which is collected about you during the course of the research will be kept strictly confidential. Any information about you which leaves the community base will not have your name associated with it so that you cannot be recognised from it.
Only your consent is needed for you to participate and no other health or social care professionals will be notified of your decision.

LREC Approval
Bath, Local Research Ethics Committee approved this study.

What will happen to the results of the study?

The results of this study will be written up for a doctoral dissertation in Clinical Psychology through the University of Southampton. No identifying information will be included in this. If you chose to participate and wish to receive a written summary of this research please make a request to the investigator when you take part.

Contact for further information

If you would like further information about this study please contact:

Lisa Frankland, Trainee Clinical Psychologist, Bath
Tel: 01225 428099.

Thank you for taking the time to read this information.

Appendix 4

Information Sheet for Control Participants

INFORMATION ABOUT THE STUDY FOR CONTROL PARTICIPANTS

November 2000 – Version 2

Study Title
An investigation into attentional bias in people addicted to opiates.

What is the purpose of the study?
The study looks at the relationship between mood, drug taking and performance on a simple computer task.

Why have I been chosen?
24 people within the service who are currently using methadone are being asked to participate and 24 people will be used as matched controls. You are being asked to participate in the control group. Please do not volunteer if you have a history of a major psychiatric illness, significant head injury, significant physical health problems or a past or current dependence on opiates.

Who is organising the study?
The study is being organised by Lisa Frankland, Trainee Clinical Psychologist along with the University of Southampton, Bath Specialist Drug and Alcohol Service and Bristol Specialist Drugs Service.
It will take place over a time period of 2 – 4 months.

What will happen to me if I take part?
If you chose to take part in the study you will be asked to attend one session for up to half an hour

.
There are a number of theories about opiate addiction, which make predictions about how drugs affect people's mood and thinking processes. This study intends to test these by asking you to fill out some simple questionnaires about your mood and to complete some simple tasks on a computer. This involves pressing a key in response to the presentation of different pictures, both drug-related and non drug-related. These pictures will have varied content and they are similar to pictures presented in magazines.
You will also be asked to complete a colour naming task which involves saying out loud the colour in which some words are printed, e.g. red, blue.
You are free to withdraw from this study at any stage without any penalty.

Are there any disadvantages or advantages in taking part in this study?
The procedures of this research have been previously conducted in Manchester and Cambridge and there have been no reported disadvantages to taking part.

Confidentiality – who will know I am taking part in the study?
All information, which is collected, about you during the course of the research will be kept strictly confidential. Any information about you which leaves the community base will not have your name associated with it so that you cannot be recognised from it.
Only your consent is needed for you to participate and no other health or social care professionals will be notified of your decision.

LREC Approval
Bath, Local Research Ethics Committee approved this study.

What will happen to the results of the study?
The results of this study will be written up for a doctoral dissertation in Clinical Psychology through the University of Southampton. No identifying information will be included in this. If you chose to participate and wish to receive a written summary of this research please make a request to the investigator when you take part.

Contact for further information
If you would like further information about this study please contact:

Lisa Frankland, Trainee Clinical Psychologist, Bath Specialist Drug and Alcohol Service.
Tel: 01225 428099.

Thank you for taking the time to read this information.

Appendix 5

Mill Hill Vocabulary Scale (Synonyms section, alternate version)

In each group of six words below underline the word which means the same as the word in heavy type above the group, as it has been done in the first example:

1 **CONNECT**

accident	join
lace	bean
flint	field

2 **STUBBORN**

obstinate	steady
hopeful	hollow
orderly	slack

3 **LIBERTY**

worry	freedom
rich	serviette
forest	cheerful

4 **RESEMBLANCE**

attendance	fondness
assemble	repose
likeness	memory

5 **PRECISE**

natural	stupid
faulty	grand
small	exact

6 **DWINDLE**

swindle	pander
diminish	wheeze
linger	compare

7 **WHIM**

complain	noise
tonic	fancy
wind	rush

8 **BOMBASTIC**

democratic	pompous
bickering	cautious
destructive	anxious

9 **ENVISAGE**

contemplate	activate
surround	estrange
enfeeble	regress

10 **GLOWER**

extinguish	shine
disguise	gloat
aerate	scowl

11 **LEVITY**

parsimony	velleity
salutary	frivolity
alacrity	tariff

12 **AMULET**

savoury	jacket
flirtation	crest
cameo	charm

13 **TEMERITY**

impermanence	rashness
nervousness	stability
punctuality	submissiveness

14 **ABNEGATE**

contradict	decry
renounce	execute
belie	assemble

15 **VAGARY**

vagabond	caprice
obscurity	vulgarity
evasion	fallacy

16 **SEDULOUS**

rebellious	dilatory
complaisant	diligent
seductive	credulous

17 **ADUMBRATE**

foreshadow	protect
detect	eradicate
elaborate	approach

Appendix 6

Questionnaire on past and current opiate use

USE OF OPIATES

1. **How old were you when you first used opiates? .....**

2. **How many years is it since your first dose? .....**

3. **How many years have you been using every day? .....**

4. **How many years is it since you first considered yourself addicted? .....**

5. **Which opiates do you usually take? (Please circle as appropriate)**

	Amount per day
Heroin	.....
Morphine	.....
Methadone (Dolophine)	.....
Meperidine (Demerol)	.....
Dilaudid	.....
Paregoric	.....
Codeine	.....
Percodan	.....
Other	.....

