Enhanced Recognition of Facial Expressions of Disgust in Opiate Users

Louise Martin

D. Clin. Psy. Thesis (Volume 1), 2005
University College London

UMI Number: U592163

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



UMI U592163

Published by ProQuest LLC 2013. Copyright in the Dissertation held by the Author.

Microform Edition © ProQuest LLC.

All rights reserved. This work is protected against unauthorized copying under Title 17, United States Code.



ProQuest LLC 789 East Eisenhower Parkway P.O. Box 1346 Ann Arbor, MI 48106-1346

Overview

The literature review focuses on the research relating to facial expressions of emotion, first addressing the question of what they are and what role they play, before going on to review the mechanisms underlying facial expression recognition (FER). It then considers the psychiatric and drug-using populations in which the ability to recognise facial expressions is compromised, and how this may impact on social behaviour. Finally, the review focuses on one particular population: opiate users. The relevance of studying this population will be discussed and the limited evidence relating to recognition of facial expressions in this group will be presented.

The empirical paper describes a study which investigated FER in an opiate using population, comparing methadone maintained clients (MM), abstinent ex-opiate users (R) and healthy controls (C). Its main finding was that, contrary to existing research predicting impaired FER in this population, MMs displayed *enhanced* recognition of one emotion: disgust. The literature around disgust recognition is considered, and characteristics of the opiate-using population that may be relevant are described. One speculation is that opiate users are hypersensitive to others' expressions of disgust due to the negative reactions they encounter from society. Further research in this area is indicated, and clinical implications discussed.

The critical review comprises a reflective account of the research process, followed by a critical appraisal of the study, the main topic of which is the validity of the study and directions for future research.

Table of Contents

Part 1: Literature Review

Abstract6 1. Facial expressions: what are they, why are they and what role do they play? 6 1.1 What are they?6 1.2 Why are they?8 1.3 What is their function?9 1.4 What do individual facial expressions communicate?10 2. The recognition of facial expressions: Mechanism of action13 2.1.2 The pre-frontal cortex14 3. Populations in which FER is impaired......21

3.1 Psychiatric illness213.1.1 Schizophrenia213.1.2 Bipolar Disorder223.1.3 Depression24

3.1.4 Obsessive-Compulsive Disorder (OCD)253.1.5 Psychopathy and Acquired Sociopathy273.2 Drugs and Alcohol283.2.1 Alcohol283.2.2 Ecstasy29

 3.2.3 Benzodiazepines
 30

 3.3 Summary
 30

Page Number

4.1 Different kinds of opiates	32
4.2 Neuropharmacological Action of opiates	33
4.3 Interpersonal functioning and personality in opiate users	33
4.4 Opiates and recognition of facial expressions	35
5. Methodological issues in the study of FER	36
6. References	39
Part 2: Empirical paper	
Enhanced recognition of facial expressions of disgust in opiate users	58
References	93
Part 3: Critical Appraisal	
A reflection on the research process	110
Critical appraisal	115
References	120
Appendices	
Appendix A: Permission letter from Camden and Islington NHS Trust Ethic	cs
Committee	121
Appendix B: Depression Anxiety Stress Scale (DASS)	124
Appendix C: Fast Alcohol Screening Test (FAST)	
rippendix C. Fust Aiconor Screening Test (17151)	126
Appendix D: Spot-the-Word Test	
Appendix D: Spot-the-Word Test.	128
Appendix D: Spot-the-Word Test	128
	128131133
Appendix D: Spot-the-Word Test. Appendix E: Barratt Impulsivity Scale (BIS) Appendix F: Gough Socialisation Scale	128131133136
Appendix D: Spot-the-Word Test. Appendix E: Barratt Impulsivity Scale (BIS) Appendix F: Gough Socialisation Scale Appendix G: Aggression Questionnaire (AQ) Appendix H: Cut-down Annoyed Guilty Eye-opener Scale: Adapted to Inc	128 131 133 136 lude
Appendix D: Spot-the-Word Test. Appendix E: Barratt Impulsivity Scale (BIS) Appendix F: Gough Socialisation Scale Appendix G: Aggression Questionnaire (AQ) Appendix H: Cut-down Annoyed Guilty Eye-opener Scale: Adapted to Inc Drugs (CAGE-AID).	128 131 133 136 lude 139
Appendix D: Spot-the-Word Test. Appendix E: Barratt Impulsivity Scale (BIS) Appendix F: Gough Socialisation Scale. Appendix G: Aggression Questionnaire (AQ).	128131133136 lude139

Acknowledgements

Recruitment of participants for this research relied heavily on the help of the staff of the Margarete Centre, Core Trust, Thurston House, SHARP, Phoenix House and Job Centre. I greatly acknowledge their time and effort in encouraging their clients to take part, and helping me to co-ordinate the interviews.

Importantly, this research would not have been possible without the clients themselves. Aside from my deep gratitude to them for them taking the time and patience to participate, their willingness to share their experiences greatly enriched my experience of conducting this research.

Many thanks also to Val Curran, Dominic O'Ryan and Paul Davis for their supervisory help and support throughout.

Lastly, a very special mention to Joanna Coyle, my fellow trainee and recruiter. We laughed together and cried together. Here's to a focus group!

Part 1: Literature Review

Abstract

This literature review focuses on the research relating to facial expressions of emotion, first addressing the question of what they are and what role they play, before going on to review the mechanisms by which they are recognised in others. It then considers the psychiatric and drug-using populations in which the ability to recognise facial expressions is compromised, and how this corresponds to the social behaviour that characterises these groups. Finally, this review will focus on one particular population, opiate users. The relevance of studying this population will be discussed and the evidence relating to recognition of facial expressions in this group will be presented.

1. Facial expressions: what are they, why are they and what role do they play?

1.1 What are they?

Research suggests that facial expressions of emotion are innate, automatic and universal displays (Ekman & Yamey, 2004). Facial expressions are present from birth and displayed independently of social learning. Thus babies and congenitally blind people display the same range of facial expressions as adults and sighted people (Galati, Scherer & Ricci-Bitti, 1997). Facial expressions are thought to be produced automatically as a result of impulses generated by the emotional state of the individual, although their display can be attenuated or enhanced depending on the rules or norms of the culture in which they are produced (e.g. Ekman, 1999b). Research has investigated the 'universality hypothesis' (i.e. that the same basic

emotions are recognised in all cultures; review by Ekman, 1999b). While the methodology of this research has been criticised by some (Fridlund, 1994; Russell, 1994), the overwhelming evidence is that the same basic emotions are present panculturally.

Of the wide range of emotions that people experience, a set of basic emotions has been identified from which other more complex emotions are thought to be derived (Oatley & Johnson-Laird, 1987). While researchers have argued for the inclusion of different emotions in this basic set, the consensus is for a set of between five and seven basic emotional facial expressions. Ekman, Friesen and Ellsworth (1972) initially argued for the existence of six basic emotions: happiness, sadness, fear, anger, disgust and surprise. Since then, Ekman has added contempt to his list (Ekman & Yamey, 2004). Others, such as Oatley and Johnson-Laird (1987) argue for five basic emotions: happiness, sadness, fear, anger and disgust. They challenge the inclusion of surprise on the basis that it is a more cognitive component that could accompany any other emotion, rather than being a unique emotion per se (Power & Dalgleish, 1997).

"There is robust, consistent evidence of a distinctive universal facial expression for anger, fear, enjoyment, sadness and disgust. This evidence is based not just on high agreement across literate and preliterate cultures in the labelling of what these expressions signal, but also from studies of the actual expression of emotion, both deliberate and spontaneous, and the association of expressions with social interactive contexts." (Ekman, 1992, pp.175-176)

1.2 Why are they?

So, why do we have the capacity to automatically display our emotional state? The innate and universal nature of facial expressions suggests that there must be some purpose behind their existence and evolutionary continuation.

Looking to other primates provides some clues to the heritage of facial expressions. Darwin initiated the formal study of facial expressions in humans and non-human primates, and concluded that they serve an important communicatory function (Darwin, 1872). He suggested that displays of facial expression are essential to the well-being of any animals living in groups, as group living necessitates co-operation. It has been observed that in non-human primates, the range of facial expressions is best developed in species that are active during daytime, live in grasslands rather than trees and live in large and complex social groups (Argyle, 1988). The presence of facial expressions under these conditions supports the idea that they play an important communicatory role. It may be that facial expressions provide a means of communication that helps to regulate social structure and hierarchy within groups (Argyle, 1988; Keltner & Haidt, 1999). Facial expressions in monkeys are thought to be an important part of communication relating to submissive, aggressive and affiliative behaviour as well as to copulation (Argyle, 1988). Interestingly, the same facial configurations can be found in humans and a number of other primates (Chevalier-Skolnikoff, 1973; Redican, 1982).

This suggests that facial expressions evolved to regulate social behaviour in order to facilitate group living in primates. But how does this relate to the function of facial expressions in humans at the present time?

1.3 What is their function?

In his earlier work, Ekman (1957) argued that while facial expressions might communicate to others, this was not their purpose. He believed that although facial expressions evolved as a way of imparting information to conspecifics, this did not mean that every time an emotion is displayed it acts as a signal to others, particularly in the present day. He took the position that expressions were generated automatically and any communication that took place was incidental and unintentional.

More recently, Ekman (1999a) has modified his view. He now proposes that emotional expressions are crucial to the development and regulation of interpersonal relationships. He describes three areas in which this is apparent: in attachment formation, both during infancy and courtship, and also in relation to regulation of aggressive behaviour. He also notes the great difficulty which people with Mobius Syndrome (congenital face paralysis) report in sustaining relationships, and concludes that this is due to their lack of facial expressiveness.

Developmental psychology provides other examples of the function of facial expressions. Facial expressions serve an important role to infants who encounter novel objects in the presence of their caregiver. 'Infant social referencing' (Klinnert, Campos, Source, Emde & Svejda, 1983) refers to the way in which infants use the expressions and actions of their caregiver to understand events and guide their behaviour. For example, if the caregiver shows an expression of disgust or fear, the

infant avoids the novel object (Blair, 2003). A similar process occurring in monkeys is termed 'observational fear learning' (Mineka &Cook, 1993).

Interestingly, the display of facial expressions appears to depend on the social context in which they take place, rather than as a function of the strength of the emotion experienced. Experimental studies show that people smile more on watching a humorous video, and show more distress at another's experience of distress if they are with others than alone (Fridlund, 1991). Fridlund's (1991) study found that participants smiled more when co-viewing a humorous video with a friend, but also when they thought that their friend was watching the same humorous video simultaneously, compared with two solitary viewing conditions. Similarly, babies' smiling behaviour is thought to be dependent on the presence of adults (Jones, Collins & Hong, 1991). Such observations provide further support against the view that facial expressions are just an automatically generated reflection of the emotional state of the individual.

1.4 What do individual facial expressions communicate?

So what is it that individual facial expressions communicate in humans? Blair (2003) argues that facial expressions act as a nonverbal "short-hand" for communicating important information to peers, in addition to reinforcing behaviour, thus regulating appropriate social interaction. Keltner and Haidt (1999) discuss the purpose of facial expressions in organising the interactions of individuals at a dyadic level. Similarly, they argue that expressions help individuals to know the other's emotions, beliefs, intentions and orientation towards that relationship (e.g. as a dominant or submissive individual) which helps to rapidly co-ordinate social interactions. They also suggest

that facial expressions of emotion convey information about objects in the environment to conspecifics, and serve as incentives or deterrents for others' social behaviour.

Blair (2003) suggests that the facial expression of fear acts as an unconditioned stimulus that communicates the aversive nature of an object or situation to others so they can avoid it (Mineka & Cook, 1993). Fear is thought to be the most difficult emotion to recognise (Ekman & Friesen, 1976), a frequent recognition error involving misidentifying fear for surprise (Rapcsak, Galper, Comer, Reminger, Nielsen, Kaszniak, et al., 2000). Darwin noted the close relationship between these two emotions, pointing out that fear is often preceded by or mixed with surprise, and emphasising the common element of startle and physiological arousal (Darwin 1872).

Expressions of sadness are also thought to act as aversive unconditioned stimuli, discouraging the behaviour that elicited the sadness and motivating reparation (Blair, 1995) and soothing (Keltner & Haidt, 1999).

The expression of happiness is thought to act as an appetitive unconditioned stimulus which increases the probability that the behaviour will be repeated in the future (Matthews & Wells, 1999). Research suggests that happiness is the easiest and quickest facial expression to recognise (Leppanen & Hietanen, 2004; Rapcsak et al., 2000).

The facial expression of disgust appears to be most often used in relation to food, to quickly convey its aversive nature to others in order to deal with the risk of contamination and disease (Rozin, Haidt, & McCauley, 1993). It has also been proposed that displays of disgust may be important in negative socialisation, for example parental displays of disgust in toilet training (Rozin et al., 1993).

Interestingly, to date, there is no literature discussing the function of facial expressions of surprise.

Blair (2003) suggests that the facial expression of anger has a different function from the other expressions as it does not act as an unconditioned stimulus. Angry expressions appear to curtail the ongoing behaviour of others in situations where social rules have been violated (Averill, 1982), rather than provide information about future behaviour. Blair proposes that expressions of anger primarily act as a trigger for response reversal in order to maintain socially appropriate behaviour. It has been suggested that expressions of anger are particularly powerful in situations involving hierarchy (Blair & Cipolotti, 2000). Displays of anger have also been shown to elicit fear-related responses, even when these displays are presented subliminally (Dimberg & Ohman, 1996).

Ekman noted that people who are unable to produce facial expressions have difficulty forming and maintaining social relationships. If there are social consequences of not displaying expressions of emotion, are there also consequences for those who's ability to recognise facial expressions is compromised? How do people who have difficulty recognising facial expressions fare in the social world?

The nature of any social difficulties arising from such a deficit would also illuminate the role played by facial expressions.

The following section will summarise our current understanding of the way in which facial expressions are recognised, before going on to review populations in which facial expression recognition (FER) is impaired. The social behaviour of such affected populations will also be considered.

2. The recognition of facial expressions: Mechanism of action

2.1 Neuroanatomy

The recognition of facial expressions is a complex process involving a number of brain structures. Much of our current understanding of how facial expressions are recognised by the brain is derived from patients with lesions and neurological disorders who have impaired FER, and from imaging studies of both healthy people and patients. There is a growing body of literature which suggests that FER depends on anatomically dissociable neural systems (Adolphs, 2002).

2.1.1 The amygdala

Research suggests that the amygdala is a crucial structure in FER, particularly in the recognition of fear. Patients with amygdaloid lesions have consistently been seen to have impaired FER, particularly for the expression of fear (Adolphs, Tranel, Damasio & Damasio, 1995), and also in some studies for sadness (Adolphs, Tranel, Hamann, Young, Calder, Anderson et al., 1999; Fine & Blair, 2000).

Rapcsak et al. (2000) argue that the research methodology that has led to the conclusion that the amygdala is specifically involved in recognition of fear is flawed. From the results of their study with lesion patients, they concluded that the amygdala is involved in recognition of all facial expressions, and that the appearance of a fear-specific deficit is a reflection of how difficult recognition of this particular emotion is. Blair (2003) counters this argument, saying that the patients involved in Rapcsak et al.'s study had lesions that extended beyond the amygdala which account for the difference in findings.

Current neuroimaging data suggests that the amygdala is not involved in FER of anger (Blair, Morris, Frith, Perrett & Dolan, 1999; Sprengelmeyer, Rausch, Eysel & Przuntek, 1998). There is however neuropsychological data to suggest that lesions of the amygdala, particularly if these extend to the temporal cortex do disrupt the processing of angry expressions (Fine & Blair, 2000). Recognition of happiness does not seem to involve the amygdala (Fine & Blair, 2000), although it has been suggested that the ease with which happiness is recognised could be a confounding factor in this (Ekman & Friesen, 1976).

2.1.2 The pre-frontal cortex

The pre-frontal cortex is also implicated in FER, and seems to be particularly important in the recognition of angry expressions. Hornak, Rolls and Wade (1996) found that patients with damage to the ventral frontal lobe demonstrated impaired FER, but the authors did not differentiate between the differing emotional expressions. Blair et al. (1999) found increased activation in the orbitofrontal and

anterior cingulate cortices when subjects were shown facial expressions of anger, but not sadness. Harmer, Thilo, Rothwell and Goodwin (2001c) found longer reaction times in response to morphed angry (but not happy) facial expressions, when processing within the medial prefrontal cortex was disrupted via transcranial magnetic stimulation. Activation of this area on presentation of angry faces corresponds with Blair's hypothesis regarding this expression as a trigger for response reversal, as the orbitofrontal cortex is also implicated in this process (Dias, Robbins & Roberts, 1996).

2.1.3 Somatosensory related cortices and basal ganglia

Adolphs (2002) reviews the evidence from lesion studies investigating the involvement of the somatosensory related cortices and the basal ganglia in FER, concluding that they play a critical role. In a study of patients with lesions in the right ventral primary and secondary somatosensory areas, and to a lesser extent the insula and anterior supramarginal gyrus, Adolphs, Damasio, Tranel, Cooper and Damasio, (2000) found compromised FER. This is consistent with the hypothesis that in order to recognise the facial expression of another, we have to first simulate it ourselves, and it is this representation of emotion in the somatosensory cortex that allows us to infer the emotion of the other (Wild, Erb & Bartels, 2001).

Blair (2003) concludes that the insula (a visceral somatosensory cortex) is key to the recognition of disgust, based on lesion and neuro-imaging studies (Cubero, Thiele & Bernstein, 1999; Sprengelmeyer et al., 1998). Adolphs' (2002) review also concludes that the recognition of disgust relies on the insula, with the support of the basal

ganglia (particularly the caudate nucleus) and other somatosensory related cortices in the right hemisphere.

2.2 Neurotransmitter involvement

Research evidence from manipulation studies suggests that there is differential neurotransmitter involvement in the recognition of different facial expressions (Blair & Curran, 1999). To summarise, it seems that serotonin, noradrenaline, GABA and possibly dopamine are involved in FER.

2.2.1 Serotonin

Harmer and colleagues have conducted much of the research investigating the effect of serotonin manipulations on FER, with the finding that the expressions of fear and happiness are differentially affected. In one study, they found that administration of nutritionally sourced tryptophan to healthy female volunteers led to significantly increased recognition of fearful facial expressions, as well as (borderline) significantly increased recognition of happy facial expressions (Attenburow, Williams, Odontiadis, Reed, Powell & Cowen, 2003). It is assumed that this effect is mediated via changes in brain serotonin function. Similar results were found when serotonin levels in healthy volunteers were increased via single injections of the SSRI antidepressant citalopram. Acute administration of citalopram increased the accuracy of recognition and decreased the response time in identifying fearful and happy facial expressions, relative to placebo (Harmer, Bhagwagar, Cowen & Goodwin, 2001a; Harmer, Bhagwagar, Perrett, Vollm, Cowen & Goodwin, 2003a).

In another study, Harmer, Rodgers, Tunbridge, Cowen and Goodwin (2003b) decreased serotonin levels in healthy volunteers using a tryptophan depletion drink, with the result that female, but not male participants demonstrated impaired recognition of fearful facial expressions. Although accuracy of recognition was only impaired in the female volunteers, both male and female volunteers showed a significantly slower response in recognising fearful facial expressions.

Harmer et al. (2003a) note that the effects of serotonin on the threat perception system are counter-intuitive, given that SSRI medications are effective treatments for anxiety disorders such as GAD and panic disorder, and conclude that acute and chronic SSRI use may have different effects on fear processing. These authors have conducted two studies investigating this difference. In the first they found that sub-chronic (7 day) treatment with citalopram decreased recognition of fearful and other negative facial expressions in healthy volunteers (Harmer, Shelley, Cowan & Goodwin, 2002a). The second study (Harmer, Shelley, Cowen & Goodwin, 2004) compared serotonergic and noradrenergic agents with a placebo, with similar results to the first study. Specifically, participants treated with the serotonergic agent (citalopram) showed decreased recognition of anger and fear, and a positive bias when shown ambiguous faces. They tended to classify negative expressions as happy, and fearful expressions as surprised.

More recently, Hoshi, Bisla, and Curran (2004) have investigated the acute and sub-acute effects of 'ecstasy' (MDMA) on FER. While MDMA causes the release of dopamine and noradrenaline, its main action is on the serotonin system, where it stimulates release of stored serotonin and prevents its reuptake (Hoshi et al., 2004).

In the days following ecstasy use, serotonin is depleted as a result of the initial acute efflux. Hoshi and colleagues found that following acute administration of the drug, the ecstasy using group recognised a greater number of fearful expressions than controls, but four days later, they recognised fewer fearful facial expressions than controls. This again supports the involvement of serotonin in the recognition of fearful facial expressions.

