# Facial expression perception in different psychiatric groups

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# ABSTRACT

Facial expressions are salient cues that guide our interpersonal behaviour. Many individuals suffering from psychiatric conditions experience difficulties interacting socially with others and often become withdrawn, which in turn serves to maintain their negative psychological state. Misinterpretation of facial expressions could contribute to such interpersonal problems.

A new interactive method was designed to assess recognition of and sensitivity to different facial expressions of emotion, to investigate the nature of any deficit in facial expression perception in different psychiatric patient groups. The efficacy and possible diagnostic value of this method, which measures more than one aspect of facial expression processing, was demonstrated by the presence of differential impairment in these subject groups.

A preliminary study revealed differential sensitivity to facial expressions according to depressive mood state in a non-psychiatric student population. A follow-up study on clinically depressed patients revealed that depressives required a greater intensity of expression to detect any emotion than controls. In spite of this lack of responsiveness to emotional cues, patients were still able to assign the correct verbal labels to different expressions. Clinically depressed individuals were also found to be relatively *more* sensitive to expressions of anger, but relatively *less* sensitive to expressions of happiness and disgust than control subjects. A subclinical group of age-matched subjects with moderately high depression scores exhibited a response pattern approximating that of clinical patients, suggesting that differences according to level of depression are quantitative rather than qualitative.

Facial expression perception was also investigated in patients undergoing psychosurgery for intractable depression. Impaired perception of fear was observed post surgery, which could relate to disturbance of neural connections with the amygdala and frontal cortex.

The hypothesis that sensitivity to expressions of disgust would be enhanced in individuals suffering from eating disorders due to extreme experience of self-loathing was confirmed. Diminished overall sensitivity to all facial expressions was revealed which could relate to difficulties in emotional awareness. In addition, relative insensitivity to happiness was exhibited which might reflect concomitant anhedonic mood state.

# CHAPTER 1: INTRODUCTION

Social interaction and communication play an integral role in human existence. In order to socialise effectively we need to be able both to comprehend other individuals' social signals to predict their behaviour as well as express ourselves appropriately in response. A great number of cues – both verbal and non-verbal – contribute to human social communication (e.g., speech content, voice intonation, facial expressions, gaze direction and bodily gestures) and enable inference about the intention of others.

The particular aspect of social communication that this thesis focuses on is facial expression perception. Facial expressions are salient non-verbal cues directing behaviour in social interactions. The ability to accurately decode and interpret others expressions is crucial for efficient social functioning. Facial expressions can communicate intention and indicate internal emotional state, from which the perceiver should be able to infer the desired and appropriate response for effective interaction. Unfortunately, this interpretative ability is not possessed equally by all individuals. Psychiatric groups seem particularly prone to experience problematic social interactions. Many individuals with psychiatric conditions tend to be socially isolated and withdrawn and often become ostracised from normal social groupings. Such social difficulties are probably a consequence of a variety of factors but deficient processing of others' facial expressions could play an important contributing role. Lack of social support is known to have a negative impact on an individual's general well-being, due to its moderating role on the effect of stressful life events (Berkman, 1995; Berkman & Syme, 1979; Cobb, 1976). In addition, social isolation will restrict the range of activities that can be undertaken with others,

which is in turn likely to maintain an internal focus of attention and exacerbate any negative self-directed cognitions. The resulting effect of such deleterious interpersonal interactions, which might be caused by incorrect appraisal of and response to others' non-verbal emotional cues, will be maintenance of negative psychological state. Therefore, even if detrimental social interactions only arise as a consequence of psychological conditions, once present, they are likely to have a pathologic effect on the course of an illness. Further research into putative contributing factors in the development of such interpersonal difficulties in relevant psychiatric populations is therefore of paramount importance.

The aim of this thesis is to examine systematically the nature of any deficits in facial expression processing in two particular psychiatric groups, both of which are known to experience interpersonal difficulties: individuals with depression and those suffering from eating disorders. (Reviews of relevant literature and a general background on each condition can be found in later chapters.) Studies to-date investigating facial expression perception in different subject populations have focused on evaluating the presence of impairments in *recognition of* certain 'basic' expressions. It was anticipated that more subtle deficits might be apparent in these two psychiatric groups and might centre on difficulties in expression *interpretation*, i.e., sensitivity to perceive an emotion, in the absence of any gross difficulties in emotion recognition. For example, a depressed individual might be as capable as a non-depressed person of recognising emotions when presented at full intensity, but might nonetheless be prone to perceive less intense, ambiguous expressions as hostile or disapproving.