6. **During the last year did you inject?**

0	1	2	3
Never	sometimes	often	Always

6a. **If never, do you find injecting unpleasant? Yes / No**

7. **During the last year did you smoke opiates?**

0	1	2	3
Never	sometimes	often	Always

8. **During the last year did you take liquid or pills?**

0	1	2	3
Never	sometimes	often	Always

9. **Have you ever stopped taking opiates for at least two weeks? Yes / No**

Appendix 7

Severity of Dependence Scale (SDS)

Severity of Dependence Scale (SDS) (Gossop et al 1995)

Please circle the number which best indicates your use of opiates in the last year:

For the purpose of this questionnaire opiates refers to any illicit or prescribed opiate drug including all those listed in the previous questionnaire.

1. Did you think that your use of opiates was out of control?

0	1	2	3
Never / Almost Never	sometimes	often	Always / Almost Always

2. Did the prospect of missing a fix (or dose), or not chasing make you anxious or worried?

0	1	2	3
Never / Almost Never	sometimes	often	Always / Almost Always

3. Did you worry about your use of opiates?

0	1	2	3
Never / Almost Never	sometimes	often	Always / Almost Always

4. Did you wish you could stop?

0	1	2	3
Never / Almost Never	sometimes	often	Always / Almost Always

5. How difficult did you find it to stop, or give up opiates?

0	1	2	3
Not difficult	**Quite difficult**	**Very difficult**	**Impossible**

Appendix 8

Short Opiate Withdrawal Scale (SOWS)

SHORT OPIATE WITHDRAWAL SCALE

Please put a tick in the appropriate box if you have had any of the following during the last 24 hours.

	None	Mild	Moderate	Severe
Feeling Sick				
Stomach Cramps				
Muscle spasms/ twitching				
Feelings of Coldness				
Heart Pounding				
Muscular Tension				
Aches & Pains				
Yawning				
Runny Eyes				
Insomnia / Problems sleeping				

What drugs have you taken in the last 24 hours and how much? e.g 30ml methadone at 10am,

.....

.....

.....

.....

Appendix 9

Measure of Craving

Measure of Craving

Level of craving

Please indicate on the scale below the level of craving for opiates that you are currently feeling.

0 = no craving for opiates at all

9 = extreme craving for opiates

No Craving **Extreme Craving**

0	1	2	3	4	5	6	7	8	9

Appendix 10

Instruction for modified Stroop task

Card Stroop Instructions

This task is a simple test of attention and concentration.

--- HAND OVER PRACTICE CARD (ANIMALS) ---

On this card there are lists of words written in 4 different colours – red, green, yellow and blue.

Ignore the words themselves, and simply name the colour of each word, reading DOWN the list like this. Red, blue, yellow, green, blue, green and so on..

You can keep your place with your finger (like this), but be careful not to cover up the words.

When you get to the bottom of each list, start immediately at the top of the next list.

I would like you to name the colours of ALL the words on the card as quickly as possible. Please be careful not to make mistakes, like saying the wrong colour.

Do you have any questions? Are you ready to start?

Now name the colours of the words as quickly as possible. Try to avoid mistakes.

Record total time with stopwatch, and count number of errors on each card.

-- SECOND CARD –

There are two more cards just like that one, only they have different words.

REMINDER BEFORE EACH CARD:

Remember, ignore the words themselves, and name the colour of each word.
Work through the whole card, going down each list, as quickly as possible. Be careful not to make mistakes.

-- Half of each group get Drug card before Household card, and vice versa for the other half.

-- REPEAT FOR THIRD CARD –

It is advisable to audiotape performance, so timing and errors can be checked later if necessary. A small (non-intrusive) tape recorder is ideal.

Appendix 11

Written Consent form

Avon and Western Wiltshire

Mental Health Care NHS Trust

Centre Number:

Study Number: BA 128/00-01

Patient Information Number for this trial:

CONSENT FORM

Title of Project: An Investigation into Attentional Bias in People Addicted to Opiates

Name of Researcher: Lisa Frankland

[Name and number of independent person]

Please initial box

1. I confirm that I have read and understand the information sheet dated November 2000 (version 2) for the above study ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time without my medical care or legal rights being affected. ☐
3. I am willing to allow access to my medical records but understand that strict confidentiality will be maintained. The purpose of this is to check that the study is being carried out correctly. ☐
4. I agree to take part in the above study. ☐

Name of patient	Date	Signature
Name of person taking consent (if different from researcher)	Date	Signature
Lisa Frankland Researcher	Date	Signature

1 for patient; 1 for researcher; 1 to be kept with hospital notes