2.2.2 Noradrenaline

Noradrenergic manipulations have been found to have a specific effect on the recognition of sad and happy facial expressions. Harmer and colleagues administered the adrenergic beta-blocker propranolol to healthy volunteers, and observed an increased reaction time but no change in accuracy of the identification of sad facial expressions, compared with a control group receiving a placebo (Harmer, Perrett, Cowen & Goodwin, 2001b). Propranolol did not have an effect on any other expression and did not cause any sedation which could account for the increased reaction time. In contrast, a study by Zangara, Blair and Curran (2002) which compared the effects of the beta-blocker metoprolol with diazepam on FER, did not find any effect of metoprolol, either in accuracy or speed of recognition. They did however, use a lower dose of beta blocker than Harmer et al. In another study, Harmer, Hill, Taylor, Cowen and Goodwin (2003c) observed that healthy volunteers recognised more happy facial expressions following a single dose of reboxetine (a noradrenergic antidepressant) than those who received a placebo. Similarly, in the aforementioned study comparing a serotonergic and noradrenergic agent, Harmer et al. (2004) found that healthy volunteers given a 7-day course of the noradrenergic

agent reboxetine showed decreased recognition of anger and fear, and a positive bias in classifying ambiguous expressions when compared with a placebo condition.

2.2.3 GABA

Evidence for the role of GABA in FER comes from studies with benzodiazepines, with results suggesting an involvement in processing expressions of anger and possibly fear. Blair and Curran (1999) used a between subjects design to investigate the effects of benzodiazepines on FER, finding that diazepam selectively impaired healthy volunteers' ability to recognise angry facial expressions. The aforementioned study by Zangara et al. (2002) replicated Blair and Curran's (1999) study, but with the inclusion of a beta blocker comparison group. Again they found that diazepam selectively impaired FER of anger, but on this occasion they also found that recognition of fear was impaired. They suggest several possibilities for this discrepancy, including task difficulty of fear recognition and an interaction effect between drug action and mood state.

Borrill, Rosen and Summerfield (1987) investigated the effects of differing doses of alcohol, which also has GABA-ergic action (as well as actions on several other neurotransmitters), on FER. They found that the high dose of alcohol had a highly significant effect impairing the recognition of anger, and also significantly impaired recognition of disgust.

2.2.4 Dopamine

To date, only one study regarding the role of dopamine in FER has been published.

Using a within subject design, Lawrence, Calder, McGowan and Grasby (2002)

administered sulpiride (a D2 receptor antagonist) to healthy male volunteers with the result that recognition of angry expressions was selectively impaired compared with placebo. They also included a control task of unfamiliar face matching, on which sulpiride had no effect. The authors discuss these results with reference to appetitive aggression and social dominance, in which dopamine is also implicated. During social-agonistic encounters, dopamine levels have been shown to be elevated (van Erp & Miczek, 2000), which seems to reflect increased attention to the provocative stimulus or attempts to deal with it. Acute administration of dopamine antagonists selectively impairs responses to agonistic encounters (Redolat, Brain & Simon, 1991), hence the clinical use of sulpiride as an anti-aggressive agent.

2.3 Summary

To summarise, facial expressions appear to be processed by at least partially separable neurocognitive (Adolphs, Damasio, Tranel & Damasio, 1996) and pharmacological (Zangara et al., 2002) systems. Serotonergic manipulations appear to affect the processing of fearful and happy facial expressions, while noradrenergic manipulations seem to affect processing of sad facial expressions. Both are likely to be mediated by the amygdala. GABAergic and possibly dopaminergic manipulations (although the evidence is limited at the current time) appear to affect processing of angry facial expressions, recognition of which is likely to be mediated prefrontally, especially by the orbitofrontal cortex. There is no evidence regarding neurotransmitter involvement in disgust, however neurological studies strongly indicate that key structures are the basal ganglia (in particular the caudate), the insula and other somatosensory related cortices.

3. Populations in which FER is impaired

The literature reviewed so far suggests a) that being able to recognise facial expressions has an important social function, and b) that this ability can be compromised, either by damage to those areas of the brain or changes to the neurotransmitters that are involved in FER. With this knowledge, one might predict that impaired ability to recognise facial expressions would have social consequences. Evidence suggests that psychiatric illness and drug use alters neuropharmacological and neurological functioning. The following section will review the literature relating to FER in people with psychiatric illness and substance misuse, and consider how this corresponds to social behaviour.

3.1 Psychiatric illness

3.1.1 Schizophrenia

Impaired FER in schizophrenia has been widely documented (Mandal, Pandey & Prasad, 1998). Hall, Harris, Sprengelmeyer, Sprengelmeyer, Young, Sanots, et al. (2004) found that schizophrenic patients displayed an overall deficit in FER compared with healthy controls, however they do not report specific effects on the different expressions. They also found that when they divided their schizophrenic group into those with and without positive symptoms, it was those patients with positive symptoms that contributed most to this deficit. Similarly, other studies have found a negative correlation between psychotic symptoms and FER accuracy (Schneider, Gur, Gur & Shtasel, 1995). Kohler, Turner, Bilker, Brensinger, Siegel, Kanes, et al. (2003) assessed FER in stable schizophrenic patients and found that overall, their schizophrenic patients had impaired FER, particularly for the

expression of fear and disgust. These patients also misinterpreted neutral expressions as emotional more often than controls, showing a negative bias. The authors discuss the meaning of this in relation to understanding psychotic symptoms, such as perceiving neutral events as personally significant.

Phase of illness has also been found to differentiate schizophrenic patients' ability to recognise facial expressions, with remitted patients performing better than those who are acutely unwell (Gessler, Cutting, Frith & Weinman, 1989). Patients with first episode psychosis have been found to perform worse than patients with affective psychosis and control subjects, especially on recognition of fear and sadness (Edwards, Pattison, Jackson & Wales, 2001).

Poor interpersonal functioning characterises this patient group (Hall et al., 2004), and it has been suggested that inaccurate recognition of emotions may underlie this (Poole, Tobias & Vinogradov, 2000). In addition to looking at FER, the aforementioned study by Hall et al. also assessed social cognition in their schizophrenic participants, finding highly significant impairments across tests of this domain.

3.1.2 Bipolar Disorder

Less research has been conducted with patients with a diagnosis of bipolar disorder. The results of a study by Lembke and Ketter (2002) suggest that manic patients have worse overall recognition of facial expressions than euthymic subjects with a diagnosis of bipolar disorder and healthy controls, particularly for the expressions of fear and disgust. These authors found a tendency for manic patients to mistake

disgust for anger, and fear for surprise, and relate this to aspects of social interaction which characterise manic patients, such as persistent approach behaviours. Interestingly, recognition of happiness was completely preserved in manic patients, and there was a negative correlation between scores on the Young Mania Rating Scale and recognition of sad faces. This suggests that the ability to recognise emotion may vary with the degree of mood disturbance.

McClure, Pope, Hoberman, Pine and Leibenluft (2003) investigated how accurately adolescents with bipolar disorder recognised the facial expressions of both peers and adults. They found that bipolar patients performed worse on FER than anxious adolescents and healthy comparison subjects. The bipolar adolescents were more likely to misinterpret the happy, sad and fearful expressions of the peers as angry, however this impairment was not present for the faces of adults. The authors do not state whether their participants were manic, euthymic or depressed at the time of testing. McClure et al. propose that such a bias in FER may predispose such a population to negative peer interactions.

A study with slightly different findings was conducted by Harmer, Grayson and Goodwin (2002b). While they found generally impaired FER performance in euthymic bipolar patients compared with healthy controls, *enhanced* recognition of disgust was displayed and fewer false positive disgust expression identifications were made. The authors discuss the impact of bipolar mood disorder on the basal ganglia, in particular the caudate, both known to be involved in processing disgust reactions. Neuroimaging studies suggest that caudate volume may be increased in bipolar patients relative to matched controls (Aylward, Roberts-Twillie, Barta, Kumr,

Harris, Geer, et al., 1995), in addition to increased levels of neural activity within the caudate during periods of acute mania (Blumberg, Stern, Martinez, Ricketts, de Asis, White, et al., 2000). They also consider whether more accurate recognition of disgust could be associated with the negative self-perception that has been found in bipolar patients (Lyon, Startup & Bentall, 1999), in particular low self-esteem and increased anxiety. However, they do not elucidate the mechanism by which this could happen.

3.1.3 Depression

Research with depressed participants has looked at both the accuracy of FER and also how FER is related to the course of depressive illness. The evidence is somewhat inconsistent, but tends to point to impaired overall FER relative to non-depressed controls (Gur, Erwin, Gur, Zwil, Heimberg & Kraemer, 1992). Which emotions are specifically impaired varies between studies. A negative bias in FER has been demonstrated with depressed participants (Rubinow & Post, 1992; Hale, 1998), and some studies suggest a correlation between depressed mood and negatively biased impaired FER (Gur et al., 1992). It may be that one of the effects of antidepressant medication is to correct this negative information processing bias, as suggested by Harmer et al.'s (2003c) study in which reboxetine was administered to healthy volunteers, producing a positive recognition bias.

Hale (1998) found that depressed patients tended to judge facial expressions more negatively (i.e. they identified more negative than positive expressions), and saw more sadness in the faces, whether the expressions were ambiguous (i.e. mixed with other emotions) or not. Interestingly, he also found that the disposition to judge facial expressions negatively, in both ambiguous and non-ambiguous faces was strongly

correlated with the severity and persistence of their depressive illness. In contrast, Bouhuys, Geerts, Mersch and Jenner (1996) found that the depressed patients in their study who were less sensitive to expressions of sadness, rejection or anger on admission experienced less symptomatic improvement over the course of their depressive episode.

Both sets of authors hypothesise about the role of facial expression decoding abilities in the interpersonal relationships of depressed patients. Hale cites Gotlib and Hamen's (1992) suggestion that the negative expectancies of depressed people, and readiness to attend to negative aspects of their social surroundings may lead to feelings of rejection, resulting in decreased social support. In this way, their negative perception of others' potentially ambiguous facial expressions may account for their interpersonal difficulties. On the other hand, on the basis of their findings, Bouhuys et al. hypothesise that hyposensitivity to others' facial expressions is related to persistence of depression via a similar mechanism. They discuss the possibility that depressed people who are less able to perceive negative emotions of others will engage in more unwelcome help-seeking behaviour, resulting in rejection.

3.1.4 Obsessive-Compulsive Disorder (OCD)

The research evidence pertaining to impaired FER in patients with OCD is inconsistent, possibly due to the heterogeneous nature of the disorder. Sprengelmeyer, Young, Pundt, Sprengelmeyer, Calder, Berrios, et al. (1997) found that patients with OCD and patients with Tourette's syndrome accompanied by obsessive or compulsive behaviours exhibited impaired disgust recognition, while patients with Tourette's syndrome but no OCD features did not. The authors found

that patients with OCD or OCD features frequently misclassified expressions of disgust as anger. Patients with anxiety disorders (panic disorder or GAD) were comparable to healthy controls in their recognition of disgust.

In contrast, Parker, McNally, Nakayama, and Wilhelm (2004) failed to replicate these results, finding no significant differences between OCD patients and healthy control subjects. Only one of their OCD participants displayed a specific disgust recognition deficit, who interestingly was the participant with the most severe OCD symptomatology. They conclude that the impairments may only occur in cases of severe OCD. Two other studies have used OCD patients and healthy volunteers as control groups when looking at FER in detoxified alcoholics and Body Dysmorphic Disorder (Kornreich, Blairy, Philippot, Dan, Foisy, Le Bon, et al. 2001a; Buhlmann, McNally, Etcoff, Tuschen-Caffier & Wilhelm., 2004, respectively). Neither found impaired disgust recognition in their OCD sample. A further study by Montagne, Kessels, de Geus, Denys, de Haan and Westenberg (2005) found that OCD patients were *more* sensitive to all emotional expressions than healthy controls, but failed to find a disgust-specific effect in the group as a whole. However, their results showed that those OCD patients with more severe symptomatology demonstrated more accurate disgust recognition than controls.

There is evidence to suggest that abnormalities exist in the basal ganglia of obsessive-compulsive disorder patients (Parker et al., 2004), the basal ganglia also being implicated in the processing of disgust expressions (Adolphs, 2002). It has been suggested that abnormal experience of disgust may be involved in the genesis of obsessions and compulsions (Power & Dalgleish, 1997), as so many OCD patients

exhibit heightened disgust in reaction to stimuli they regard as contaminated (Parker et al., 2004). Intuitively, it might be expected that OCD patients would be hypersensitive to disgust cues, however the research in this area is inconclusive.

3.1.5 Psychopathy and Acquired Sociopathy

People diagnosed with psychopathy have been found to demonstrate markedly impaired FER for fear and sadness (Blair, Colledge, Murray & Mitchell, 2001) and also disgust (Kosson, Suchy, Mayer & Libby, 2002). The case of psychopathy is particularly interesting when considering the impact of impaired FER on social behaviour, given that antisocial behaviour and unstable relationships are diagnostic markers for psychopathy. It is proposed that psychopathic individuals have a limited capacity for understanding and experiencing emotion, anticipating the emotional consequences of their behaviour and do not learn from punishment (Blair, 2001). Blair's (1995) account of psychopathy links the condition to early amygdala dysfunction and consequent impairments in processing fearful and sad emotions. It has been found that compared with normal individuals, psychopaths show reduced activation of the amygdala during aversive conditioning tasks (Veit, Flor, Erb, Hermann, Lotze, Grodd & Birbaumer, 2002). Unlike non-psychopaths, psychopaths also fail to exhibit startle potentiation while viewing slides depicting mutilation (Levenston, Patrick, Bradley & Lang, 2000). If, as has been suggested, disgust is important in negative socialisation, longstanding insensitivity to other's disgust could conceivably contribute to poorly socialised behaviour, as seen in the psychopathic population.

Damasio, Tranel and Damasio (1990) introduced the term 'acquired sociopath', to refer to individuals who, following acquired lesions to the orbitofrontal cortex, fulfil criteria for DSM-III diagnosis of sociopathic disorder. Such individuals present with frustration or threat induced reactive aggression (Blair & Cipolotti, 2000). Acquired sociopaths have been found to display generally impaired FER, but particularly for expressions of anger (Blair & Cipolotti, 2000; Hornak, Rolls & Wade, 1996). This finding is consistent with the hypothesis that areas of the prefrontal cortex are responsible for recognising and responding to expressions of anger.

3.2 Drugs and Alcohol

3.2.1 Alcohol

Alcohol appears to have different effects on FER depending on the dose administered. As mentioned in 2.2.3, acute high dose alcohol use appears to have a significant impact on FER in normal social drinkers, particularly affecting the recognition of anger (Borrill et al., 1987). However, in low doses, alcohol has been found to enhance FER (Borrill et al., 1987). Kano, Gyoba, Kamachi, Mochizuki, Hongo and Yanai (2003) found that administration of a low dose of alcohol to healthy young men was associated with significantly better discrimination of happy faces, but that this performance deteriorated with higher doses. This corresponds with the differential effects of alcohol at high and low doses on social behaviour. Initially alcohol increases sociability and talkativeness (Kano et al., 2003), while at higher doses it is related to interpersonal aggression (Chermack & Giancola, 1997) and poor interpersonal functioning (Kornreich, Philippot, Foisy, Blairy, Raynaud, Dan, et al., 2002).

Chronic alcohol use has also been associated with both poor FER (Kornreich, Blairy, Philippot, Dan, Foisy, Le Bon, et al., 2001a) and interpersonal difficulties (Kornreich et al., 2002). Philippot, Kornreich, Blairy, Baert, Den Dulk, Le Bon, et al., (1999) found impaired FER in recently detoxified alcoholics, with significant deficits in recognising anger, sadness, happiness and disgust and a special bias towards overattribution of anger and contempt. Similarly, Townshend and Duka (2003) found a group difference between two-week detoxified alcoholic patients and controls in the recognition of anger and disgust. The alcoholic group underestimated the presentation of anger compared to controls, frequently misidentifying disgust as anger. Research has also found that recently detoxified alcoholics overestimate the emotional intensity of facial stimuli (Oscar-Berman, Hancock, Mildwordf, Hutner &Altman-Weber, 1990; Philippot et al., 1999; Verbanck, 2001). Furthermore, deficits in recognition of anger, disgust and to a lesser extent sadness, appear to persist even after abstinence from alcohol of two months or more. Verbanck (2001) found specifically impaired anger and disgust recognition in alcoholics who had been detoxified for at least 2-months, compared with healthy controls. In contrast, ratings of emotional intensity return to the level of normal controls with abstinence (Kornreich et al., 2001; Verbanck, 2001). Kornreich et al., (2002) found that selfreported interpersonal difficulties correlated with FER deficits in a sample of 30 alcoholics, although no causal relationship can be inferred from this.

3.2.2 Ecstasy

Research with ecstasy users and FER is still in its infancy. As already mentioned in 2.2.1, Hoshi et al. (2004) found that ecstasy use impacted on fear recognition, enhancing recognition of fearful expressions following acute administration and

impairing this ability four days later. Hoshi et al. also used a measure of aggression at these two time points, with the finding that users had higher self rated aggression scores than controls at day 4, despite there being no difference in this rating on the night of drug use.

3.2.3 Benzodiazepines

As already reviewed in 2.2.3, benzodiazepines have been seen to selectively impair the recognition of anger (Blair & Curran, 1999), and in one study also fear (Zangara et al., 2002). While benzodiazepines are used for their anxiolytic properties, they have been noted to have 'paradoxical' side-effects such as disinhibition, hostility and aggression. Chronic alprazolam use has been found to increase behavioural aggression in lab settings, particularly under conditions of provocation (Bond, Curran, Bruce, O'Sullivan & Shine, 1995). However, this is not accompanied by subjective ratings of increased anger or hostility, reflecting a lack of insight into the emotional changes.

3.3 Summary

In summary, FER is impaired in many psychiatric and drug-using populations. Impairments can be general, affecting the accuracy of recognition of many or all expressions (e.g. in schizophrenia), and also specific to certain emotions (e.g. in psychopathy and with acute use of benzodiazepines). Broadly-speaking, it appears that those populations which demonstrate impaired recognition of anger and fear (e.g. psychopaths, manic patients, alcohol and benzodiazepine use) are characterised by less sensitive social behaviour. This generally seems to take the form of aggressive or persistent approach behaviours. Impaired recognition of sad

expressions (e.g. in manic patients, and in some depressed patients) also appears to be associated with poor social behaviour and a less favourable outcome in depression.

It also appears that several populations show interpretation biases (e.g. depressed, bipolar and alcoholic patients), and that these biases may correspond to mood state, social behaviour and course of illness in the case of depression.

Other populations demonstrate enhanced FER (e.g. enhanced recognition of happiness with low dose alcohol use and acute antidepressant treatment) which may correspond to prosocial behaviour and improved mood state.

4. Opiate Use

One major class of drugs which is underrepresented in the literature on emotion processing is that of opiates. Morphine, codeine, heroin and methadone are all opiates that are widely used for pain relief, as drugs of abuse or as treatment for drug abuse. The prevalence of heroin use is hard to gauge as it is largely a hidden problem. However, a recent estimation suggested a prevalence of 3.7% among men aged 25-44 in London (Hickman, Higgins, Hope & Bellis, 2004). Every year, community pharmacies across England dispense over 1.25 million NHS prescriptions for methadone, suggesting that about 50,000 opiate users are receiving methadone at any one time (Strang & Sheridan, 2003). Despite such high levels of use, we know relatively little about the effects of this class of drug upon psychological functioning. It is of note that heroin use almost never occurs in isolation, with cocaine,

amphetamines and benzodiazepines also being widely used by drug misusers seeking treatment for heroin dependence (Gossop, 2005). The National Treatment Outcomes Research Study (Gossop, Marsden, Stewart, Lehmann, Edwards, Wilson & Segar, 1998) found that almost two thirds of drug users in treatment had been using three or more substances before admission to treatment, and more than a third were using stimulants on a frequent basis. The most commonly used stimulant among drug misusers seeking treatment for heroin dependence was crack cocaine (Gossop et al., 1998).

4.1 Different kinds of opiates

Opiates can be classified into two fundamentally different groups; morphine-like agonists and opiates producing mixed actions. Morphine-like agonists act primarily at mu receptors, but also at kappa and delta receptors. They are strong analgesics, indicated for use in clinical situations requiring moderate to severe pain relief. Morphine, heroin and methadone are examples of opiates in this class. Opioids producing mixed actions are associated with a lower level of analgesia, and are clinically indicated for mild to moderate pain. They can be subdivided into mixed agonist-antagonist such as buprenorphine, and partial agonists such as codeine.

Treatment of heroin abuse involves substituting longer acting morphine-like agonists such as methadone for heroin (Zacny, 1995). There is now substantial evidence for the effectiveness of methadone maintenance treatment for opiate users (National Treatment Agency, 2004). Methadone treatment is also associated with improvements in health and social functioning, lower levels of crime and a lower risk of premature mortality (National Treatment Agency, 2004).

4.2 Neuropharmacological Action of opiates

Like virtually all drugs of abuse, opiates enhance dopamine release in the nucleus accumbens by increasing the activity of dopamine neurons in the ventral tegmental area. It is thought that opiates activate mu opioid receptors located on GABA neurons within the ventral tegmental area. Opiates also have dopamine-independent effects within the nucleus accumbens, which play an important part in opiate reward. Pharmacological challenges with drugs that interact with dopamine receptors have demonstrated that the activity of brain dopaminergic systems is altered by long term opiate use in heroin addicts (Casas, Guardia, Prat & Trujols, 1995).

4.3 Interpersonal functioning and personality in opiate users

Most opiate users seeking treatment present with a range of difficulties including severe family and social problems (National Treatment Agency, 2004). Research indicates that poor interpersonal functioning is present among opiate users (Meulenbeek, 2000).