Methodologies to-date have only really been sufficient to assess severe deficits in recognition. As sensitivity to different expressions, i.e., the ability to

detect an expression at different intensities, is believed to be altered in psychiatric groups, there has been a corresponding necessity to develop a more sophisticated test. This thesis has endeavoured to address this issue with the extension of a method developed by Calder, Young and colleagues in studies of categorical perception (see section 1.2).

The purpose of this introductory chapter has been firstly to give a brief exposé of the rationale behind this thesis. The following sections of this chapter will focus on evidence for the presence of neural systems dedicated to processing emotional signals, with specific focus on brain areas involved in facial expression perception, by reviewing neurophysiological, neuropsychological and imaging literature. Subsequently the psychological evidence for the presence of several 'basic' universal expressions of emotion will be discussed, with regard to the stimuli chosen for study in the current thesis. A brief outline and justification for the type of methodology employed here is then stated. The chapter will conclude with a short discussion of the relation of emotional experience (particularly in altered psychiatric states) to emotion perception.

## 1.1 A NEURAL BASIS FOR EMOTION

The likelihood that primates have evolved a specialised neural system underlying social communication is high, due to increasingly complex social cultures. Indeed, primate brain size has been shown to increase relative to the social complexity of the environment inhabited (Dunbar, 1998, 1992). The neural basis underlying processing of emotional cues is gradually becoming better understood as

research findings from animal and human physiology, neuropsychological studies of functional impairment and imaging studies are accumulated.

### 1.1.1 Early visual processing of social signals

The presence of cells with selective response properties for faces was first observed by Gross et al. (1972) in the macaque monkey. Face-selective neurons have been identified in the inferior temporal (IT) and superior temporal sulcus (STS) of the monkey (Perrett et al., 1984, 1982; Baylis et al., 1985), most of which have been shown to exhibit view selectivity. In spite of preferential response to particular face view (for example, profile over frontal), generalisation across different lighting and orientation conditions does occur, enabling identity recognition across different settings.

Several research groups (Mistlin & Perrett, 1990; Hasselmo et al., 1989; Perrett et al., 1984) discovered cells selectively responsive to facial expressions in the monkey STS, which appear to be anatomically distinct from those coding face identity. These cells do not seem to merely respond to morphological changes in the face (such as an open mouth for anger), but appear to code the overall 'sense' of the expression (i.e., do not respond to a similar visual image of an animal chewing food). A small percentage of visually responsive neurons in the amygdala also appear to be dedicated to processing facial (identity and expression) information (Brothers, 1997; Leonard et al., 1985). Face processing is also apparent at the cellular level in several other cortical areas, particularly in orbitofrontal (Thorpe et al., 1983) and prefrontal (O'Scalaidhe et al., 1997) cortex.

The ability to monitor attention is also fundamental in social behaviour. Perrett et al. (1992, 1985) found subgroups of cells in the STS (intermixed with

those tuned to expression) that appeared to code attention, in that differential response was shown according to direction of gaze, head view and body posture. Processing of gaze information reveals the object of an animal's attention, i.e., to whom a threat or affiliative gesture is directed. It would appear therefore that cellular systems have appropriate response capabilities for analysing and interpreting the emotions and intentions of others from visual signals.

Prkachin and Prkachin (1994) provided psychological evidence of the presence of expression-specific neurons in humans, using an adaptation paradigm. Subjects were instructed to attend continuously to an image of a particular facial expression for 5 minutes, before being asked to identify a set of different facial expressions (posed by various individuals). Poorer recognition was subsequently shown for the adapted emotion, in spite of accurate identification of non-adapted emotions. The authors believed this indicated the presence of feature-detectors selective for expression, in accordance with the physiological findings above, in that perceptual ability decreased following fatigue (due to overexposure) *only* for the relevant emotion.

### 1.1.2 Neural circuits implicated in processing emotion

Early physiology studies on animals implicated limbic circuitry[1] as a probable neural substrate for affect, with particular focus on the amygdala (situated in the medial temporal lobe) as an area encoding emotional salience following demonstration of its role in fear conditioning (LeDoux, 1995, 1992). The complex network of projections surrounding the amygdala supports the prediction that it is an area of significance, with efferent connections to the striatum, brain stem and

[1] See Figure 1.1 for a simplistic diagram of limbic regions implicated in emotion processing

hypothalamus, as well as incoming projections from STS and other higher cortical areas (Amaral et al., 1992; Emery & Perrett, in press). The area implicated as a centre for these inputs is the basolateral nucleus of the amygdala (Emery & Perrett, in press). Highly specialised visual information processed in temporal cortex is projected to the amygdala, where it is believed that sensory signals are related to emotional states. Integration of socially significant visual information has been postulated to occur in anterior STS, which projects to the amygdala (Perrett et al., 1997) amongst other regions.