Increased aggression is one of the primary features discussed in the literature surrounding opiate users' interpersonal functioning, although with inconsistent research findings. Hoaken (2003) highlights four possible reasons for the existence of a relationship between drug use and aggression. Firstly, aggression may be a result of the direct action of the drug on behaviour. Secondly, aggression may be used to get drugs or get the resources necessary to buy drugs. Thirdly, aggression and drug use may both be the result of a personality factor such as high sensation-seeking or impulsivity. Lastly, withdrawal effects of the drug may increase aggression.

While animal research suggests that opiates temporarily reduce aggressive behaviour (e.g. Miczek, Weerts & DeBold, 1993), controlled human studies have demonstrated heightened aggression on laboratory measures in participants administered codeine (Spiga, Cherek, Roache & Cowan, 1990) and morphine (Berman, Taylor & Marged, 1993) compared to controls. In a more naturalistic study, Morentin, Callado and Meana (1998) reviewed the files of 578 arrestees in Spain, concluding that among those who used heroin, aggression towards arresting police or non-fatal violent offences against other people were *less* frequent than among arrestees with no drug or psychiatric history. In contrast, Gerra, Amir, Raggi, Giusti, Delsignore, Bertacca and Brambilla (2001) found increased aggression in methadone maintained participants compared with healthy controls, using a laboratory measure of aggression, but concluded that this was more related to personality traits than drug effects.

Fassino, Daga, Delsedime, Rogna & Goggio (2004) summarise the main conclusions drawn from research which has sought to classify the personality type of heroin users. Firstly, personality disorder is commonly found in heroin users, with prevalence figures of around 50%, in which borderline, antisocial and dependent types are over-represented (Fassino et al., 2004). Secondly, two subtypes of heroin users have been identified; the first characterised by a low level of psychopathology and good relational skills, and a second with high levels of personality disorder, severe relational difficulties and a worse prognosis.

A dimensional approach has also been taken to identify specific personality traits of heroin users, with Khantzian (1997) suggesting that heroin users are "immature and unable to take care of themselves". Other studies propose that users have poor self-esteem (Fieldman, Woolfolk & Allen, 1995), unstable personal relationships and identities (Fassino, Scarso, Barbero, Taylor, Pezzini & Furlan, 1992) and poorly regulated emotions and impulse control (Bartholomew, Sher & Wood, 2000; Conway, Kane, Ball, Poling & Rounsaville, 2003).

4.4 Opiates and recognition of facial expressions

The literature reviewed so far demonstrates that FER is impaired in many populations that are characterised by poor social behaviour. Moreover, it suggests that the specific nature of the FER impairment may be connected to the type of social functioning deficit. Given the interpersonal difficulties encountered by opiate users, in addition to the pharmacological changes that are brought about by opiate use, it is possible that accuracy (and/or speed) of FER is affected in this population.

The only published study in this area is by Kornreich, Foisy, Philippot, Tecco, Noel, Hess, et al. (2003), who compared FER in five groups of participants: 1) methadone maintained, 2) recently detoxified alcoholics, 3) recently detoxified alcohol and opiate users, 4) recently detoxified opiate users and 5) normal controls. They found that the recently detoxified alcoholic group and mixed detoxified alcohol and opiate group scored significantly lower on accuracy of FER than normal controls. The methadone maintained group and recently detoxified opiate group accuracy scores were better than those of the mixed detoxified alcohol and opiate group and detoxified alcohol only group, but worse than the normal controls. Unfortunately this

study did not differentiate between the individual emotional expressions. The authors also found that the length of abstinence in the opiate abstinent group did not influence FER scores. This could be taken to mean that opiates do not have a detrimental effect on FER and that pre-existing deficits account for the lower scores than normal controls, or that the impairments caused by opiate use persist long after discontinuation, as was found with alcoholic subjects (Kornreich et al., 2001). The lack of research investigating specific FER impairments in opiate users constitutes a significant gap in the literature, given the prevalence of opiate use today. Any significant findings could be of use in better understanding the specific difficulties faced by opiate users, and may provide a target for intervention.

5. Methodological issues in the study of FER

The differing methodology used by studies of FER make it more difficult to draw the literature together conclusively. Different versions of the faces task have been used across studies. Some researchers (e.g. Bouhuys et al., 1996; Hale, 1998) have used a set of 12 schematic faces (i.e. line drawings) where the key differential features are eyebrow and mouth types. They argue that the recognition of emotional facial expressions is of such importance that expressions can be detected from highly abstract facial displays, and that line drawings are free from cultural differences in interpretation. Other studies have used photographs of different people (of different gender and race), each depicting one of the six expressions. It has been suggested that this method may introduce factors other than expression that could influence participants' judgement of emotions, such as the age, gender and/or attractiveness of the photographed person (Hale, 1998). The range of expressions used in faces tasks

also varies, with some looking only at selected emotions (e.g. McClure, Pope, Hoberman, Pine & Leibenluft, 2003; Surguladze, Young, Senior, Brebion, Travis & Phillips, 2004), some just looking at overall impairment (e.g. Kornreich et al., 2003), and some investigating all of the six basic emotions (e.g. Hoshi et al., 2004). A useful extension of the basic expression recognition paradigm that has been used by several researchers, is the investigation of specific misidentification error patterns (e.g. Lembke & Ketter, 2002) which can provide information about specific biases present in populations. A further variant is the separation of facial expressions into high and low intensity stimuli (e.g. Kohler et al., 2003), in order to look at whether impairments are present for subtle expressions.

The most sophisticated version of the faces task uses stimuli from the Facial Expressions of Emotions: Stimuli and Tests (FEEST; Young, Perrett, Calder, Sprengelmeyer & Ekman, 2002), and is being increasingly used in FER research (e.g. Harmer et al., 2001; 2003a; Hoshi et al., 2004; Lawrence et al., 2002). It consists of a set of photographs from the Ekman and Freisen (1976) series, and shows 30 photographs of a single individual displaying different expressions that are morphed together to differing degrees. This creates subtle blends of expressions which require more stringent discrimination, and the use of a single individual reduces response variability. This version of the task also looks at response reaction time in addition to accuracy of recognition, which allows for more meaningful interpretation of results, for example detection of speed-accuracy trade-offs.

In summary, there are various tools available for investigating facial expression recognition, some of which may be better suited to certain research questions than

others. However, for looking at expression-specific impairments, the FEEST (Young et al., 2002) provides a comprehensive measure of FER which is able to pick up subtle deficits.

References

Adolphs, R. (2002). Neural systems for recognising emotion. Current Opinion in Neurobiology, 12, 169-177.

Adolphs, R., Damasio, H., Tranel, D., Cooper, G. and Damasio, A.R. (2000). A role for somatosensory cortices in the visual recognition of emotion as revealed by 3-D lesion mapping. *Journal of Neuroscience*, 20, 2683-2690.

Adolphs, R., Damasio, H., Tranel, D. and Damasio, A.R. (1996). Cortical systems for the recognition of emotions in facial expressions. *Journal of Neuroscienc*, *e* 16, 7678-7687.

Adolphs, R., Tranel, D., Damasio, H. and Damasio, A.R. (1995). Fear and the human amygdala. *Journal of Neuroscience*, 15, 5879-5892.

Adolphs, R., Tranel, D., Hamann, S., Young, A., Calder, A., Anderson, A., Phelps, E., Lee, G.P. and Damasio, A.R. (1999). Recognition of facial emotion in nine subjects with bilateral amygdala damage. *Neuropsychologia*, 37, 1111-1117.

Argyle, M. (1988). Bodily Communication. Second Edition. Routledge.

Attenburow, M.J., Williams, C., Odontiadis, J, Reed, A., Powell, J. and Cowen, P. (2003). Acute administration of nutritionally sourced tryptophan increases fear recognition. *Psychopharmacology*, *169*, 104-107.

Averill, J.R. (1982). Anger and aggression: an essay on emotion. Springer, New York.

Aylward, E.H., Roberts-Twillie, J.V., Barta, P.E., Kumr, A.J., Harris, G.J. and Geer, M. (1994). Basal ganglia volumes and white matter hyperintensities in patients with bipolar disorder. *American Journal of Psychiatry*, 151, 687–693.

Bartholomew, B.D., Sher, K.J. and Wood, M.D. (2000). Personality and substance use disorders: a prospective study. *Journal of Clinical and Consulting Psychology*, 68, 818-829.

Berman, M., Taylor, S. and Marged, B. (1993). Morphine and human aggression. *Addictive Behaviours*, 18, 263-268.

Blair, R.J.R. (1995). A cognitive developmental approach to morality: investigating the psychopath. *Cognition* 57, 1-29.

Blair, R.J.R (2003). Facial expressions, their communicatory functions and neuro-cognitive substrates. *Philosophical Transactions of the Royal Society of London B.*, 358, 561-572.

Blair, R.J.R. and Cipolotti, L. (2000). Impaired social response reversal: A case of 'acquired sociopathy'. *Brain*, 123,1122-1141.

Blair, R.J.R., Colledge, E., Murray, L. and Mitchell, D.G. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology*, 29, 491-498.

Blair, R.J.R. and Curran, H.V. (1999). Selective impairment in the recognition of anger induced by diazepam. *Psychopharmacology*, 147, 335-338.

Blair, R.J.R., Morris, J.S., Frith, C.D., Perrett, D.I. and Dolan, R. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain*, 122, 883-893.

Blumberg, H.P., Stern, E., Martinez, D., Ricketts, S., de Asis, J. and White T. (2000). Increased anterior cingulate and caudate activity in bipolar mania. *Biological Psychiatry*, 48, 1045–1052.

Bond, A.J., Curran, H.V., Bruce, M., O'Sullivan, G. and Shine, P. (1995). Behavioural aggression in panic disorder after 8 weeks treatment with alprazolam. Journal of Affective Disorders, 35, 117-123.

Borrill, J.A., Rosen, B.K. and Summerfield, A.B. (1987). The influence of alcohol on judgement of facial expressions of emotion. *British Journal of Medical Psychology*, 60, 71-77.

Bouhuys, A.L., Geerts, E., Mersch, P.P.A and Jenner, J.A. (1996). Nonverbal interpersonal sensitivity and persistence of depression: Perception of emotions in schematic faces. *Psychiatry Research*, 64, 193-203.

Buhlmann, U., McNally, R.J., Etcoff, N.L., Tuschen-Caffier, B. and Wilhelm, S. (2004). Emotion recognition deficits in body dysmorphic disorder. *Journal of Psychiatry Research*, 38, 201-6.

Burgoon, J.K. (1985). Nonverbal signals. In M.L. Knapp & G.R. Miller (Eds.), Handbook of Interpersonal Communication (pp. 344-390). Sage Publications, Beverly Hills, CA.

Casas, M., Guardia, J., Prat, G. and Trujols, J. (1995). The apomorphine test in heroin addicts. *Addiction*, 90, 831-5.

Chermack, S. and Giancola, P.R. (1997). The relationship between alcohol and aggression: an integrated biopsychosocial approach. *Clinical Psychology Review*, 6, 621-649.

Chevalier-Skolnikoff, S. (1973). Facial expressions of emotion in non-human primates. In P. Ekman (Ed.), *Darwin and Facial Expression* (pp. 11-98.) New York: Academic Press.

Conway, K.P., Kane, R.J., Ball, S.A., Poling, J.C. and Rounsaville, B.J. (2003). Personality, substance choice and polysubstance involvement among substance dependent patients. *Drug and Alcohol Dependence*, 71, 65-75.

Cornell, D.G., Warren, J., Hawk, G., Stafford, E., Oram, G. and Pine, D. (1996). Psychopathy in instrumental and reactive violent offenders. *Journal of Consulting and Clinical Psychology*, 64, 783-790.

Cubero, I., Thiele, T.E. and Bernstein, I.L. (1999). Insular cortex lesions and taste aversion learning: effects of conditioning method and timing of lesion. *Brain Research*, 839, 323-330.

Damasio, A.R., Tranel, D. and Damasio, H. (1990). Individuals with sociopathic behaviour caused by frontal damage fail to respond automatically to social stimuli. *Behaviour Brain Research*, 41, 81-94.

Darwin, C. (1872). The expressions of the emotions in man and animals. London: Albemarle.

Dias, R., Robbins, T.W. and Roberts, A.C. (1996). Dissociation in prefrontal cortex of affective and attentional shifts. *Nature*, 380, 69-72.

Dimberg, U. and Ohman A. (1996). Behold the wrath: Psychophysiological responses to facial stimuli. *Motivation and Emotion*, 20, 149-182.

Edwards, J., Pattison, P.E., Jackson, H.J. and Wales, R.J. (2001). Facial affect and affective prosody recognition in first episode schizophrenia. *Schizophrenia Research*, 48, 235-253

Ekman, P. (1957). A methodological discussion of nonverbal behaviour. *Journal of Psychology*, 43, 141—149.

Ekman, P. (1992). An argument for basic emotions. *Cognition and Emotion*, 6, 169-200.

Ekman, P. (1999a). Basic Emotions. In T. Dalgleish, and M. Power (Eds.), *The Handbook of Cognition and Emotion* (pp. 45-60). Sussex, UK: John Wiley and Sons Ltd.

Ekman, P. (1999b). Facial Expressions. In T. Dalgleish, and M. Power (Eds.), *The Handbook of Cognition and Emotion* (pp. 301-320). Sussex, UK: John Wiley and Sons Ltd.

Ekman, P. and Friesen, W.V. (1976). Facial action coding system. Palo Alto, Consulting Psychologists Press.

Ekman, P., Friesen, W. V., & Ellsworth, P. (1972). Emotion in the human face: Guidelines for research and an integration of findings. New York: Pergamon Press.

Ekman, P. and Yamey, G. (2004). Emotions revealed: recognising facial expressions. BMJ Career Focus, 328, 75-76.

Fassino, S., Abbate Daga, G., Delsedime, N., Rogna, L. and Goggio, S. (2004). Quality of life and personality disorders in heroin abusers. *Drug and Alcohol Dependence*, 76, 73-80.

Fassino, S., Scarso, G., Barbero, L., Taylor, J., Pezzini, F. and Furlan, P.M. (1992). The image of self and of the environment in drug abusers: a comparative study using the TAT. *Drug and Alcohol Dependence*, 30, 253-261.

Fieldman, N.P., Woolfolk, R.L. and Allen, L.A. (1995). Dimensions of self-concept: a comparison of heroin and cocaine addicts. *American Journal of Drug and Alcohol Abuse*, 21, 315-326.

Fine, C. and Blair, R.J.R. (2000). Mini review: the cognitive and emotional effects of anygdala damage. *Neurocase*, 6, 435-450.

Fridland, A.J. (1991). Sociality of solitary smiling: potentiation by an implicit audience. *Journal of Personality and Social Psychology*, 60, 229-246.

Fridland, A. J. (1994). *Human Facial Expression: An Evolutionary View*. San Diego. CA: Academic Press.

Galati, D., Scherer, K.R. and Ricci-Bitti, P.E. (1997). Voluntary facial expression of emotion: comparing congenitally blind with normally sighted encoders. *Journal of Personality Sociology and Psychology*, 73, 1363-79.

Gerra, G., Amir, Z., Raggi, M.A., Giusti, F., Delsignore, R., Bertacca, S. and Brambilla, F. (2001). Aggressive responding of male heroin addicts under methadone treatment: psychometric and neuroendocrine correlates. *Drug and Alcohol Dependence*, 65, 85-95.

Gessler, S., Cutting, J., Frith, C.D. and Weinman, J. (1989). Schizophrenic inability to judge facial emotion: a controlled study. *British Journal of Clinical Psychology*, 28, 19-29.

Gossop, M. (2005). Treatment outcomes: what we know and what we need to know. National Treatment Agency for Substance Misuse.

www.nta.nhs.uk/publications/docs/te2.pdf (retrieved 10.06.05).

Gossop, M., Marsden, J., Stewart, D., Lehmann, P., Edwards, C., Wilson, A. and Segar, G. (1998). Substance misuse, health and social problems of clients at 54 drug treatment agencies: intake data from the National Treatment Outcome Research Study (NTORS). *British Journal of Psychiatry*, 173, 166-171.

Gotlib, I.H. and Hamen, C.L. (1992). Psychological aspects of depression: toward a cognitive-interpersonal integration. John Wiley, Chichester.

Gur, R.C., Erwin, R.J., Gur, R.E., Zwil, A.S., Heimberg, C. and Kraemer, H.C. (1992). Facial emotion discrimination: II. Behavioral findings in depression. *Psychiatry Research*, 42, 241-251.

Hale, W.W. (1998). Judgment of facial expressions and depression persistence. Psychiatry Research 80, 265-274.

Hall, J., Harris, J.M., Sprengelmeyer, R., Sprengelmeyer, A., Young, A.W., Sanots, I.M., Johnstone, E.C. and Lawrie, S.M. (2004). Social cognition and face processing in schizophrenia. *The British Journal of Psychiatry*, 185, 169-170.

Harmer, C.J., Bhagwagar, Z., Cowen, P.J. and Goodwin G.W. (2001a). Acute administration of citalopram in healthy volunteers facilitates recognition of happiness and fear. *Journal of Psychopharmacology*, 15 (suppl.), A16.

Harmer, C.J., Bhagwagar, Z., Perrett, D.I., Vollm, B.A., Cowen, P. and Goodwin, G. (2003a). Acute SSRI administration affects the processing of social cues in healthy volunteers. *Neuropsychopharmacology*, 28, 148-152.

Harmer, C.J., Grayson, L. and Goodwin, G.M. (2002b). Enhanced recognition of disgust in bipolar illness. *Biological Psychiatry*, 51, 298-304.

Harmer, C.J., Hill, S.A., Taylor, M.J., Cowen, P.J. and Goodwin, G.M. (2003c). Towards a neuropsychological theory of antidepressant drug action: Increase in positive emotional bias after potentiation of norepinephrine activity. *American Journal of Psychiatry*, 160, 990-992.

Harmer, C.J., Perrett, D.I, Cowen, P.J and Goodwin, G.M. (2001b). Administration of the beta-adrenoceptor blocker propranolol impairs the processing of facial expressions of sadness. *Psychopharmacology Berl.*, 154, 383-389.

Harmer, C.J., Rodgers, R.D., Tunbridge, E., Cowen, P. and Goodwin, G. (2003b). Tryptophan depletion decreases the recognition of fear in female volunteers. *Psychopharmacology*, 167, 411-417.

Harmer, C.J., Shelley, N.C., Cowen, P.J. and Goodwin, G.M. (2002a). Repeated administration of citalopram or reboxetine reduces the perception of negative emotions from facial expressions. *Journal of Psychopharmacology*, *S16*, A70.

Harmer, C.J., Shelley, N.C., Cowen, P.J. and Goodwin, G.M. (2004). Increased positive versus negative affective perception and memory in healthy volunteers following selective serotonin and norepinephrine reuptake inhibition. *American Journal of Psychiatry*, 161, 1256-1263.

Harmer, C.J., Thilo, K.V., Rothwell, J.C. and Goodwin, G.M. (2001c). Transcranial magnetic stimulation of medial-frontal cortex impairs the processing of angry facial expressions. *Nature Neuroscience*, 4, 17-18.

Hickman, M., V. Higgins, V. Hope and M. A. Bellis (2004). Estimating prevalence of problem drug use: multiple methods in Brighton, Liverpool and London. Home

Office Online Report 36/04.

http://www.homeoffice.gov.uk/rds/pdfs04/rdsolr3604.pdf. (retrieved 10.06.05)

Hoaken, P.N.S. and Stewart, S.H. (2003). Drugs of abuse and the elicitation of human aggressive behaviour. *Addictive Behaviours*, 28, 1533-1554.

Hornak, J., Rolls, E.T. and Wade, D. (1996). Face and voice expression identification in patients with emotional and behavioural changes following ventral frontal damage. *Neuropsychologia*, 34, 247-261.

Hoshi, R., Bisla, J. and Curran, V. (2004). The acute and sub-acute effects of 'ecstasy' (MDMA) on processing of facial expressions: preliminary findings. *Drug and Alcohol Dependence*, 76, 297-304.

Jones, S.S., Collins, K. and Hong, H.W. (1991). An audience effect on smile production in 10-month old infants. *Psychological Science*, 2, 45-49

Kano, M., Gyoba, J., Kamachi, M., Mochizuki, H., Hongo, M. and Yanai, K. (2003). Low doses of alcohol have a selective effect on the recognition of happy facial expressions. *Human Psychopharmacology*, 18, 131-9.

Keltner, D. and Haidt, J. (1999). Social functions of emotions at four levels of analysis. *Cognition and Emotion*, 13, 505-521.

Khantzian, E.J. (1997). The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harvard Review of Psychiatry*, *4*, 231-244.

Klinnert, M., Campos, J.J., Source, J. Emde R, Svejda M. (1983). Emotions as behaviour regulators: social referencing in infancy. In R. Plutchik and H. Kellerman (Eds.), *Emotions in Early Development* (pp. 57-86). New York, NY: Academic Press.

Kohler, C.G., Turner, T.H., Bilker, W.B., Brensinger, C.M., Siegel, S.J., Kanes, S.J., Gur, R.E. and Gur, R.C. (2003). Facial emotion recognition in schizophrenia: intensity effects and error pattern. *American Journal of Psychiatry*, 160, 1768-1774.

Kornreich, C., Blairy, S., Philippot, P., Dan, B., Foisy, M-L., Le Bon, O., Pelc, I. and Verbanck, P. (2001a). Impaired emotional facial expression recognition in alcoholism compared with obsessive compulsive disorder and normal controls. *Psychiatry Research*, 102, 235-248

Kornreich, C., Blairy, S., Philippot, P., Hess, U., Noel, X., Streel, E., Le Bon, O., Dan, B., Pelc, I. and Verbanck, P. (2001b). Deficits in recognition of emotional facial expression are still present after mid-long term abstinence in alcoholics. *Journal of Studies on Alcohol*, 62, 533-542.

Kornreich, C., Foisy, M-L., Philippot, B.D, Tecco, J., Noel, X., Hess, U., Pelc, I. And Verbanck, P. (2003). Impaired emotional facial expression recognition in alcoholics, opiate dependence subjects, methadone maintained subjects and mixed

alcohol-opiate antecedent subjects compared with normal controls. *Psychiatry Research*, 119, 251-260.

Kornreich, C., Philippot, P., Foisy, M-L., Blairy, S., Raynaud, E., Dan, B., Hess, U., Noel, X., Pelc, I. and Verbanck, P. (2002). Impaired emotional facial expression recognition is associated with interpersonal problems in alcoholism. *Alcohol and Alcoholism*, 37, 394-400.

Kosson, D.S., Suchy, Y., Mayer, A.R. and Libby, J. (2002). Facial affect recognition in criminal psychopaths. *Emotion*, 2, 398-411.

Lawrence, A.D., Calder, A.J., McGowan, S.W. and Grasby, P.M. (2002). Selective disruption of the recognition of facial expressions of anger. *Neuroreport*, 13, 881-884.

Lembke, A. and Ketter, T.A. (2002). Impaired recognition of facial emotion in mania. *American Journal of Psychiatry*, 159, 302-304.

Leppanen, J.M. and Hietanen, J.K. (2004). Positive facial expressions are recognised faster than negative facial expressions, but why? *Psychological Research*, 69, 22-9.

Levenston, G.K., Patrick, C.J., Bradley, M.M. Lang, P.J. (2000). The psychopath as observer: Emotion and attention in picture processing. *Journal of Abnormal Psychology*, 109, 373–385.

Lyon, H.M., Startup M. and Bentall R.P. (1999). Social cognition and the manic defense: Attributions, selective attention, and self-schema in bipolar affective disorder. *Journal of Abnormal Psychology*, 108, 273–282.

Mandal, M.K., Pandey, R. and Prasad, A.B. (1998). Facial expressions of emotions and schizophrenia: a review. *Schizophrenia Bulletin*, 24, 299-412.

Matthews, G. and Wells, A. (1999). The cognitive science of attention and emotion. In T. Dalgleish and M.J. Power (Eds.), *Handbook of Cognition and Emotion* (pp. 171-192). New York: Wiley.

McClure, E.B., Pope, K., Hoberman, A.J., Pine, D.S. and Leibenluft, E. (2003). Facial expression recognition in adolescents with mood and anxiety disorders. American Journal of Psychiatry, 160, 1172-1174.

Meulenbeek, P.A.M. (2000). Addiction problems and methadone treatment. *Journal of Substance Abuse Treatment*, 19, 2171-2174

Miczek, K.A. Weerts, E.M. and DeBold, J.F. (1993). Alcohol, benzodiazepine-GABA receptor complex and aggression: Ethological analysis of individual differences in rhodents and primates. *Journal of Studies on Alcohol (suppl. 11)*, 170-179.

Mineka, S. and Cook, M. (1993). Mechanisms involved in the observational learning of fear. *Journal of Experimental Psychology Gen.* 122, 23-38.

Montagne, B., Kessels, R.P.C., de Geus, F., Denys, D., de Haan, E.F.H. and Westenberg, H.G.M. (2005). Recognition of facial expressions in obsessive-compulsive disorder: A dimensional approach. In B. Montagne (Ed.), *Perception of facial expressions in psychiatric and neurological disorders (pp.93-108*). Febodruk B.V. Netherlands.

Morentin, B., Callado, L.F. and Meana, J.J. (1998). Differences in criminal activity between heroin abusers and subjects without psychiatric disorders-analysis of 578 detainees in Bilbao, Spain. *Journal of Forensic Science*, 43, 993-999.

National Treatment Agency for Substance Misuse (2004). Enhancing outcomes of methadone maintenance treatment with counselling and other psychosocial and 'ancillary' services. Source: NTA Briefing documents on methadone treatment: National Treatment Agency for Substance Misuse, UK, 31 May 2004. www.nta.nhs.uk/publications/research_briefing4.htm (retrieved 01.06.05)

Oatley and Johnson-Laird (1987). Towards a cognitive theory of emotions. Cognition and Emotion, 1, 29-50.

Oscar-Berman, M., Hancock, M., Mildwordf, B., Hutner, N. and Altman-Weber, D. (1990). Emotional perception and memory in alcoholism and aging. *Alcoholism:* Clinical and Experimental Research, 14, 384-393.

Parker, H.A., McNally, R.J., Nakayama, K. and Wilhelm, S. (2004). No disgust recognition deficit in Obsessive-Compulsive disorder. *Journal of Behaviour Therapy and Experimental Psychiatry*, 35, 183-192.

Philippot, P, Kornreich, C., Blairy, S., Baert, I., Den Dulk, A., Le Bon, O., Streel, E., Hess, U., Pelc, I. and Verbanck, P. (1999). Alcoholics deficits in the decoding of emotional facial expression. *Alcoholism: Clinical and Experimental Research*, 23, 1031-1038.

Poole, J.H., Tobias, F.C. and Vinogradov, S. (2000). The functional relevance of affect recognition errors in schizophrenia. *Journal of the International Neuropsychological Society*, 6, 649-658

Power, M. and Dalgleish, T. (1997). Cognition and Emotion: From order to disorder. Psychology Press Ltd.

Rapcsak, S.Z., Galper, S.R., Comer, J.F., Reminger, S.L., Nielsen, L., Kaszniak, A.W., Verfaellie, M., Laguna, J.F., Labiner, D.M. and Cohen, R.A. (2000). Fear recognition deficits after focal brain damage: A cautionary note. *Neurology*, *54*, 575-581.

Redican, W.K. (1982) An evolutionary perspective on human facial displays. In P. Ekman (Ed.), *Emotion in the Human Face, 2nd Edition*. (pp.212-280.) Elmsford, New York: Pergamon.

Redolat, R., Brain, P.F. and Simon, V.M. (1991). Sulpiride has an antiaggressive effect in mice without markedly depressing motor activity. *Neuropharmacology*, 30, 41-46.

Rozin, P., Haidt, J. and McCauley, C.R. (1993) Disgust. In M. Lewis and J.M. Haviland (Eds.), *Handbook of Emotions* (pp. 575-594). New York: The Guildford Press.

Rubinow, D.R. and Post, R.M. (1992). Impaired recognition of affect in facial expression in depressed patients. *Biological Psychiatry*, 31, 947-953.

Russell, J.A. (1994). Is there universal recognition of emotion from facial expression? A review of cross-cultural studies. *Psychological Bulletin*, 115, 102-141.

Schneider, F., Gur, R.C., Gur, R.E. and Shtasel, D.L. (1995). Emotional processing in schizophrenia: Neurobehavioural probes in relation topsychopathology. *Schizophrenia Research*, *34*, 133-142.

Spiga, R.. Cherek, D.R., Roache, J.D. and Cowan, K.A. (1990). The effects of codeine on human aggressive responding. *International Clinical Psychopharmacology*, 5, 195-204.

Sprengelmeyer, R., Rausch, M., Eysel, U.T. and Przuntek, H. (1998). Neural structures associated with the recognition of facial basic emotions. *Procedures of the Royal Society of London B.*, 265, 1927-1931.

Sprengelmeyer, R., Young, A.W., Pundt, I., Sprengelmeyer, A., Calder, A.J., Berrios, G., Winkel, R., Vollmoeller, W., Kuhn, W., Sartory, G. and Przuntek, H. (1997). Disgust implicated in obsessive-compulsive disorder. *Procedures of the Royal Society of London B.*, 264, 1767-1773.

Strang, J. and Sheridan, J. (2003). Effect of national guidelines on prescription of methadone: analysis of NHS prescription data, England 1990-2001. *British Medical Journal*, *9*, *327*, 321–322.

Surguladze, S.A., Young, A.W., Senior, C., Brebion, G., Travis, M.J. and Phillips, M.L. (2004). Recognition Accuracy and Response Bias to Happy and Sad Facial Expressions in Patients With Major Depression. *Neuropsychology*, 18, 212-218.

Townshend, J.M. and Duka, T. (2003). Mixed emotions: alcoholics' impairments in the recognition of specific emotional facial expressions. *Neuropsychologia*, 41, 773-782.

van Erp, A. M. M. and Miczek, K. A. (2000). Aggressive behaviour, increased accumbal dopamine and decreased cortical serotonin in rats. *Journal of Neuroscience*, 20, 9320-9325.

Veit, R., Flor, H., Erb, M., Hermann, C., Lotze, M.Grodd, W. and Birbaumer, N. (2002). Brain circuits involved in emotional learning in antisocial behaviour and social phobia in humans. *Neuroscience Letters*, 38, 233-236.

Verbanck, P. (2001). Deficits in recognition of emotional facial expressions are still present in alcoholics after mid-long term abstinence. *Journal of Studies on Alcohol*, 62, 533-542.

Wild, B., Erb, M. and Bartels, M. (2001). Are emotions contagious? Evoked emotions while viewing emotionally expressive faces: quality, quantity, time course and gender differences. *Psychiatry Research*, 102, 109-124.

Young, A., Perrett, D., Calder, A., Sprengelmeyer, R. & Ekman, P. (2002). Facial expressions of emotion: Stimuli and Tests (FEEST). UK, Thames Valley Test Company.

Zacny, J.P. (1995). A review of the effects of opioids on psychomotor and cognitive functioning in humans. *Experimental and Clinical Psychopharmacology*, 3, 432-466.

Zangara, A., Blair, R.J. and Curran, H.V. (2002). A comparison of the effects of a beta-adrenergic blocker and a benzodiazepine upon the recognition of human facial expressions. *Psychopharmacology Berl.*, 163, 36-41.

Part 2: Enhanced recognition of facial expressions of disgust in opiate users

Abstract

Impaired recognition of facial expressions (FER) has been found in many psychiatric and drug-using populations. It has been proposed that this deficit may underlie the poor interpersonal functioning seen in such populations. This study set out to investigate FER in opiate users, a client group in which interpersonal difficulties are commonly seen. 20 methadone maintained clients (MM), 20 abstinent ex-opiate users (R) and 21 healthy controls (C) were compared on a test of FER looking at accuracy and reaction time to recognise pictures of the six basic emotional facial expressions morphed together to varying degrees. Self-report measures of aggression, impulsivity, mood and socialisation were also used to compare the different group profiles. The main findings were that MM were not significantly impaired in recognising any emotion, instead showing significantly enhanced recognition of disgust over R (and non-significantly over C). The literature around disgust recognition is considered, and characteristics of the opiate-using population that may be relevant are described. One speculation is that opiate users are hypersensitive to others' expressions of disgust due to the negative reactions they encounter from society. Further research in this area is indicated, and clinical implications discussed.

Introduction

Facial expressions of emotion are innate, automatic and universal displays (Ekman & Yamey, 2004), which can be attenuated or enhanced depending on the rules or norms of the culture in which they are produced (e.g. Ekman, 1999). Of the wide range of emotions that people experience, a set of six basic emotions has been identified: happiness, sadness, fear, anger, disgust and surprise, from which more complex emotions are thought to be derived (Oatley & Johnson-Laird, 1987). Blair (2003) argues that facial expressions act as a nonverbal "short-hand" for communicating important information to peers, in addition to reinforcing behaviour, thus regulating appropriate social interaction. It is likely that facial expressions provide a means of communication that helps to regulate social structure and hierarchy within groups (Argyle, 1988; Keltner & Haidt, 1999).

Blair (2003) suggests that the facial expression of fear acts as an unconditioned stimulus that communicates the aversive nature of an object or situation to others so they can avoid it (Mineka & Cook, 1993). Expressions of sadness are also thought to act as aversive unconditioned stimuli, discouraging the behaviour that elicited the sadness and motivating reparation (Blair, 1995) and soothing (Keltner & Haidt, 1999). The expression of happiness is thought to act as an appetitive unconditioned stimulus which increases the probability that the behaviour will be repeated in the future (Matthews & Wells, 1999). The facial expression of disgust appears to be most often used in relation to food, to quickly convey its aversive nature to others in order to deal with the risk of contamination and disease (Rozin, Haidt & McCauley, 1993). It has also been proposed that displays of disgust may be important in

negative socialisation, for example parental displays of disgust in toilet training (Rozin et al., 1993). Interestingly, there is no literature discussing the function of facial expressions of surprise. Blair (2003) suggests that the facial expression of anger has a different function from the other expressions as it does not act as an unconditioned stimulus. Angry expressions appear to curtail the ongoing behaviour of others in situations where social rules have been violated (Averill, 1982), thus maintaining socially appropriate behaviour. It has been suggested that expressions of anger are particularly powerful in situations involving hierarchy (Blair & Cipolotti, 2000).

There is a growing body of literature which suggests that the recognition of facial expressions is a complex process involving a number of anatomically dissociable neural systems (Adolphs, 2002), in particular the amygdala, the prefrontal cortex, the somatosensory related cortices and basal ganglia. Patients with amygdaloid lesions have consistently been seen to have impaired facial expression recognition (FER), particularly for the expression of fear (Adolphs, Tranel, Damasio & Damasio, 1995). The pre-frontal cortex seems to be particularly important in the recognition of angry expressions (Blair, Morris, Frith, Perrett & Dolan, 1999). Activation of this area on presentation of angry faces corresponds with Blair's hypothesis regarding this expression as a trigger for response reversal, as the orbitofrontal cortex is also implicated in this process (Dias, Robbins & Roberts, 1996). Research consistently concludes that the insula (a visceral somatosensory cortex) is key to the recognition of disgust, based on lesion and neuro-imaging studies (Blair, 2003; Cubero, Thiele & Bernstein, 1999; Sprengelmeyer, Rausch, Eysel & Przuntek, 1998). Adolphs' (2002) review also concludes that the recognition of disgust relies on the insula, with the

support of the basal ganglia (particularly the caudate nucleus) and other somatosensory related cortices in the right hemisphere.

Research evidence from pharmacological studies suggests that there is also differential neurotransmitter involvement in the recognition of different facial expressions (Blair & Curran, 1999). To summarise, it seems that serotonin (Harmer, Bhagwagar, Cowen & Goodwin, 2001a; Harmer, Bhagwagar, Perrett, Vollm, Cowen & Goodwin, 2003a), noradrenaline (Harmer, Hill, Taylor, Cowen and Goodwin, 2003c; Harmer, Perrett, Cowen & Goodwin, 2001b), GABA (Blair & Curran, 1999; Zangara, Blair & Curran, 2002) and possibly dopamine (Lawrence, Calder, McGowan & Grasby, 2002) are involved in FER.

Impaired FER has been found in those affected by psychiatric illness. It has been hypothesised that this may underlie the poor interpersonal functioning that characterises such populations (Poole, Tobias & Vinogradov, 2000). For example, deficits have been noted in schizophrenia (Hall, Harris, Sprengelmeyer, Sprengelmeyer, Young, Sanots, et al., 2004; Kohler, Turner, Bilker, Brensinger, Siegel, Kanes et al., 2003; Mandal, Pandey & Prasad, 1998), the manic phase of bipolar disorder (Lembke & Ketter, 2002), depression (Gur, Erwin, Gur, Zwil, Heimberg & Kraemer, 1992; Hale, 1998; Rubinow & Post, 1992), Obsessive Compulsive Disorder (OCD) (Sprengelmeyer, Young, Pundt, Sprengelmeyer, Calder, Berrios, et al., 1997), psychopathy (Blair, Colledge, Murray & Mitchell, 2001; Kosson, Suchy, Mayer & Libby, 2002) and acquired sociopathy (Blair & Cipolotti 2000; Hornak, Rolls & Wade, 1996).

FER has also been investigated in several drug and alcohol using populations. Chronic alcohol use has been associated with both poor FER (Kornreich, Blairy, Philippot, Dan, Foisy, Le Bon, et al., 2001a) and interpersonal difficulties (Kornreich, Philippot, Foisy, Blairy, Raynaud, Dan, et al., 2002). Philippot, Kornreich, Blairy, Baert, Den Dulk, Le Bon, et al. (1999) found impaired FER in recently detoxified alcoholics, with significant deficits in recognising anger, sadness, happiness and disgust and a special bias towards over-attribution of anger and contempt. Research with ecstasy users is still in its infancy, however Hoshi, Bisla and Curran (2004) found that ecstasy impacted on FER, enhancing recognition of fearful expressions following acute administration and impairing this ability four days later. Hoshi et al. also used a measure of aggression at these two time points, with the finding that users had higher self rated aggression scores than controls at day 4, despite there being no difference in this rating on the night of drug use. Benzodiazepines have been seen to selectively impair the recognition of anger (Blair & Curran, 1999) and fear (Zangara, Blair & Curran, 2002). While benzodiazepines are used for their anxiolytic properties, they have been noted to have 'paradoxical' side-effects such as disinhibition, hostility and aggression (Bond, Curran, Bruce, O'Sullivan & Shine, 1995; Zangara et al., 2002).

One major class of drugs which is underrepresented in the literature on emotion processing is that of opiates. Morphine, codeine, heroin and methadone are all forms of opiate that are widely used for pain relief, as drugs of abuse or as treatment for drug abuse. The prevalence of opiate use is difficult to gauge as it is a largely hidden problem. However, a recent estimation suggested a prevalence of 3.7% among men aged 25-44 in London (Hickman, Higgins, Hope & Bellis, 2004). Every year,

community pharmacies across England dispense over 1.25 million NHS prescriptions for methadone, suggesting that about 50,000 opiate users are receiving methadone at any one time (Strang & Sheridan, 2003). There is now substantial evidence for the effectiveness of methadone maintenance treatment (National Treatment Agency, 2004) for opiate users. Methadone treatment is also associated with improvements in health and social functioning, lower levels of crime and a lower risk of premature mortality (National Treatment Agency, 2004). Despite such high levels of use, relatively little is known about the effects of this class of drug upon psychological functioning.

It is of note that heroin use almost never occurs in isolation, with cocaine, amphetamines and benzodiazepines also being widely used by drug misusers seeking treatment for heroin dependence (Gossop, 2005). The National Treatment Outcomes Research Study (Gossop, Marsden, Stewart, Lehmann, Edwards, Wilson & Segar, 1998) found that almost two thirds of drug users in treatment had been using three or more substances before admission to treatment, and more than a third were using stimulants on a frequent basis. The most commonly used stimulant among drug misusers seeking treatment for heroin dependence was crack cocaine (Gossop, 2005).

Most opiate users seeking treatment present with a range of difficulties including severe family and social problems (National Treatment Agency, 2004). Personality disorder is commonly found in heroin users, with prevalence figures of around 50%, in which borderline, antisocial and dependent types are over-represented (Fassino, Daga, Delsedime, Rogna & Goggio, 2004). A dimensional approach has also been

taken to identify specific personality traits of heroin users, with Khantzian (1997) suggesting that heroin users are "immature and unable to take care of themselves". Other studies propose that users have poor self-esteem (Fieldman, Woolfolk & Allen, 1995), unstable personal relationships and identities (Fassino, Scarso, Barbero, Taylor, Pezzini & Furlan, 1992), as well as poorly regulated emotions and impulse control (Bartholomew, Sher & Wood, 2000; Conway, Kane, Ball, Poling & Rounsaville, 2003).

The literature thus suggests that not only is FER impaired in many populations that are characterised by poor social behaviour, but also that the specific nature of the FER impairment may be connected to the type of social functioning deficit. Given the interpersonal difficulties encountered by opiate users, in addition to the pharmacological changes that are brought about by opiate use, it is possible that accuracy and/or speed of FER is affected in this population.

Opiate receptors are widely distributed throughout the brain, being particularly dense in the amygdala (involved in emotional processing), nucleus accumbens (important in opiate reward) as well as areas concerned with pain perception.

The only published study in this area is by Kornreich, Foisy, Philippot, Tecco, Noel, Hess, et al. (2003), who compared FER in five groups of participants: 1) methadone maintained opiate users, 2) recently detoxified alcoholics, 3) recently detoxified alcohol and opiate users 4) recently detoxified opiate users, 5) normal controls. They found that the recently detoxified alcoholic group and mixed detoxified alcohol and opiate group scored significantly lower on accuracy of FER than normal controls.

The methadone maintained group and recently detoxified opiate group accuracy scores were comparable and better than those of the mixed detoxified alcohol and opiate group and detoxified alcohol only group, but worse than the normal controls. Unfortunately this study did not differentiate between the individual emotional expressions. The authors also found that the length of abstinence in the opiate abstinent group did not influence FER scores. This could be taken to mean that opiates do not have a detrimental effect on FER and that pre-existing deficits account for the lower scores than normal controls, or that the impairments caused by opiate use persist long after discontinuation, as was found with alcoholic subjects (Kornreich, Blairy, Philippot, Hess, Noel, Streel, et al., 2001b).