Damage following amygdala lesions in both humans and animals suggests that the amygdala plays a particularly important role in both perception and experience of fear (see also section 1.1.5). Surgical lesions of the anterior temporal lobes, initially examined by Klüver and Bucy (1937), lead to inappropriate sexual behaviour and tameness in socially isolated monkeys. Lesions of the amygdala alone in group-caged monkeys resulted in the same set of behaviours (for review see Kling & Brothers, 1992). Kling emphasised the importance of studying animals in their natural habitat when investigating brain areas believed to mediate social function. He performed amygdalectomies (see Kling & Brothers, 1992) on several different species of monkey living in social groups in a semi-natural environment and found the common effects of the lesion across all species were the inappropriate fear displayed (particularly to affiliative gestures from conspecifics) and an increase in emotional unresponsiveness. For example, lesioned animals would withdraw from a friendly approach made by a normal animal. Likewise, social interaction was diminished and amygdala lesioned animals lost rank in the dominance hierarchy. Hypersexual behaviour, as seen in caged animals in the Klüver-Bucy syndrome, was

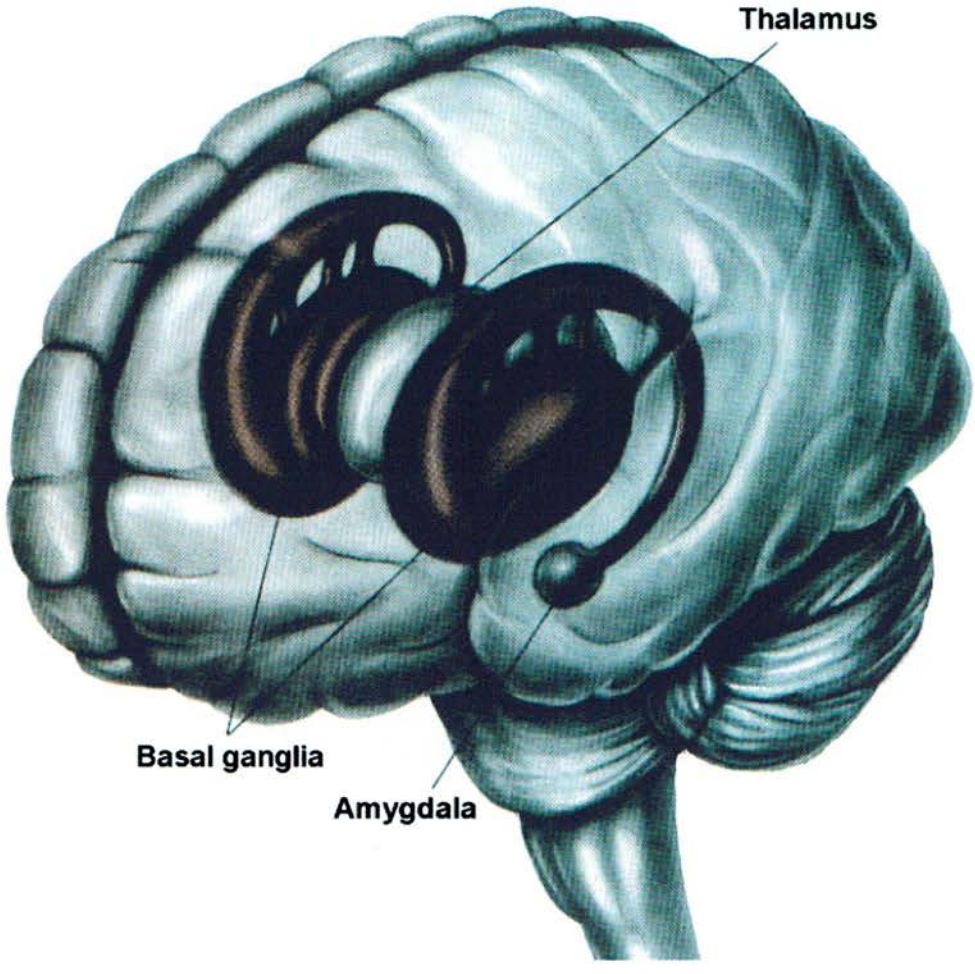


**Figure 1.1: A crude diagram of basic limbic circuitry (amygdala, basal ganglia) taken as a transparent view of the brain.**

not present in free-ranging animals with amygdalotomy however, revealing the influence of the social environment on neural processing.

The suggestion that the amygdala plays a role in the modulation of social behaviour is further strengthened by observation of the effects of amygdala damage in humans, with selectively impaired recognition of expressions of fear resulting (see section 1.1.5).