No study has yet looked at the effect of opiates on the recognition of individual facial expressions. This study investigated accuracy and speed of FER in current opiate users, abstinent ex-users and healthy controls. Male samples were used to minimise variation due to gender differences in FER ability (Hall, 1984; Hoffman, 1977). Demographic variables such as age, IQ, years of education, alcohol use and years of exposure to different drugs were recorded in order to measure comparability of groups. If the groups differed significantly on these variables, they were used as covariates in the analyses. Self report measures of impulsivity, aggression and socialisation were also used in order to compare the profiles of the groups. These particular constructs were selected on the basis of the literature regarding defining personality characteristics of opiate users. A mood measure was used in order to investigate any correlation with recognition of particular expressions as suggested in the literature. On the basis of Kornreich et al.'s (2003) findings, it was hypothesised that both the methadone maintained and detoxified opiate using groups would show

impaired FER relative to normal controls. Given the lack of existing data regarding individual facial expressions, this aspect of the study was exploratory. It was hoped that group numbers would be sufficient for some exploratory sub-group analysis, comparing crack and non-crack using participants, as well as perpetrators and non-perpetrators of violent crime, on both demographic measures and the faces task.

A power calculation was carried out using group differences obtained on the same version of the faces task in Blair and Curran's (1999) study investigating the effects of diazepam on recognition of angry expressions. With an alpha value of 0.05 and power of 0.8, the suggested sample size was 20 participants per group.

Materials and Method

Design and Participants

An independent groups design was used to compare current methadone users (MM) with ex-opiate users in rehabilitation (R) and healthy controls (C). MMs were recruited from a substance misuse clinic where they were receiving daily methadone maintenance treatment. Participants were identified as potentially suitable by their key worker. Rs were recruited from three drug rehabilitation programmes, two of which were residential and one a day programme. All R participants had been abstinent from opiates for at least six weeks and they had an mean time since last using opiates of 23.7 (±19.8) weeks. The healthy controls were recruited from a local Job Centre. Control participants were screened for both current and past problematic substance use, using the Cut-down Annoyed Guilty Eye-opener Scale: Adapted to Include Drugs (CAGE-AID; Brown & Rounds, 1995). Individuals with current

diagnosed mental health problems such as depression, schizophrenia and significant anxiety were excluded from the study in all three groups. The study was approved by the institutional ethics committee and all participants gave written informed consent.

Procedure

Potential MM and R participants were given an information sheet by their key worker and time to consider the study. If interested, they were then taken individually to the testing room where the study was explained more fully and they were given the opportunity to ask questions. If they were willing to participate they were asked for written consent. They then completed the assessments detailed below. At the end of testing they provided a urine sample which was tested for methadone and a range of illicit drugs. They received payment in the form of supermarket vouchers. Testing was conducted on site (i.e. at the substance misuse clinic for MMs, and at the different rehab centres for Rs).

Control subjects were recruited and tested in a quiet room on site at the Job Centre. They were approached while waiting to see Job Centre staff, given an information sheet, after which they were given time to consider the study and ask questions. If willing to participate, they were then asked for written consent, following which they completed the assessments detailed below. At the end of testing they were given payment in the form of supermarket vouchers. Urine samples were not collected from the control group.

Questionnaire Measures

Trait Measures and pre-morbid IQ

The Aggression Questionnaire (AQ; Buss & Perry, 1992) was used to index trait aggression, and the Barratt Impulsivity Scale (BIS; Barratt, 1994) was used as a measure of trait impulsivity. The Gough Socialisation Scale (Gough, 1960) was used as a measure of the extent to which societal values are internalised (high scores being indicative of socialised behaviour). This scale correlates with antisocial behaviour. The 'Spot the Word' test (Baddeley, Emslie & Nimmo-Smith, 1993) was used to index pre-morbid IQ as this measure correlates highly with the National Adult Reading Test (NART; Nelson & Willison, 1978).

Mood state and alcohol use

The Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1995) was used to assess participants' levels of anxiety, depression and stress over the past week. Alcohol use was assessed using the Fast Alcohol Screening Test (FAST; Hodgson, John, Abbasi, Hodgson, Waller, Thom & Newcombe, 2002). This measure gives an indication of hazardous alcohol use.

Drug use history

Frequency and quantity of illicit drug and alcohol use, both historically and in the 30 days prior to testing was recorded. Number of opiate overdoses and history of head injury was also noted.

Recognition of Facial Expressions

This task used the stimuli from the Facial Expressions of Emotions: Stimuli and Tests (FEEST; Young, Perrett, Calder, Sprengelmeyer & Ekman, 2002). Thirty stimuli were presented, featuring a male face portraying the six basic emotions – happiness, surprise, sadness, fear, disgust and anger – from the Ekman (1976) series. These basic emotions were used to create stimuli that are morphed from one emotion to another in 5 stages (10%, 30%, 50%, 70%, 90%). The expressions morphed are: anger to happiness, happiness to surprise, surprise to fear, fear to sadness, sadness to disgust and disgust back to anger. All stimuli involved the face JJ (Ekman & Friesen, 1976).

The task was performed on a laptop computer and consisted of 6 presentation blocks. In each block, the 30 morphed stimuli were presented in pseudo-random order (i.e. constraints were in place to ensure that no more than two stimuli with the same majority expression appeared consecutively). Each stimulus appeared on the screen for 500ms. The task was specially programmed with a response facility whereby the 6 emotions were arranged in an equilateral hexagon in the centre of the right side of the screen, next to the faces which were presented on the left hand side of the screen. Each emotion was equidistant from the central cursor base. Participants were asked to choose the emotion that corresponded to the facial expression by clicking on it with the mouse, as quickly and as accurately as they could. This response facility was designed to both lessen the working memory load required to remember six different response keys and to ensure that the response to each emotion required equal motor movement. Both response and reaction time were automatically recorded, and the recognition accuracy and reaction time scores that were used in the

analysis were calculated from the correct responses to the stimuli that had a dominant percentage (90% or 70%) of each emotion (see Hoshi et al., 2004 for details).

Other Information

At the end of testing, each participant was asked whether they had personal experience of being either a victim or perpetrator of violent crime.

Statistical Analysis

3 x 6 repeated measures ANOVAs were used to analyse the facial expression recognition task accuracy and response time scores, with group (methadone maintained (MM), ex-opiate users in rehabilitation (R) and control (C)) as a between subjects factor and expression (happiness, surprise, fear, sadness, disgust, anger) as a within subjects factor. Greenhouse-Geisser corrections were used where appropriate. Because of the group differences that emerged with years of education, this variable was used as a covariate in these analyses. One-way ANOVAs and t-tests were used to compare the groups' demographic and questionnaire data. Simple effects (Bonferroni corrected) post-hoc analysis was conducted on variables showing significant group differences. Bivariate correlations (Pearsons) were performed within the MM and R groups to analyse relationships between demographic information, questionnaire measures (total scores only) and the faces task. Independent sample t-tests were used in exploratory analyses of differences between the following subgroups: crack-using and non crack-using MM participants, perpetrators of violent crime and those who had not committed violent crime.

All data were analysed using SPSS for Windows version 11.5.

Results

Demographics

There were no significant group differences in age or Spot-the-Word scores (Table 1). The groups differed in years of education ($F_{2,58} = 9.36$, p<0.001) with controls (C) having longer than methadone maintained (MM) (p=0.007) and ex-opiate users (R) (p<0.001). The groups scored comparably on the FAST suggesting no group differences in problematic alcohol use over the past year. MM had first used heroin at a significantly younger age than R (t_{38} = -2.406, p=0.021) and had subsequently used for significantly more years (t_{38} =2.172, p=0.036). Both R and MM had comparable self-reported years of exposure to crack cocaine and alcohol.

The methadone maintained (MM) group consisted of 20 male participants, 19 of whom were taking daily methadone, and 1 Subutex as part of substitute prescribing treatment. The vast majority of the group had a long and chaotic history of polysubstance abuse, including alcohol, benzodiazepines, crack, cocaine, cannabis and amphetamines. However, at the time of testing none was using crack or benzodiazepines more than twice per week, and none had significantly problematic alcohol use, as defined by their keyworker. 85% (n=17) classified their ethnicity as white British, while 15% (n=3) were made up of Afro-Caribbean and Other.

The ex-opiate user (R) group consisted of twenty male participants who were attending drug rehabilitation programmes; 4 attended a day programme only, while the remaining 16 were in residential treatment. All participants had been abstinent

from opiates for at least 6 weeks, with an average time since last use of 23.7 (19.8) weeks. Similar to the MM group, the vast majority of this group also had a long and chaotic history of polysubstance abuse, but all identified opiates as their primary problematic substance. 80% (n=16) classified their ethnicity as White British and the remaining 20% (n=4) were Afro-Caribbean, Black British and American.

The control group consisted of 21 unemployed males, none of whom had used opiates in the past 30 days, nor had a significant history of opiate use. Two participants had tried heroin once, and one had used heroin twice over 2 months. In the 30 days prior to testing, 2 participants had used cocaine, 9 used cannabis and 1 used ecstasy. This was deemed to be recreational use that did not meet the threshold for problematic substance use, as assessed by the CAGE-AID (Brown & Rounds, 1995). Twenty participants had used alcohol in the 30 days prior to testing, and 1 defined his alcohol use as problematic for 2 years, 2 years prior to testing. 57% (n=12) of this group classified themselves as White British, with 19% (n=4) Irish and the remaining 24% coming from Afro-Caribbean, Black British, British Asian and Other ethnic backgrounds.

Current substance use in clinical groups

MM group

In the 30 days prior to testing, 13 participants reported having used cannabis, 11 used heroin, 11 used alcohol, 8 used crack, 5 used benzodiazepines, 2 used cocaine, 1 used amphetamines and 18 smoked tobacco. This additional drug use varied in frequency from once to every day during the 30-day period. Urine screening was conducted with 18/20 participants. Methadone was detected in the urine of 17/18 participants

Table 1: Group mean scores (and standard deviations) for demographic information (different subscripts indicate significantly different means).

	Group		
	Methadone Maintained (MM)	Rehab (R)	Control (C)
N	20	20	21
Age	38.40 (4.57) _a	37.55 (9.11) _a	35.81 (6.82) _a
Years of education	11.7 (2.08) _a	10.85 (2.43) _a	14.62 (3.91) _b
% unemployed	85% a	100% a	100% a
Spot the word score	49.10 (4.88) _a	48.00 (5.33) _a	50.24 (4.16) _a
Methadone dose mgs (prior to detox for R group)	64.95 (26.05) _a	60.71 (23.03) _a	93.4
Number of opiate overdoses	1.0 (1.37) _a	1.3 (2.13) _a	
Age heroin first used	19.70 (5.98) a	25.61 (9.25) _b	
Years of exposure to heroin	14.08 (6.58) a	10.50 (7.42) _b	1.00
Years of exposure to crack cocaine	5.9 (7.32) _a	5.75 (5.00) _a	
Years of exposure to alcohol	19.5 (9.08) _a	19.75 (11.83) _a	17.57 (6.71) _a
FAST score	3.05 (4.32) _a	4.10 (5.70) _a	2.14 (1.82) _a
N meeting threshold for hazardous alcohol use in past year (FAST>/=3)	7.	9 _a	8 _a
DASS total score	20.9 (15.66) _a	23.30 (11.54) a	8.05 (7.86) _b
Anxiety score	7.55 (5.68) _a	7.80 (5.13) _a	2.62 (2.58) _b
Depression score	5.35 (5.37) _a	5.75 (4.18) _a	1.00 (1.82) _b
Stress score	8.00 (5.26) _a	9.75 (3.86) a	4.48 (4.33) _b
BIS total score	75.7 (13.46) a	77.35 (9.70) a	66.95 (10.39) _b
Non-planning score	32.5 (5.91) _a	32.65 (3.94) a	28.33 (4.74) _b
Motor score	23.2 (5.44) _a	23.65 (5.0) _a	20.71 (3.44) _a
Attentional score	20 (4.92) _a	21.05 (3.33) a	16.48 (3.84) _b
Gough score	24.7 (7.4) a	22.95 (6.71) a	31.9 (5.9) _b
AQ total score	79.6 (24.12) _{a,b}	91.7 (20.48) a	69.81 (15.81) _b
Physical aggression score	24.75 (8.94) _{a,b}	29.45 (8.53) _a	19.86 (4.86) _b
Verbal aggression score	15.9 (3.28) _a	16.15 (4.3) _a	15.38 (4.26) _a
Anger score	18.65 (6.43) _{a,b}	22.05 (5.39) _a	15.81 (4.8) _b
Hostility score	20.6 (7.73) _{a,b}	24.05 (7.61) _a	18.33 (6.4) _b
Guilt score	26.15 (6.95) a	31.55 (6) _b	23.1 (7.48) a

(the 18th participant was taking Subutex), non-methadone opiates in 5/18 participants, benzodiazepines in 4/18, cocaine (including crack) in 5/18, and amphetamines in no participants. Self-reported drug use corresponded to that indicated by urine screening.

R Group

All participants reported abstinence from other drugs and alcohol for at least 30 days, with the exception of 1 participant who had used cannabis once, and 2 participants who had used alcohol once. Urine screening was conducted with 17/20 participants, detecting cannabis in one participant. The remainder of the screens were clear for all drugs tested.

Questionnaire Measures (Table 1)

DASS:

Group differences ($F_{2,58}$ =9.55, p<0.001) reflected lower scores of controls than MM (p=0.004) and R (p<0.001) on DASS total score. This was also the case for each of the subscales: anxiety ($F_{2,58}$ =8.21, p=0.001), depression ($F_{2,58}$ =8.79, p<0.001) and stress ($F_{2,58}$ =7.27, p=0.002). Controls had lower scores than both MM (depression: p=0.003; anxiety: p=0.004; stress: p=0.046) and R (depression: p=0.001; anxiety: p=0.002; stress: p=0.001). There were no differences between the MM and R group on any DASS score.

Barratt Impulsivity Scale:

Group differences ($F_{2,58}$ =5.06, p=0.009) reflected lower scores of Controls than MM (p=0.048) and R (p=0.014) on the BIS total score. This was also the case for the following subscales: non-planning impulsivity subscale ($F_{2,58}$ =5.11, p=0.009),

attentional impulsivity subscale score ($F_{2,58}$ =7.11, p=0.002). There were no group differences on the motor impulsivity subscale score. The MM and rehab group scored comparably on all aspects of the Barratt Impulsivity Scale.

Gough Socialisation Scale:

Group differences ($F_{2,58}$ =10.41, p<0.001) reflected higher scores of controls than MM (p=0.003) and R (p<0.001). MM and R scored comparably on the Gough scale. Aggression Questionnaire:

Group differences ($F_{2,58}$ =5.94, p=0.005) reflected higher scores of R than Controls (p=0.003) on AQ total score. There were no significant differences between MM and R group, or the MM and Controls on total AQ score. Group differences on subscales measuring physical aggression ($F_{2,58}$ =8.11, p=0.001), anger ($F_{2,58}$ =6.45, p=0.003) and hostility ($F_{2,58}$ =3.22, p=0.47) reflected higher scores of R than controls (p<0.001, p=0.002 and p=0.043 respectively). All groups scored comparably on the verbal aggression subscale. Group differences on the guilt subscale ($F_{2,58}$ =7.97, p=0.001) reflected higher scores of R than both MM (p=0.047) and controls (p=0.001).

Facial expression task

Accuracy scores

Repeated measures ANOVA showed a significant expression x group interaction $(F_{10,108}=2.04, p=0.036)$, and a main effect of expression $(F_{5,54}=79.53, p<0.001)$. Years of education was not a significant covariate. Given this interaction, one-way ANOVAs were conducted on each separate emotion and group, using Bonferroni corrected post-hoc analysis.

There was a significant group difference in identifying disgust ($F_{2,58}$ =3.72, p=0.03), reflecting higher accuracy scores of MM than R (p=0.033) (see Figure 1 (Table 2)). Covariance of years of education did not affect this finding. Additionally, ANOVA suggested a trend regarding group differences in the identification of sad expressions ($F_{2,58}$ =3.083, p=0.053). However, this was no longer significant after covarying years of education (p=0.23).

Table 2: Mean scores (and standard deviations) on facial expression task (different subscripts indicate significantly different means).

		Group		
		Methadone Maintained	Rehab	Control
Accuracy scores Happy Surprise Fear Sad Disgust Anger	Нарру	22.95 (1.28) _a	22.70 (1.84) _a	23.10 (2.17) _a
	Surprise	21.00 (2.55) _a	19.55 (3.03) _a	19.52 (3.43) _a
	12.55 (4.3) _a	11.55 (6.83) _a	14.90 (4.29) _a	
	15.90 (2.47) _{a,b}	14.55 (4.67) a	17.52 (4.03) _b	
	17.60 (4.65) a	11.95 (7.63) _b	13.43 (7.63) _{a,b}	
	Anger	14.70 (5.7) _a	14.25 (5.5) _a	13.29 (6.37) _a
Fear Sad	Нарру	2571.33 (500.58) a	2030.59 (431.39) _b	1736.64 (384.59) _b
	Surprise	3131.84 (675.42) _a	2672.60 (498.53) c	2237.16 (508.55) _b
	Fear	3429.44 (844.96) a	2491.90 (1203.17) _b	2151.25 (509.13) b
	Sad	4006.70 (1076.61) a	3493.72(1053.65) a	2457.02 (722.03) _b
	Disgust	3468.98 (879.71) a	2846.12(1075.36) a,b	2587.70 (719.38) _b
	Anger	3221.82 (862.1) a	2842.00 (1015.51) _{a,b}	2356.89 (680.95) _b

Reaction Times

There was no group x expression interaction, but a significant main effect of both group ($F_{2, 58}$ =16.76, p<0.001) and expression ($F_{5,54}$ =30.51, p<0.001). Covarying years of education did not change the significance of group differences (p<0.001), however there was no longer a main effect of expression. Covariance showed a trend

towards an expression x group interaction (p=0.089). Subsequently, one-way ANOVAs were conducted on each separate emotion and between groups on overall reaction time for the task, using Bonferroni corrected post-hoc analysis.

Across all expressions, the control group showed the fastest reaction times followed by the rehab group, with the MM group having the slowest reaction times (Figure 2 (Table 1)). Overall group reaction times were significantly different: MM-R: p=0.008, MM-C: p<0.001, R-C: p=0.034. There were significant differences between the groups on all 6 expressions (happy, surprise, fear and sad p<0.001, disgust p=0.009 and anger p=0.008). Covariance of years of education did not affect this pattern of results. Post-hoc analysis showed that the MM group was significantly slower on all expressions than the Control group (p<0.001 for happy, surprise, fear and sadness; p=0.008 for disgust and p=0.006 for anger). The Rehab group was significantly slower than the Control group on expressions of surprise (p=0.05) and sadness (p=0.003). The Rehab group were significantly faster than the MM group in responding to expressions of happiness (p=0.001), surprise (p=0.039) and fear (p=0.005).

Correlations

MM group

The only correlation with the faces task was found between BIS total score and accuracy of fear recognition (r=-0.529, p=0.016). Age at which heroin was first used correlated negatively with total DASS score (r=-0.511, p=0.021), total BIS score (r=-0.548, p=0.012), total AQ score (r=-0.491, p=0.028), and positively with

Figure 1: Mean accuracy score of each group on the facial expression recognition task

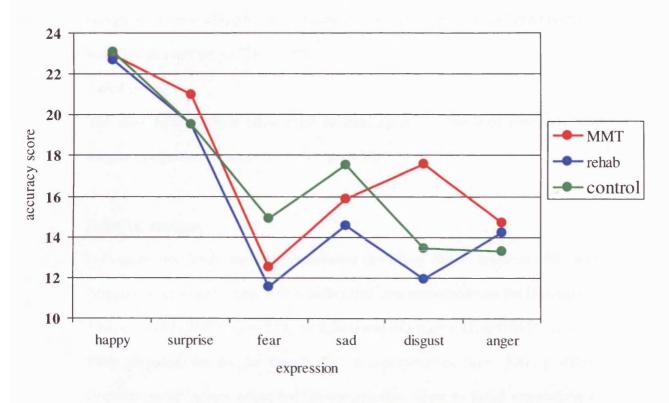
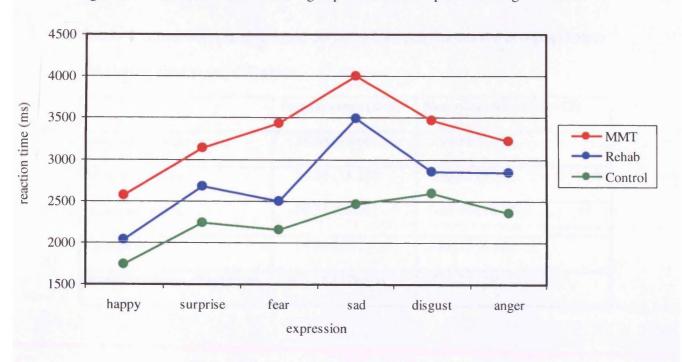


Figure 2: Mean reaction times of each group on the facial expression recognition task



Gough score (r= 0.673, p= 0.001). Years of exposure to heroin correlated negatively with Gough score (r= -0.529, p=0.017).

Rehab group

The dose of methadone taken prior to entering rehab correlated positively with disgust recognition accuracy (r= 0.612, p= 0.02).

Subgroup analysis

i) Perpetrators (n=8) and non-perpetrators (n=12) of violent crime in MM group Perpetrators of violent crime scored higher than non-perpetrators on the DASS ($t_{8.53}$ = 2.64, p= 0.028), BIS ($t_{10.52}$ = 2.23, p= 0.049) and AQ (t_{18} = 4.42, p<0.001) (Table 3). They scored lower on the Gough than non-perpetrators (t_{18} = -5.41, p<0.001). Perpetrators of violent crime had slower reaction times to facial expressions of happiness than non-perpetrators (t_{18} = 3.25, p= 0.004).

Table 3: Mean scores (and standard deviations) for perpetrators and non-perpetrators of violent crime in the MM group.

	Perpetrators (n=8)	Non-perpetrators (n=12)
DASS total score	31.88 (18.66)	13.58 (7.5)
BIS total score	83.75 (15.23)	70.33 (9.33)
AQ total score	100.38 (20.35)	65.75 (14.78)
Gough score	17.75 (4.2)	29.33 (4.98)
Reaction time – Happiness	2934.55 (579.43)	2329.19 (243.45)

ii) Perpetrators (n=13) and non-perpetrators (n=7) of violent crime in R group Perpetrators of violent crime had lower spot-the-word scores than non-perpetrators (t_{18} = -2.9, p=0.009) (Table 4). Perpetrators of violent crime tended to score higher on the AQ (t_{18} = 1.99, p=0.062) than non-perpetrators. Perpetrators of violent crime had less accurate recognition of anger than non-perpetrators (t_{18} = -2.32, p= 0.032).

Table 4: Mean scores (and standard deviations) for perpetrators and non-perpetrators of violent crime in the R group.

Perpetrators (n=13)	Non-perpetrators (n=7)
45.85 (5.05)	52 (3.22)
97.92 (20.12)	80.14 (16.71)
12.62 (5.91)	17.29 (3.1)
	45.85 (5.05) 97.92 (20.12)

iii) Crack (n=14) and non-crack users (n=6) in MM group

Crack users had less accurate recognition of disgust (t_{18} = -2.72, p=0.014) and anger (t_{18} = -2.91, p=0.009) than non-crack users (Table 5). There was a trend towards crack-users reporting more impulsivity than non-crack users (t_{18} =1.996, p=0.061).

Table 5: Mean scores (and standard deviations) for crack and non-crack users in the MM group.

	Crack users (n=14)	Non-crack users (n=6)
Accuracy score - Disgust	16.00 (4.57)	21.33 (1.97)
Accuracy score - Anger	12.64 (5.33)	19.50 (3.21)
BIS total score	79.36 (14.57)	67.17 (3.43)

Discussion

Profile of Groups

The clinical groups (i.e. MM and R) experienced more symptoms of depression, anxiety and stress (DASS), reported more impulsivity (BIS) and a lower level of socialisation (Gough) than the Control group. R reported more aggression (AQ) than controls and MM, but only the R-C difference was significant. Correlations suggested that within MM, the earlier participants had started using opiates, the higher the levels of self-reported depression, anxiety, stress, impulsivity and aggression were, and the lower the levels of socialisation. It is unsurprising that the clinical groups reported more symptoms of depression, anxiety and stress than controls, given that drug use often acts as an avoidant coping strategy for difficult feelings (LeBon, 2004). Similarly, the literature supports the finding that opiate users are more impulsive (Franques, Auriacombe & Tignol, 2000), antisocial (Fassino et al., 2004) and aggressive (Gerra, Amir, Raggi, Giusti, Delsignore, Bertacca & Brambilla, 2001) in personality than controls. It should be noted that with no additional objective measures of aggression, impulsivity or socialisation to corroborate the self report of participants, these profiles should be treated with caution, as self report is based on individual judgement and therefore open to bias.

Main research question and findings

On the basis of Kornreich et al.'s (2003) findings, it was hypothesised that both MM and R would show impaired FER on the faces task relative to controls. Investigation of individual facial expressions was exploratory due to the lack of existing literature.

Firstly, in accord with other studies of FER (e.g. Leppanen & Hietanen, 2003; Rapcsak, Galper, Comer, Reminger, Nielsen, Kaszniak, et al., 2000), this study showed happiness to be the easiest emotion to recognise, with accuracy scores near ceiling, the fastest reaction times and no group differences. The more difficult emotions to recognise were fear, disgust, sadness and anger.

The results of the faces task showed a clear group hierarchy in reaction times; the reaction times of MM were the slowest across all expressions, followed by R, with the controls responding fastest. This reached significance on some but not all expressions. This could reflect the effects of opiates on processing speed (Verdejo, Toribio, Orozco, Puente & Perez-Garcia, 2005), psychomotor speed (Hiltunen, Lafolie, Martel, Ottosson, Boreus, Beck, et al., 1995; Mintzer & Stitzer, 2002) and/or decision making (Mintzer & Stitzer, 2002), which research indicates may start to remit with abstinence (Davis, Liddiard & McMillan, 2002; Verdejo et al., 2005).

However, in terms of accuracy there was no such clear hierarchy. MM did not display any significant deficits in recognition accuracy. The most notable finding was the group difference in recognition of disgust, with MM showing enhanced recognition of disgust over R (and non-significantly over controls). Moreover, the absence of a difference between the reaction times of the two groups to expressions of disgust suggests that a speed/accuracy trade-off does not account for this improved accuracy. Within R, dose of methadone prior to detoxing was correlated with accuracy of disgust recognition. Within MM, participants' poorer recognition of fear was associated with increased impulsivity.

The current findings do not support those of Kornreich et al. (2003). Kornreich et al. found globally impaired FER in both methadone maintenance treatment (MMT) and detoxified opiate addicts (DOA) compared with healthy controls. The opiate using samples in Kornreich et al.'s study had a similar history of polydrug use to the samples in the current study but a shorter history of using opiates (MMT=9.32 years vs MM=14.08 years, DOA=7.8 years vs R=10.50) and lower average age (MMT=31.53 years vs MM=38.4 years, DOA=28.93 years vs R=37.55 years). Kornreich et al. also excluded participants on the basis of current or past alcohol dependence. While current problematic alcohol use was an exclusion criteria for MM in the current study, past alcohol dependence was not screened out, in either MM or R. These differences in the samples are unlikely to account for the discrepant findings as greater exposure to opiates and alcohol would seem more likely to increase FER impairments.

So how do we explain the finding that MM show hypersensitivity to expressions of disgust? Psychiatric and drug-using populations in which *impaired* disgust recognition has been found include OCD patients (Sprengelmeyer et al., 1997), psychopaths (Kosson et al., 2002) and detoxified alcoholics (Townshend & Duka, 2003). Impaired disgust recognition has also been seen in schizophrenia (e.g. Kohler et al., 2003), although as only one of several impaired expressions, suggesting a more global deficit.

The case of OCD and disgust recognition is particularly interesting. It has been suggested that abnormal experience of disgust may be involved in the genesis of obsessions and compulsions (Power & Dalgleish, 1997), as so many OCD patients

exhibit heightened disgust in reaction to stimuli they regard as contaminated (Parker, McNally, Nakayama & Wilhelm, 2004). Additionally, evidence suggests that abnormalities exist in the basal ganglia of obsessive-compulsive disorder patients (Parker et al., 2004). However, the research evidence regarding FER is contradictory. One study has found selectively impaired recognition of disgust in OCD patients syndrome patients with obsessive-compulsive features (and Tourette's (Sprengelmever et al., 1997), while three others have failed to replicate this result (Buhlmann, McNally, Etcoff, Tuschen-Caffier & Wilhelm, 2004; Kornreich et al., 2001a; Parker et al., 2004). Parker et al. (2004) conclude that impairments in disgust recognition may only occur in cases of severe OCD. A further study by Montagne, Kessels, de Geus, Denys, de Haan & Westenberg (2005) found that OCD patients were more sensitive to all emotional expressions than healthy controls, but failed to find a disgust-specific effect in the group as a whole. Additionally, in contrast with Sprengelmeyer et al. and Parker et al., their results showed that those OCD patients with more severe symptomatology demonstrated more accurate disgust recognition than controls.

Specific impairment of disgust recognition has also been found in psychopaths (Kosson et al., 2002). It has been proposed that psychopaths have a limited capacity for understanding and experiencing emotion (Blair, Colledge, Murray & Mitchell, 2001), and unlike non-psychopaths, they also fail to exhibit startle potentiation while viewing slides depicting mutilation (Levenston, Patrick, Bradley & Lang, 2000). If, as has been suggested, disgust is important in negative socialisation, longstanding insensitivity to other's disgust could conceivably contribute to the poorly socialised behaviour seen in the psychopathic population.

Although detoxified alcoholics have also been found to show a disgust recognition deficit (e.g. Philippot, Komreich, Blairy, Baert, Den Dulk, Le Bon, et al., 1999; Townshend & Duka, 2003), this is often seen alongside deficits with other emotions, in particular anger. Such individuals have been seen to frequently misidentify disgust as anger (Townshend & Duka, 2003), suggesting an attributional bias which may be associated with the interpersonal difficulties seen in this population (Kornreich et al., 2002).

To date, only one other study has found *enhanced* disgust recognition in any population. In studying euthymic bipolar disorder patients, Harmer, Grayson and Goodwin (2002b) found generally impaired FER performance, but enhanced recognition of disgust and fewer false positive disgust expression identifications, compared with controls. The authors discuss the impact of mood disorder on the basal ganglia, in particular the caudate. Neuroimaging studies suggest that caudate volume may be increased in bipolar patients relative to matched controls (Aylward, Roberts-Twillie, Barta, Kumr, Harris, Geer, et al., 1995), in addition to increased levels of neural activity within the caudate during periods of acute mania (Blumberg, Stern, Martinez, Ricketts, de Asis, White, et al., 2000). They also consider whether more accurate recognition of disgust could be associated with the negative self-perception that has been found in bipolar patients (Lyon, Startup & Bentall, 1999), in particular low self-esteem and increased anxiety. However, they do not elucidate the mechanism by which this could happen.

Evidence from the populations reviewed begins to indicate that the story surrounding disgust is far from straightforward. It may prove helpful to think more about the function of disgust and how this could relate to opiate users. It has been proposed that the emotion of disgust is based on the appraisal of objects and events for their potential role in contamination and transmission of diseases (Rozin et al., 1993), often in relation to food. Opiate users are at particularly high risk of disease, firstly due to the lifestyle they lead (e.g. injecting heroin, sleeping rough) but secondly because opiates suppress the immune system (Vallejo, de Leon-Casasola & Benyamin, 2004), leaving users more open to infection and illness. This would make heightened awareness of others' disgust particularly valuable for protecting themselves against potentially contaminating stimuli.

Power and Dalgleish (1997) argue that our understanding of disgust in relation to contaminating foodstuffs and disease has been over-emphasized at the expense of the potentially more interesting application of disgust to the self and other people. Disgust seems to serve an important function in negative socialisation (Rozin et al., 1993), with Rozin and Fallon (1987) noting that disgust is one of the most powerful ways of transmitting cultural and moral values. The typical opiate-user encounters a great deal of stigma and negative attention from society (Bell, Dru, Fischer, Levit & Sarfraz, 2002; Viney, Westbrook & Preston, 1985), which often involves reactions of disgust (Payte, Khuri, Joseph & Woods, 1999; Thaca, 1997). It could be hypothesized that this repeated exposure results in hypersensitivity to other's disgust, particularly if a concept of the self as disgusting becomes internalised.

Johnson-Laird and Oatley (1989) propose that disgust is the key basic emotion underlying a number of more complex emotions such as shame. Moreover, Power and Dalgleish (1997) define shame as disgust directed at the self. Shame is defined as involving a global negative feeling about the self in response to some misdeed or shortcoming (Lewis, 1971), and can affect one's core sense of self (Lindsay-Hartz, de Rivera & Mascolo, 1995). Shame evolved from the need to behave submissively to threats from more powerful others (Gilbert, 2000) and acts to distance the self from others. One could speculate that society's evolutionary socialisation tactic of disgust towards drug users has resulted in them internalising this disgust and experiencing shame. The literature around shame in drug users suggests higher levels than in the normal population (Blatt, Rounsaville, Eyre & Wilber, 1984; O'Connor, Berry, Inaba, Weiss & Morrison, 1994, Viney et al., 1985), with Fossum and Mason (1986) proposing that "addiction and shame are inseparable". Testimonials written by heroin users also describe intense feelings of shame (e.g. "Kerry", 2004). Research in the same clinical setting as the current study also found high levels of characterological shame in polydrug users compared with non-drug using controls (Andersen, 2004). It has also been suggested that MMs have high levels of shame that predate the onset of substance use, and that while they use substances as a coping strategy for such feelings, their substance use is likely to result in additional feelings of shame (Dearing, Stuewig & Tangney, 2005). This association between enhanced disgust recognition and shame is speculative given that no shame measure was used in the current study. This is a potentially important area for future research to investigate.

That R participants showed a different pattern of responding to disgust compared with MM participants is curious, as one might expect this population to have a similar experience of other's disgust reactions to MMs. The discrepancy could suggest several things. Firstly, it is possible that the acute neuropharmacological effects of opiates (and other drugs used) account for the difference between the two groups. As the MMs were using a combination of different substances, it is not possible to accurately gauge which neurotransmitters or parts of the brain were affected, making this hypothesis untestable. Secondly, it is possible that the immediate environment of the participant impacts on their sensitivity to disgust. Stigma associated with attending methadone clinics and negative treatment by health care workers is well documented (Bell et al., 2002; Payte et al., 1999). Entering the drug treatment clinic in which MMs were tested may be sufficient to activate a negative self-concept with heightened feelings of disgust, directed at both the self and other service users, which could act to prime emotional processing. In contrast, the rehabilitation centres used for testing are likely to lack many of those cues and the participants may have access to a healthier self-concept. The process of rehabilitation is also likely to have an impact on self-concept, although it might be expected to take more time than had elapsed for many of the R participants.

Alternatively, there may be qualitative differences between MM and R participants which enabled the R participants to enter into the rehabilitation process. R scored comparably with MM on trait and state measures, suggesting that both groups were drawn from a similar population. However, MM and R differed in years of exposure to opiates and age of first use (MM starting to use earlier and for longer). This could indicate different reasons for using substances (e.g. R having fewer premorbid

difficulties) and/or less exposure to the lifestyle associated with heroin use and negative reactions of others. The correlation between methadone dose prior to entering rehabilitation and disgust recognition in R could suggest that those Rs requiring a higher dose are more like the MM population, given that dose is an index of addiction severity (Ghodse, Reynolds, Baldacchino, Dunmore, Byrne, Oyefeso, et al., 2002).

Alternatively, it is possible that the mixed psychopathology within the groups accounts for the group difference on disgust recognition, rather than the effect of opiate use. As previously described, this population is known to have a high incidence of personality disorders, other substance use besides opiates, low self-esteem and interpersonal difficulties.

Clearly, these are all speculative hypotheses given the scope of the current study and the paucity of existing literature. Replication will be important in clarifying whether the main finding is robust and what other factors (e.g. shame, emotional priming, physical setting) are relevant.

Within MM, participants' poorer recognition of fear was correlated with increased impulsivity, an association that has not been reported in previous studies. As fear is used to communicate danger to others, a deficit in recognising this, coupled with greater impulsivity may mean that individuals are less likely to engage in harm avoidant behaviour.

Subgroup analysis

i) Perpetrators of violent crime

Although exploratory given the small and unequal group sizes, the subgroup analysis yielded interesting results. It should be noted that the potential for type 1 errors is increased here due to the number of t-tests conducted. This means that the results should be interpreted cautiously.

MM participants who had perpetrated violent crime experienced more depression, anxiety and stress, reported more impulsivity and aggression and less socialisation than those who had not. They were also slower at responding to expressions of happiness. R participants who had perpetrated violent crime also reported more aggression and had lower premorbid IQ scores (Spot-the-Word). Additionally, they showed less accurate recognition of anger. It is unsurprising that increased aggression, impulsivity and poor socialisation are associated with perpetrating violent crime. The impaired recognition of anger in R perpetrators compared with non-perpetrators of violent crime has also been seen in other populations (for example, psychopaths, manic patients, alcohol and benzodiazepine users) that are characterised by less sensitive social behaviour which seems to take the form of aggressive or persistent approach behaviours. Intuitively, it also makes sense that those who transgress social boundaries may be less sensitive to anger, if the function of angry expressions is to curtail the ongoing behaviour of others in situations where social rules have been violated (Averill, 1982). It is unclear however, why this impairment would only be present in R participants, not MMs, though small sample size could account for this. It is also unclear why MM perpetrators would be slower in recognising happiness than non-perpetrators. One could speculate that such a deficit might reflect lower sensitivity to reinforcers of prosocial behaviour (i.e.

expressions of happiness), making the avoidance of activities such as violent crime less important.

ii) Crack users

While achieving better disgust recognition accuracy scores than R or controls, crackusing MMs were significantly poorer at recognising disgust than non-crack using MMs. This difference is striking. One could speculate that crack-users form a qualitatively different subgroup who have poorer emotional perception, or that crack is used in part for its dampening effect on the perceptions of others' negative emotions. This subgroup also showed significantly less accurate recognition of anger and a trend towards reporting more impulsivity than non-crack using MMs. It could be hypothesised that the same process underlies the crack-using MMs' reduced accuracy in anger recognition (as discussed in relation to perpetrators of violent crime), as the literature suggests that crack use can be associated with violent crime (Gossop, 2005; Haynes, 1998) where heroin use alone is not (Hammersley, Forsyth, Morrison, & Davies, 1989; Parker & Newcombe 1987). Unfortunately, due to small subgroup size it is not possible to analyse the relationship between crack use and violent crime. It is also noteworthy that crack cocaine use affects the neurotransmitter dopamine which is thought to play a role in appetitive aggression and social dominance (Lawrence, Calder, McGowan & Grasby, 2002).

Limitations, implications and future directions of the study

Although the samples used in the current study were not purely methadone maintained and ex-opiate using individuals, nor homogenous in terms of their drug and alcohol use, they do provide an ecologically valid look at the typical opiate-using

drug service user. Small sample sizes preclude firm conclusions being drawn regarding the subgroups.

It would be interesting, given the lesion and neuroimaging studies suggesting that recognition of disgust involves the basal ganglia (Adolphs, 2002), anterior insula (Calder, Keane, Manes, Antoun & Young, 2000; Phillips, Young, Senior, Brammer, Andrews, Calder, et al., 1997), and caudate (Gray, Young, Barker, Curtis & Gibson, 1997; Phillips et al., 1997), to compare methadone maintained and rehab participants in fMRI. The hypothesis would be that methadone maintained participants show enhanced activation in these areas, in response to disgust expressions.

In order to support the hypothesised meaning of enhanced disgust recognition, future research should aim to clarify both the relationship between recognition of disgust and feelings of shame, as well as the experience of shame in this population. The findings of the present study also point to the importance of investigating service users' experience of the physical setting in which they receive treatment, and how this might impact upon their journey through treatment.

Methadone maintained individuals' heightened perception of disgust has important implications for service providers. Staff need to be aware of their non-verbal reactions to clients, and, if supported by further research evidence, of the potential they and the environment in which they work have to evoke feelings of shame in their clients. Such feelings are likely to be extremely aversive and could be implicated in treatment dropout (Viney et al., 1985).

References

Adolphs, R. (2002). Neural systems for recognising emotion. *Current Opinion in Neurobiology*, 12, 169-177.

Adolphs, R., Tranel, D., Damasio, H. and Damasio, A.R. (1995). Fear and the human amygdala. *Journal of Neuroscience*, 15, 5879-5892.

Andersen, K.V., Curran, H.V. & Davis, P. (2004). Exploration of shame and Disclosure in long-term drug dependence. Abstract of paper presented at European Association for Behavioural & Cognitive Therapies, 34th Annual Congress, University of Manchester, September 2004.

Argyle, M. (1988). Bodily Communication. Second Edition. Routledge.

Averill, J.R. (1982). Anger and aggression: an essay on emotion. Springer, New York.

Aylward, E.H., Roberts-Twillie, J.V., Barta, P.E., Kumr, A.J., Harris, G.J. and Geer, M. (1994). Basal ganglia volumes and white matter hyperintensities in patients with bipolar disorder. *American Journal of Psychiatry*, 151, 687–693.

Baddeley, A., Emslie, H. and Nimmo-Smith, I. (1993). The Spot the Word test: a robust estimate of verbal intelligence based on lexical decision. *British Journal of Clinical Psychology*, 32, 55-65.

Barratt, E. S. (1994). Impulsiveness and aggression. In J. Monahan & H. J. Steadman (Eds). *Violence and mental disorder: Developments in risk assessment* (pp.61-79). Chicago: Chicago University Press. SV.

Bartholomew, B.D., Sher, K.J. and Wood, M.D. (2000). Personality and substance use disorders: a prospective study. *Journal of Clinical and Consulting Psychology*, 68, 818-829.

Bell, J., Dru, A., Fischer, B., Levit, S. and Sarfraz, M.A. (2002). Substitution therapy for heroin addiction. *Substance Use Misuse*, *37*, 1149-78.

Blair, R.J.R. (1995). A cognitive developmental approach to morality: investigating the psychopath. *Cognition*, 57, 1-29.

Blair, R.J.R (2003). Facial expressions, their communicatory functions and neuro-cognitive substrates. *Philosophical Transactions of the Royal Society of London B.*, 358, 561-572.

Blair, R.J.R. and Cipolotti, L. (2000). Impaired social response reversal: A case of 'acquired sociopathy'. *Brain*, 123, 1122-1141.

Blair, R.J.R., Colledge, E., Murray, L. and Mitchell, D.G. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology*, 29, 491-498.

Blair, R.J.R. and Curran, H.V. (1999). Selective impairment in the recognition of anger induced by diazepam. *Psychopharmacology*, 147, 335-338.

Blair, R.J.R., Morris, J.S., Frith, C.D., Perrett, D.I. and Dolan, R. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain*, 122, 883-893.

Blatt, S.J., Rounsaville, B., Eyre, S.L. and Wilber, C. (1984). The psychodynamics of opiate addiction. *Journal of Mental Disorders*, 172, 342-52

Blumberg, H.P., Stern, E., Martinez, D., Ricketts, S., de Asis, J. and White T. (2000). Increased anterior cingulate and caudate activity in bipolar mania. *Biological Psychiatry*, 48, 1045–1052.

Brown, R.L., and Rounds, L.A. (1995). Conjoint screening questionnaires for alcohol and drug abuse. *Wisconsin Medical Journal*, 94, 135-140,

Buhlmann, U., McNally, R.J., Etcoff, N.L., Tuschen-Caffier, B. and Wilhelm, S. (2004). Emotion recognition deficits in body dysmorphic disorder. *Journal of Psychiatry Research*, 38, 201-6.

Buss, A.H. and Perry, M. (1992). The Aggression Questionnaire. *Journal of Personality and Social Psychology*, 63, 452-459.

Calder, A.J., Keane, J., Manes, F., Antoun, N. and Young, A.W. (2000). Impaired recognition and experience of disgust following brain injury. *Nature Neuroscience*, 3, 1077–1078.

Conway, K.P., Kane, R.J., Ball, S.A., Poling, J.C. and Rounsaville, B.J. (2003). Personality, substance choice and polysubstance involvement among substance dependent patients. *Drug and Alcohol Dependence*, 71, 65-75.

Cubero, I., Thiele, T.E. and Bernstein, I.L. (1999). Insular cortex lesions and taste aversion learning: effects of conditioning method and timing of lesion. *Brain Research*, 839, 323-330.

Davis, P.E., Liddiard, H. and McMillan, T.M. (2002). Neuropsychological deficits and opiate abuse. *Drug and Alcohol Dependence*, 1; 67, 105-8.

Dearing, R.L., Stuewig, J. and Tangney, J.P. (2005). On the importance of distinguishing shame from guilt: Relations to problematic alcohol and drug use. *Addictive Behaviours, In Press*.

Dias, R., Robbins, T.W. and Roberts, A.C. (1996). Dissociation in prefrontal cortex of affective and attentional shifts. *Nature*, 380, 69-72.

Ekman, P. (1999). Basic Emotions. In T. Dalgleish, and M. Power (Eds.), *The Handbook of Cognition and Emotion* (pp. 45-60). Sussex, UK: John Wiley and Sons Ltd.

Ekman, P. and Yamey, G. (2004). Emotions revealed: recognising facial expressions. BMJ Career Focus, 328, 75-76.

Fassino, S., Abbate Daga, G., Delsedime, N., Rogna, L. and Goggio, S. (2004). Quality of life and personality disorders in heroin abusers. *Drug and Alcohol Dependence*, 76, 73-80.

Fassino, S., Scarso, G., Barbero, L., Taylor, J., Pezzini, F. and Furlan, P.M. (1992). The image of self and of the environment in drug abusers: a comparative study using the TAT. *Drug and Alcohol Dependence*, 30, 253-261.

Fieldman, N.P., Woolfolk, R.L. and Allen, L.A. (1995). Dimensions of self-concept: a comparison of heroin and cocaine addicts. *American Journal of Drug and Alcohol Abuse*, 21, 315-326.

Fossum, M.A. and Mason, M.J. (1986). Facing shame: Families in recovery. New York: W.W. Norton.

Franques, P., Auriacombe, M. and Tignol, J. (2000). Personality of substance abusers. Encephale, 26, 68-78.

Gerra, G., Amir, Z., Raggi, M.A., Giusti, F., Delsignore, R., Bertacca, S. and Brambilla, F. (2001). Aggressive responding of male heroin addicts under methadone treatment: psychometric and neuroendocrine correlates. *Drug and Alcohol Dependence*, 65, 85-95.

Ghodse, A. H., Reynolds, M., Baldacchino, A.M., Dunmore, E., Byrne, S., Oyefeso, A., Clancy, C & Crawford, V. (2002). Treating an opiate dependent inpatient population: A one year follow-up study of treatment completers and noncompleters. *Addictive Behaviours*, 27, 765-778.

Gilbert, P. (2000). Overcoming depression: A self-help guide using Cognitive Behavioural Techniques. Robinson.

Gossop, M. (2005). Treatment outcomes: what we know and what we need to know. National Treatment Agency for Substance Misuse.

www.nta.nhs.uk/publications/docs/te2.pdf (retrieved 10.06.05).

Gossop, M., Marsden, J., Stewart, D., Lehmann, P., Edwards, C., Wilson, A. and Segar, G. (1998). Substance misuse, health and social problems of clients at 54 drug treatment agencies: intake data from the National Treatment Outcome Research Study (NTORS). *British Journal of Psychiatry*, 173, 166-171.

Gough, H.G. (1960). Theory and measurement of socialisation. *Journal of Consulting Psychology*, 24, 23-30.

Gray, J.M., Young, A.W., Barker, W.A., Curtis, A. and Gibson, D. (1997). Impaired recognition of disgust in Huntington's disease gene carriers. *Brain*, 120, 2029–2038.

Gur, R.C., Erwin, R.J., Gur, R.E., Zwil, A.S., Heimberg, C. and Kraemer, H.C. (1992). Facial emotion discrimination: II. Behavioural findings in depression. *Psychiatry Research*, 42, 241-251.

Hale, W.W. (1998). Judgment of facial expressions and depression persistence. *Psychiatry Research*, 80, 265-274.

Hall, J. (1984). Nonverbal sex differences: Communication accuracy and expressive style. John Hopkins University Press, Baltimore, MD.

Hall, J., Harris, J.M., Sprengelmeyer, R., Sprengelmeyer, A., Young, A.W., Sanots, I.M., Johnstone, E.C. and Lawrie, S.M. (2004). Social cognition and face processing in schizophrenia. *The British Journal of Psychiatry*, *185*, 169-170.

Hammersley, R.H., Forsyth, A.J., Morrison, V. & Davies, J.B. (1989). The relationship between crime and opioid use. *British Journal of Addiction*, 84, 1029-1043.

Harmer, C.J., Bhagwagar, Z., Cowen, P.J. and Goodwin G.W. (2001a) Acute administration of citalopram in healthy volunteers facilitates recognition of happiness and fear. *Journal of Psychopharmacology*, 15 (suppl.), A16.

Harmer, C.J., Bhagwagar, Z., Perrett, D.I., Vollm, B.A., Cowen, P. and Goodwin, G. (2003a). Acute SSRI administration affects the processing of social cues in healthy volunteers. *Neuropsychopharmacology*, 28, 148-152.

Harmer, C.J., Grayson, L. and Goodwin, G.M. (2002b). Enhanced recognition of disgust in bipolar illness. *Biological Psychiatry*, 51, 298-304.

Harmer, C.J., Hill, S.A., Taylor, M.J., Cowen, P.J. and Goodwin, G.M. (2003c). Towards a neuropsychological theory of antidepressant drug action: Increase in positive emotional bias after potentiation of norepinephrine activity. *American Journal of Psychiatry*, 160, 990-992.

Harmer, C.J., Perrett, D.I, Cowen, P.J and Goodwin, G.M. (2001b). Administration of the beta-adrenoceptor blocker propranolol impairs the processing of facial expressions of sadness. *Psychopharmacology Berl.*, 154, 383-389.

Haynes P. (1998). Drug using offenders in south London. Trends and outcomes.

Journal of Substance Abuse and Treatment, 15, 449-56.

Hickman, M., V. Higgins, V. Hope and M. A. Bellis (2004). Estimating prevalence of problem drug use: multiple methods in Brighton, Liverpool and London. Home Office Online Report http://www.homeoffice.gov.uk/rds/pdfs04/rdsolr3604.pdf. (retrieved 10.06.05).

Hiltunen AJ, Lafolie P, Martel J, Ottosson EC, Boreus LO, Beck O, Borg S, Hjemdahl P. (1995). Subjective and objective symptoms in relation to plasma

methadone concentration in methadone patients. *Psychopharmacology (Berl)*, 118, 122-6.

Hodgson, R.J., John, B., Abbasi, T., Hodgeson, R.C., Waller, S., Thom, B. and Newcombe, R.G. (2003). Fast screening for alcohol misuse. *Addictive Behaviour*, 28, 1453-63.

Hoffman, M.L. (1977). Sex differences in empathy and related behaviours. Psychological Bulletin, 84, 712-722.

Hornak, J., Rolls, E.T. and Wade, D. (1996). Face and voice expression identification in patients with emotional and behavioural changes following ventral frontal damage. *Neuropsychologia*, 34, 247-261.

Hoshi, R., Bisla, J. and Curran, V. (2004). The acute and sub-acute effects of 'ecstasy' (MDMA) on processing of facial expressions: preliminary findings. *Drug and Alcohol Dependence*, 76, 297-304.

Oatley, K. and Johnson-Laird, P.N. (1987). Towards a cognitive theory of emotions. Cognition and Emotion, 1, 29-50.

Keltner, D. and Haidt, J. (1999). Social functions of emotions at four levels of analysis. *Cognition and Emotion*, 13, 505-521.

"Kerry" (2004). Heroin - A Distant Memory.

http://www.users.globalnet.co.uk/~edges/online/issue37/p6.htm (retrieved 01.06.05).

Khantzian, E.J. (1997). The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harvard Review of Psychiatry*, 4, 231-244.

Kohler, C.G., Turner, T.H., Bilker, W.B., Brensinger, C.M., Siegel, S.J., Kanes, S.J., Gur, R.E. and Gur, R.C. (2003). Facial emotion recognition in schizophrenia: intensity effects and error pattern. *American Journal of Psychiatry*, 160, 1768-1774.

Kornreich, C., Blairy, S., Philippot, P., Dan, B., Foisy, M-L., Le Bon, O., Pelc, I. and Verbanck, P. (2001a). Impaired emotional facial expression recognition in alcoholism compared with obsessive compulsive disorder and normal controls. *Psychiatry Research*, 102, 235-248

Kornreich, C., Blairy, S., Philippot, P., Hess, U., Noel, X., Streel, E., Le Bon, O., Dan, B., Pelc, I. and Verbanck, P. (2001b). Deficits in recognition of emotional facial expression are still present after mid-long term abstinence in alcoholics. *Journal of Studies on Alcohol*, 62, 533-542.

Kornreich, C., Foisy, M-L., Philippot, B.D, Tecco, J., Noel, X., Hess, U., Pelc, I. And Verbanck, P. (2003). Impaired emotional facial expression recognition in alcoholics, opiate dependence subjects, methadone maintained subjects and mixed

alcohol-opiate antecedent subjects compared with normal controls. *Psychiatry Research*, 119, 251-260.

Kornreich, C., Philippot, P., Foisy, M-L., Blairy, S., Raynaud, E., Dan, B., Hess, U., Noel, X., Pelc, I. and Verbanck, P. (2002). Impaired emotional facial expression recognition is associated with interpersonal problems in alcoholism. *Alcohol and Alcoholism* 37, 394-400.

Kosson, D.S., Suchy, Y., Mayer, A.R. and Libby, J. (2002). Facial affect recognition in criminal psychopaths. *Emotion*, 2, 398-411.

Lawrence, A.D., Calder, A.J., McGowan, S.W. and Grasby, P.M. (2002). Selective disruption of the recognition of facial expressions of anger. *Neuroreport*, 13, 881-884.

LeBon, O., Basiaux, P., Streel, E., Tecco, J., Hanak, C., Hansenne, M., Ansseau, M., Pelc, I., Verbanck, P. and Dupont, S. (2004). Personality profile and drug of choice; a multivariate analysis using Cloninger's TCI on heroin addicts, alcoholics, and a random population group. *Drug and Alcohol Dependence*, 73, 175-182.

Lembke, A. and Ketter, T.A. (2002). Impaired recognition of facial emotion in mania. *American Journal of Psychiatry*, 159, 302-304.

Leppanen, J.M. and Hietanen, J.K. (2004). Positive facial expressions are recognised faster than negative facial expressions, but why? *Psychological Research*, 69, 22-9.

Levenston, G.K., Patrick, C.J., Bradley, M.M. Lang, P.J. (2000). The psychopath as observer: Emotion and attention in picture processing. *Journal of Abnormal Psychology*, 109, 373–385.

Lewis, H.B. (1971). Shame and guilt in neurosis. New York: International Universities Press.

Lindsay-Hartz, J., de Rivera, J. and Mascolo, M. (1995). Differentiating shame and guilt and their effects on motivation. In J.P. Tangney and K.W. Fischer (Eds.), *Self-conscious emotions: Shame, guilt, embarrassment, and pride (pp. 274-300)*. New York: Guilford.

Lovibond, S.H. and Lovibond, P.F. (1995). Manual for the Depression Anxiety Stress Scales, (2nd Ed.). Sydney, Australia: Psychology Foundation of Australia.

Lyon, H.M., Startup M. and Bentall R.P. (1999). Social cognition and the manic defense: Attributions, selective attention, and self-schema in bipolar affective disorder. *Journal of Abnormal Psychology*, 108, 273–282.

Mandal, M.K., Pandey, R. and Prasad, A.B. (1998). Facial expressions of emotions and schizophrenia: a review. *Schizophrenia Bulletin*, 24, 299-412.

Matthews, G. and Wells, A. (1999). The cognitive science of attention and emotion. In T. Dalgleish and M.J. Power (Eds.), *Handbook of Cognition and Emotion* (pp. 171-192). New York: Wiley.

Mineka, S. and Cook, M. (1993). Mechanisms involved in the observational learning of fear. *Journal of Experimental Psychology Gen.*, 122, 23-38.

Mintzer, M.Z. and Stitzer, M.L. (2002). Cognitive impairment in methadone maintenance patients. *Drug and Alcohol Dependence*, 67, 41-51.

Montagne, B., Kessels, R.P.C., de Geus, F., Denys, D., de Haan, E.F.H. and Westenberg, H.G.M. (2005). Recognition of facial expressions in obsessive-compulsive disorder: A dimensional approach. In B. Montagne (Ed.), *Perception of facial expressions in psychiatric and neurological disorders (pp.93-108*). Febodruk B.V.Netherlands.

Meulenbeek, P.A.M. (2000). Addiction problems and methadone treatment. *Journal of Substance Abuse Treatment*, 19, 2171-2174

National Treatment Agency for Substance Misuse (2004). Enhancing outcomes of methadone maintenance treatment with counselling and other psychosocial and 'ancillary' services. Source: NTA Briefing documents on methadone treatment: National Treatment Agency for Substance Misuse, UK, 31 May 2004. www.nta.nhs.uk/publications/research briefing4.htm (retrieved 01.06.05).

Nelson, H.E. and Willison, J.R. (1991). *National adult reading test (NART)*. 2nd ed. Windsor: NFER-Nelson.

Oatley, K. and Johnson-Laird, P.N. (1987). Towards a cognitive theory of emotions. Cognition and Emotion, 1, 29-50.

O'Connor, L.E., Berry, J.W., Inaba, D., Weiss, J. and Morrison, A. (1994). Shame, guilt and depression in men and women in recovery from addiction. *Journal of Substance Abuse Treatment*, 11, 503-510.

Parker, H.A., McNally, R.J., Nakayama, K. and Wilhelm, S. (2004). No disgust recognition deficit in Obsessive-Compulsive disorder. *Journal of Behaviour Therapy and Experimental Psychiatry*, 35, 183-192.

Parker, H. and Newcombe, R. (1987). Heroin use and acquisitive crime in an English community. *British Journal of Sociology*, 38, 331-350.

Payte, J.T., Khuri, E., Joseph, H. and Woods, J. (1999). The methadone maintained patient and the treatment of pain. National Alliance of Methadone Advocates Education series number 9.

www.methadone.org/downloads/namadocuments/es09pain_management.pdf (retrieved 01.06.05)

Philippot, P, Kornreich, C., Blairy, S., Baert, I., Den Dulk, A., Le Bon, O., Streel, E., Hess, U., Pelc, I. and Verbanck, P. (1999). Alcoholics deficits in the decoding of

emotional facial expression. Alcoholism: Clinical and Experimental Research, 23, 1031-1038.

Phillips, M.L., Young, A.W.C. (Senior), Brammer, M.C. Andrew, Calder, A.J. (1997). A specific neural substrate for perceiving facial expressions of disgust. *Nature*, 389, 495–498.

Poole, J.H., Tobias, F.C. and Vinogradov, S. (2000). The functional relevance of affect recognition errors in schizophrenia. *Journal of the International Neuropsychological Society*, 6, 649-658

Power, M. and Dalgleish, T. (1997). Cognition and Emotion: From order to disorder. Psychology Press Ltd.

Rapcsak, S.Z., Galper, S.R., Comer, J.F., Reminger, S.L., Nielsen, L., Kaszniak, A.W., Verfaellie, M., Laguna, J.F., Labiner, D.M. and Cohen, R.A. (2000). Fear recognition deficits after focal brain damage: A cautionary note. *Neurology*, *54*, 575-581.

Rozin, P. and Fallon, A.E. (1987). A Perspective on disgust. *Psychological Review*, 94, 23-41.

Rozin, P., Haidt, J. and McCauley, C.R. (1993) Disgust. In M. Lewis and J.M. Haviland (Eds.), *Handbook of Emotions* (pp. 575-594). New York: The Guildford Press.

Rubinow, D.R. and Post, R.M. (1992). Impaired recognition of affect in facial expression in depressed patients. *Biological Psychiatry*, 31, 947-953.

Sprengelmeyer, R., Rausch, M., Eysel, U.T. and Przuntek, H. (1998). Neural structures associated with the recognition of facial basic emotions. *Procedures of the Royal Society of London B.*, 265, 1927-1931.

Sprengelmeyer, R., Young, A.W., Pundt, I., Sprengelmeyer, A., Calder, A.J., Berrios, G., Winkel, R., Vollmoeller, W., Kuhn, W., Sartory, G. and Przuntek, H. (1997). Disgust implicated in obsessive-compulsive disorder. *Procedures of the Royal Society of London B.*, 264, 1767-1773.

Strang, J. and Sheridan, J. (2003). Effect of national guidelines on prescription of methadone: analysis of NHS prescription data, England 1990-2001. *British Medical Journal*, 9, 32, 321-322.

Thaca, I. (1997). One Junkie's Odyssey: Drug Users and the Health Care System. http://www.harmreduction.org/pubs/news/fall97/onejunkiesodyssey.html (retrieved 01.06.05).

Townshend, J.M. and Duka, T. (2003). Mixed emotions: alcoholics' impairments in the recognition of specific emotional facial expressions. *Neuropsychologia*, 41, 773-782.

Vallejo, R., de Leon-Casasola, O. and Benyamin, R. (2004). Opioid therapy and immunosuppression: a review. *American Journal of Therapy*, 11, 354-65.

Verdejo, A., Toribio, I, Orozco, C., Puente, K.L. and Perez-Garcia, M. (2005). Neuropsychological functioning in methadone maintenance patients versus abstinent heroin abusers. *Drug and Alcohol Dependence*, 1; 78, 283-8.

Viney, L.L., Westbrook, M.T. and Preston, C. (1985). Sources of anxiety in drug addiction. *Journal of Clinical Psychology*, 41, 124-9.

Young, A., Perrett, D., Calder, A., Sprengelmeyer, R. & Ekman, P. (2002). Facial expressions of emotion: Stimuli and Tests (FEEST). UK, Thames Valley Test Company.

Zangara, A., Blair, R.J. and Curran, H.V. (2002). A comparison of the effects of a beta-adrenergic blocker and a benzodiazepine upon the recognition of human facial expressions. *Psychopharmacology Berl.*, 163, 36-41.

Chapter 3: Critical appraisal

A reflection on the research process

At the start of this research, intentions were clear. Two groups of opiate using clients, one current and one abstinent, would be tested for their recognition of emotional facial expressions. These clients would not be using any other substances or alcohol as this would make any results difficult to link directly to opiate use. However, as recruitment began, it became clear that this was just not a clinical reality. Keyworkers at the treatment centre initially looked through their caseloads to identify potentially suitable participants, but as the list of exclusion criteria were applied to each candidate, names were crossed off one by one. Certainly in the group of methadone maintained clients served by this North London treatment centre, there were few who did not additionally use some combination of crack, benzodiazepines, cannabis and/or alcohol. Therefore a pragmatic decision was taken to include participants with some concomitant drug use, which also served to improve the ecological validity of the study (this is discussed in more detail later).

This highlights how unfamiliar with the area of substance misuse I was prior to this research, and how much I learnt about this client group over the year-long process of recruitment. A more careful look at the profile of service users while formulating my research question would have been prudent.

The recruitment involved in conducting this research was arduous, given the methadone maintained client group. While they were very pleasant, warm individuals, they were amongst the most disorganised and difficult to access that I

have come across doing research. The transient lifestyles that they lead do not lend them to being reliable participants; appointments with drug treatment staff are the only appointments that many of them have in their lives, and they find these hard to keep, instead using an informal 'drop-in' system. Anecdotally, the DNA rate with this population tends to be around 50%. Many participants also found it difficult to be in a room concentrating for the one and a half hours that testing required; this was certainly a factor that discouraged many from participating. On the whole, those who did take part initially seemed to see it as a favour to their drugs worker. Additional use of drugs and alcohol also proved a problem, as some participants arrived for testing having recently used alcohol or crack, and had to be asked to return when sober.

In the methadone maintained group, for some the interview process was an opportunity to talk to somebody outside of their world who did not have the same time pressures as treatment centre staff. The use of a mood state measure frequently led to discussion of how they were feeling and difficulties they were going through. The need to be vigilant for signs of significant distress or suicidal thoughts, and to deal with these appropriately and responsibly was clear. In gathering information regarding history of drug use and crime, participants ended up sharing many of their experiences, both positive and negative. Some participants enjoyed this while others found it distressing. A great deal of personal information was shared during each testing session, which enriched the research process and gave me a window into a client group that I previously knew little about. Certainly one of the most useful learning points of the study was my increased awareness of the highly complex needs of this client group.

Consideration was given to the ethical dilemma of offering payment to methadone maintained participants. As a drug-using population in receipt of little income, there was a question over whether participants would take part irrespective of whether they wished to, in order to receive the payment. In order to maximise informed consent, participants were given time to consider the study which was then explained fully. I was also aware of the possibility that payment would be exchanged for drugs on leaving the site. Therefore, payment was given in the form of supermarket vouchers in order to encourage the purchase of non-drug items.

Testing the rehab participants was a very different experience to testing the methadone maintained group. I tested them after the methadone maintained group, which gave a feeling of progression to the process. Having seen the entrenched difficulties faced by the first group, it was a relief to see that there could be a 'next stage'. This made me feel less despondent about the apparent hopelessness of those caught in the cycle of serious drug use.

While the rehabilitation programmes used for recruitment varied in length, therapeutic style and whether they were daytime-only or residential, they all offered clients an intensive therapeutic experience in a pleasant, empathic setting. Although testing had to be carefully scheduled around therapy groups and community meetings, clients were eager to participate in their free time and receive payment that would be spent on things other than drugs. Several told me that they had participated in research during their drug-using days, and exchanged their voucher payment for

some drug-related commodity. They were pleased to be able to do something different this time.

Some participants had entered rehab relatively recently (i.e. 6 weeks before) while others were approaching the end after one year. This time had been spent going through an intensive therapeutic process which involved confronting many unpleasant and long-avoided issues, experiences and aspects of themselves. Many of the clients had become used to 'telling their story' about drug use and themselves, which often poured out during interviewing, giving me an insight into the changes that occur in how people see their drug use at different stages of use. For example, one of the questions used to elicit information about the severity of other drug problems was "and has your x use ever been problematic, as you see it?" Where the current drug-using group invariably answered "no", particularly regarding cannabis and alcohol, the clients in rehab invariably responded "if you'd have asked me x months ago, I would have said no, but now I would say without a doubt, yes".

Two rehab participants reported that they had found the interview unpleasant, due to memories of their past lives and selves that they would rather forget, being prompted by my questions about past drug use. It might be relevant that these two participants were two of the four who were enrolled in the day rehab programme. One could speculate that day programmes make continued avoidance easier than their residential counterparts, which immerse clients in the therapeutic process to a greater degree. Of the remainder of the participants, most commented that they had enjoyed the interview, saying that it had been interesting to do something different.

I was fortunate enough to be able to share the recruitment and interviewing process with a fellow trainee clinical psychologist who was conducting research with the same population. This certainly added to my experience of the research. We were able to help maintain each other's motivation and interest through the difficult times in recruitment in the way of a 'tag-team', and share the thrill of completing testing after a year of hard work. However, this meant that I did not test all the participants myself, and did not get the experience of testing the control group.

This research was initially approached from a pharmacological theoretical viewpoint, i.e. that the pharmacological changes brought about by opiate use were likely to have an impact on FER, as in other populations with altered neurotransmitter functioning. Literature in the area of facial expression recognition leans quite heavily in this direction, although more recent research seems to be integrating the neuropharmacological with the psychological to a greater extent. When trying to interpret the results of this study, it became clear that there were so many potential pharmacological effects brought about by opiate use, and in combination with crack or benzodiazepine use, and possibly a history of other drug or alcohol dependence, the results simply could not be attributed purely to direct opiate action. It also became clear that characteristics of the population such as their upbringing, current living environment, antisocial behaviour, low self-esteem and psychological difficulties were likely to have a significant impact on perception of emotion.

Additionally, spending so much time in the treatment centre gave a flavour of how it must feel to be a service user, with all that this entails for one's sense of identity. It is a large intimidating building located in an inner city area, towards which local

residents are hostile, and there are groups of service users drinking cans of strong lager congregating outside. Inside, a security guard, restricted entry and a partition between staff and clients, and most strikingly of all a smell that fluctuates between mildly unpleasant and abject rotting. The service provides treatment for the highest number of methadone maintained clients in the UK, many of whom are homeless and suffer significant psychological, social, legal and health problems. It should be said that the staff are highly skilled and work hard to counter this aversive environment. However, the experience of being in this environment (also where testing was conducted) seems very relevant, especially when discussing a result such as enhanced recognition of disgust. This was in comparison to the clean, welcoming, well decorated, Edwardian and Tudor buildings that constituted the rehabilitation centres. In this way, psychological and social factors began to seem at least as relevant as the neuropharmacological as the study progressed.

Critical Appraisal

The main finding of the study was that opiate users demonstrated enhanced recognition of other peoples' disgust expressions. This finding is striking given that enhanced FER has rarely been reported in the vast literature on FER in psychiatric, neurologically impaired and drug-using populations. Moreover, the literature surrounding the expression of disgust paints a picture that is so inconclusive that it is unclear what such a finding might mean. There are theoretical indications that disgust may be important in negative socialisation, theoretical and (some) empirical evidence that disgust is an important emotion in OCD (Power and Dalgleish, 1997), and empirical evidence of enhanced disgust recognition in bipolar individuals

(Harmer, Grayson & Goodwin, 2002). However, the literature does not draw the evidence together in a meaningful way.

I have speculated about the possible roles of neuropharmacological changes due to opiate use, the possible survival advantage of enhanced disgust recognition conferred to opiate users due to their living environment and consequent susceptibility to illness, hypersensitivity of this population to others' disgust reactions and a possible connection with shame. Beyond this, the paucity of literature in this area makes conclusions difficult to draw. Replication of this study is now needed, with the addition of measures of self-perception and shame. It is clear that more research into the function of disgust with regard to psychopathology and social functioning would illuminate our understanding of this phenomenon.

Methadone maintained individuals' heightened perception of disgust has important implications for service providers. Staff need to be aware of their non-verbal reactions to clients, and if supported by further research evidence, of the potential that they and the environment in which they work, have to evoke feelings of shame in their clients. Such feelings are likely to be extremely aversive and could be implicated in treatment dropout (Viney, Westbrook & Preston, 1985). If it is found that enhanced disgust recognition is a measure of shame, this provides further support regarding the need for shame-based interventions with this population.

The clients used in this study were not homogenous in their drug use or purely-opiate using, meaning that it is difficult to draw tight theoretical conclusions about the effects of opiates on the basis of any results. However the clinical usefulness of any

conclusions drawn from such a homogeneous sample would be questionable, as this population is so rarely seen in real-life settings. A considerable strength of the current study is the degree of ecological validity it attains due to the samples used. Limits were set on how frequent and problematic additional drug use was, in order to minimise the effect of other factors such as crack use. This, in addition to the motivational (i.e. being interested), organisational (i.e. turning up for a testing appointment) and time requirements of the testing procedure (one and a half hours), necessarily created a selection bias in favour of the less chaotic clients. In this sense, the sample used in this study are likely to represent the more stable end of the opiate-using spectrum seen for treatment in North London. This said, a more representative sample simply was not testable.

The comparability of the groups also adds to the validity of the current study. As demonstrated by the demographic measures used, the rehab group was broadly equivalent to the methadone maintained group. The only difference between the two clinical groups seems to have been the age of onset of opiate use and length of opiate exposure. It is possible that this is an indicator of qualitative differences between the groups, although this could only be verified using a measure of pre-morbid functioning. That the control group consisted of unemployed individuals living in a similar inner-city area also adds to the comparability of the groups. Additionally, the use of urine screening with the clinical groups confirmed their current drug use status. All of these features increase the potential for meaningful conclusions to be drawn.

117

The use of all male samples was important for several reasons. Firstly, gender differences have been shown in FER ability, with women outperforming men on tests of emotion recognition (Hall, 1978; Montagne, Kessels, de Geus, Denys, de Haan, Westenberg, et al., 2005). Secondly, research indicates that men and women with substance misuse problems may enter treatment with different problems and emotional needs. Women and men in treatment have also been found to differ in their use of substances other than heroin, interpersonal relationships, drug dealing, employment and criminal behaviours (Anglin & Hser, 1987), as well as with respect to why they started using drugs (Dobler-Mikola & Zimmer-Hofler, 1993). In addition, a higher incidence of depression has also been found in drug-addicted women than men (O'Connor, Berry, Inaba, Weiss & Morrison, 1994). Therefore, the use of a purely male sample reduced variability not due to opiate use.

Although not the primary focus of the study, the collection of information about involvement in violent crime and the use of measures such as the AQ, BIS and Gough build an interesting profile of the type of clients using drug treatment services. That the picture which emerged fits with existing research findings again improves the validity of this study.

Conducting this research, alongside completing a clinical placement in the substance misuse service from which the methadone maintained sample were recruited, has fuelled my interest in working clinically with this population post-qualification. I also have a better grasp of the potential difficulties involved in conducting research with this client group, and how to minimise these pitfalls. This is an underresearched client group, due no doubt in part to the difficulties I have already

described, but one with complex needs, including a significant prevalence of dual diagnosis. More research in clinical settings will be important in better understanding these needs, both for individual client work and service planning. This is true for most areas of clinical psychology practice, where, in my experience through training placements, research conducted alongside clinical work is simply not a reality. I would hope to be able to incorporate my research skills into my clinical work with this client group in the future. In reviewing the literature for this thesis, a particularly interesting area in which little research has been conducted was highlighted: the process of change that service users undergo as they make the transition from user to ex-user via rehab. For example, the motivational factors that prompt the decision to change, the changes in how they perceive their drug use and their sense of self, as well as what it is that they undergo in rehab that helps or does not help with abstinence would all make for interesting future research.

References

Anglin, M. and Hser, Y. (1987). Addicted Women and Crime. Criminology, 25, 359-97.

Dobler-Mikola, A. & Zimmer-Hofler, D. (1993). K'ientinnen drogentherapeutischer Gemeinschaften in der Schweiz zwischen 1978/79 und 1990/91. *Drogalkohol* 2, 69-82.

Hall, J. (1978). Gender effects in decoding nonverbal cues. *Psychological Bulletin* 85, 845-857.

Harmer, C.J., Grayson, L. and Goodwin, G.M. (2002). Enhanced recognition of disgust in bipolar illness. *Biological Psychiatry*, 51, 298-304.

Montagne, B., Kessels, R.P.C., de Geus, F., Denys, D., de Haan, E.F.H. and Westenberg, H.G.M. (2005). Recognition of facial expressions in obsessive-compulsive disorder: A dimensional approach. In B. Montagne (Ed.), *Perception of facial expressions in psychiatric and neurological disorders (pp.93-108*). Febodruk B.V.Netherlands.

O'Connor, L.E., Berry, J.W., Inaba, D., Weiss, J. and Morrison, A. (1994). Shame, guilt and depression in men and women in recovery from addiction. *Journal of Substance Abuse Treatment*, 11, 503-510.

Power, M. and Dalgleish, T. (1997). Cognition and Emotion: From order to disorder. Psychology Press Ltd.

Viney, L.L., Westbrook, M.T. and Preston, C. (1985). Sources of anxiety in drug addiction. *Journal of Clinical Psychology*, 41, 124-9.

Appendix A

Permission letter from Camden and Islington NHS Trust Ethics Committee



Camden & Islington Community Health Services Local Research Ethics Committee

LREC Ref: 03/106

PRIVATE AND CONFIDENTIAL
Professor V Curran
Sub-Department of Clinical Health Psychology
University College London

30 March 2004

Dear Professor Curran

Title: Methadone maintenance and the interpretation of sentences and emotions.

Thank you for your email of 26th March 2004, which addressed the points raised by the Ethics Committee at their meeting on 23rd February 2004. I am pleased to inform you that after careful consideration the Local Research Ethics Committee has no ethical objections to your project proceeding. This opinion has also been communicated to the North Central London Community Research Consortium.

PLEASE NOTE THAT THIS OPINION ALONE DOES NOT ENTITLE YOU TO BEGIN RESEARCH, YOU MUST RECEIVE AN APPROVAL FROM EACH NHS TRUST HOSTING YOUR RESEARCH.

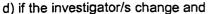
Camden and Islington Community Health Service LREC considers the ethics of proposed research projects and provides advice to NHS bodies under the auspices of which the research is intended to take place. It is that NHS body which has the responsibility to decide whether or not the project should go ahead, taking into account the ethical advice of the LREC¹. Where these procedures take place on NHS premises or using NHS patients, the researcher must obtain the agreement of local NHS management, who will need to be assured that the researcher holds an appropriate NHS contract, and that indemnity issues have been adequately addressed.

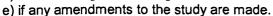
N.B. Camden and Islington Community Health Service LREC is an independent body providing advice to the North Central London Community Research Consortium. A favourable opinion from the LREC and approval from the Trust to commence research on Trust premises or patients are NOT one and the same. Trust approval is notified through the Research & Development Unit (please see attached flow chart).

The following conditions apply to this project:

- You must write and inform the Committee of the start date of your project. The Committee (via the Local Research Ethics Committee Administrator or the Chair at the above address) <u>must</u> also receive notification:
 - a) when the study commences;
 - b) when the study is complete;
 - c) if it fails to start or is abandoned;

¹ Governance Arrangements for NHS Research Ethics Committees, *July 2001 (known as GAFREC)*An advisory committee to North Central London Strategic Health Authority







- The Committee <u>must</u> receive immediate notification of any adverse or unforeseen circumstances arising out of the project.
- It is the responsibility of the investigators to ensure that all associated staff, including nursing staff, are informed of research projects and are told that they have the approval of the Ethics Committee and management approval from the body hosting the research.
- The Committee will require a copy of the report on completion of the project and may request details of the progress of the research project periodically (i.e. annually for longer projects).
- If data is to be stored on a computer in such a way as to make it possible to identify individuals, then the project must be registered under the Data Protection Act 1998. Please consult your department data protection officer for advice.
- Failure to adhere to these conditions set out above will result in the invalidation of this letter of no objection.

Please forward any additional information/amendments regarding your study to the Local Research Ethics

Committee Administrator or the Chair at the above address.

LRÉC Chair	
Email:	(administrator)
Enc/s:	
Copy to:	

Yours sincerely

Appendix B

Depression Anxiety Stress Scale (DASS)

Appendix C

Fast Alcohol Screening Test (FAST)

Appendix D

Spot-the-Word Test

Appendix E

Barratt Impulsivity Scale (BIS)

Appendix F

Gough Socialisation Scale

Appendix G

Aggression Questionnaire (AQ)

Appendix H

Cut-down Annoyed Guilty Eye-opener Scale : Adapted to Include Drugs (CAGE-AID)

Appendix I

Participant Information Sheet (methadone maintained clients)

Participant information sheet

Research Study: Methadone and the interpretation of sentences and emotions

Researchers: Louise Martin and Jo Coyle (Trainee Clinical Psychologists)

You are invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please read the following information. Please ask us if there is anything that is unclear or if you would like more information. Take time to decide whether you wish to take part.

What is the purpose of the research project?

To understand what effect methadone has on the way people understand sentences and facial expressions. Research has shown that different drugs affect these two things. In this study we are looking at 1) people who use methadone at the moment, 2) people who no longer use methadone or heroin, and 3) people who have never used methadone.

Why have I been chosen?

We have asked you to take part in the study because you are using methadone at the moment. We will also be approaching around 30 other people who currently use methadone.

Do I have to take part?

You do not have to take part in this study if you do not wish to. Your decision to take part will not affect your care management in any way. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you do decide to take part, you can withdraw at any time without having to give a reason.

What will happen if I take part?

We will arrange to meet you once for about 1-hour at the Margarete Centre, after you have taken your methadone. First we will ask you a little about your drug use. You will then be shown some sentences and faces on a computer and asked to make some decisions about them. We will also ask you to complete some questionnaires and provide a urine sample. When this is completed, we will give you a voucher worth £6. All information collected about you during the study is strictly confidential and will be coded by number. Your name will not appear on any forms.

What are the advantages and disadvantages of taking part?

We do not foresee that taking part will cause you distress. We hope that the information we collect from this study will improve our understanding of the effects of methadone, and so help to improve services to methadone clients.

What will happen to the results of the study?

The results will be written up as part of a thesis, which we hope will be published in a scientific journal. A summary of the findings will be available to all who took part.

Who is organising and funding the study?

The study is organised and funded by Camden and Islington NHS Trust and University College London.

Contact for further information:

If you would like further information or have any questions, then please leave a message for us at the Margarete Centre.

Thank you for taking time to read this.

Date: 14th July 2004

All proposals for research using human subjects are reviewed by an ethics committee before they can proceed. This proposal was reviewed by Camden and Islington Health Services NHS Trust Ethics Committee.

Appendix J

Participant Information Sheet (rehab clients)

Participant information sheet (opiate abstinent clients)

(CORE Trust)

Research Study: Methadone maintenance and the interpretation of sentences and emotions

Researchers: Louise Martin and Joanna Coyle

You are invited to take part in a research study. Before you decide, it is important for you to understand why the research is being done and what it will involve. Please read the following information. Please ask us if there is anything that is unclear or if you would like more information. Take time to decide whether you wish to take part.

What is the purpose of the research project?

To understand what effect using methadone has on the way people interpret sentences and facial expressions. Research has shown that different drugs affect these two functions. In this study, we are looking at 1) people who are using methadone at the moment, 2) people who no longer use methadone or heroin, and 3) people who have never used.

Why have I been chosen?

We have asked you to take part in the study because you are no longer using methadone or heroin. We will also be approaching around 20 other people who are currently abstinent.

Do I have to take part?

You do not have to take part in this study if you do not wish to. Your decision to take part will not affect your healthcare or management in any way. If you do decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you do decide to take part, you can withdraw at any time without having to give a reason.

What will happen if I take part?

We will arrange to meet you once for around 1 hour at the CORE Trust. First we will ask you a little about yourself and your drug use. Then we will ask you to complete some questionnaires. After this you will spend around half an hour doing some tasks on a computer. These will include making decisions about sentences and faces that you are shown. When this is completed, we will give you a voucher worth £6. We would like to take a urine sample, just to confirm that you are not using drugs. The results of this would be confidential and not fed back to the CORE Trust. All information collected about you during the study is strictly confidential and will be coded by number. Your name will not appear on any forms.

What are the advantages and disadvantages of taking part?

We do not foresee that taking part will cause you distress. We hope that the information we collect from this study will improve our understanding of the effects of methadone, and have implications for improving services to clients.

What will happen to the results of the study?

The results will be written up as part of a thesis, which we hope will be published in a scientific journal. A summary of the findings will be available to all who took part.

Who is organising and funding the study?

The study is organised and funded by Camden and Islington NHS Trust and University College London.

Contact for further information:

If you would like further information or have any questions, you can contact us at the Margarete Centre on 020 75303086.

Thank you for taking time to read this.

Date: 15.12.04

All proposals for research using human subjects are reviewed by an ethics committee before they can proceed. This proposal was reviewed by Camden and Islington Health Services NHS Trust Ethics Committee.

Appendix K

Consent Form (both methadone maintained and rehab clients)

Participant identification code:

Consent Form (Methadone maintained and opiate abstinent clients)

Confidential

	Research Stud	y: Methadone maintenance and the interpre	etation of sentences and en	notions
		Name of researchers: Louise Martin an	d Joanna Coyle	
1.	I confirm that I have read and that I understand the infoabove study.		nation sheet dated	for the
			YES/NO	
2.	I have had an o	opportunity to ask questions and discuss thi	s study. YES/NO	
3.	•	at I am free to withdraw from this study: at any time without reason without affecting my healthcare and manage	gement at the Margarete C	entre.
			YES/NO	
4.	I agree to take	part in the above study.	YES/NO	
Nam	e of participant	Date	Signature of participant	
 Rese	archer	Date	Signature of researcher	