The orbital frontal cortex (located in the ventral part of the frontal lobe, see Figure 1.2) also appears to play a role in regulating social behaviour, which has lead to the proposal of the 'basolateral circuit' (Brothers, 1996) - composed of the amygdala (basolateral nucleus), orbitofrontal and temporal cortex - as a mediator of social information processing. The rich connectivity of these areas with brain regions known to be involved in memory (e.g., the hippocampus) indicates that the meaning of different social experiences is stored and such information later accessed to guide responses in similar encounters.

Disruption of the normal extinction process following aversive conditioning has been shown to occur after prefrontal lesions in rats (Morgan et al., 1993), indicating the involvement of orbital frontal cortex in rapid learning of associations between stimuli and reinforcers. Facial expressions are important social signals, which act as reinforcers to alter behaviour. Orbital frontal damage might therefore result in inappropriate behavioural change in addition to incorrect recognition of facial affect. Indeed, orbital frontal lesions in monkeys result in aversion to social interactions (Raleigh & Steklis, 1981) and alteration of social roles. In humans, a consequence of orbital frontal damage is a condition known as 'acquired sociopathy' (Damasio et al., 1990), which manifests itself in inappropriate social behaviour, disinhibition and inability to make effective social judgements.

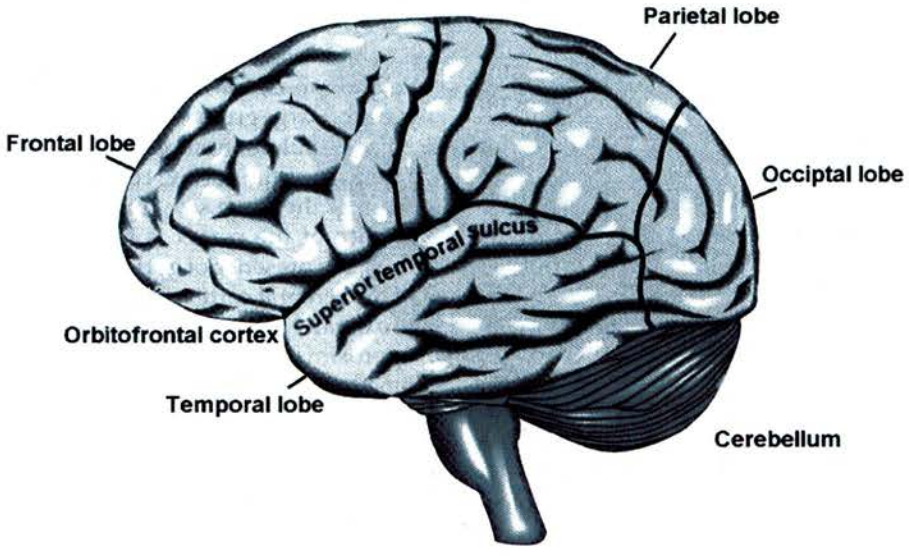


**Figure 1.2: A simplistic diagram of the human brain, including regions specifically implicated in face perception (superior temporal sulcus) and emotion processing (frontal lobe, particularly orbitofrontal cortex).**

Ability to identify facial expressions was assessed in a group of individuals with damage to a similar area, in the ventral part of the frontal lobe, by Hornak et al. (1996). Subjective emotional experience and behaviour was examined concurrently in an attempt to establish the relationship between perception, experience and expression of emotion. General impairments in processing facial expressions were revealed in most of these patients. Of particular interest was the association between subjective emotional experience, objectively observed behaviour and affect perception, with patients who experienced and manifested the most severe alterations in emotional state in turn exhibiting the greatest impairment in decoding expression (both facial and vocal). These individuals were also rated as demonstrating extremely dysfunctional behaviour by hospital staff. Although further research is required on emotion processing in this patient group, it might be expedient for rehabilitation strategies to focus on retraining of emotional cues.

Neuronal responsiveness to both positively and negatively valenced emotional stimuli has also been observed in orbitofrontal cortex in the monkey (Thorpe et al., 1983). Consistent with this, functional imaging has revealed activation of lateral orbitofrontal cortex in normal subjects following *both* depressed *and* elated mood induction procedures (Baker et al., 1997), implicating the role of this area in both positive and negative mood states.

Converging evidence from neurophysiology, neuropsychology and functional imaging therefore seems to suggest a fairly general but important role for the orbitofrontal cortex in regulation of emotional behaviour. Indeed, disconnection of orbitofrontal cortex from thalamic centres (*subcaudate tractotomy*) has been conducted as an extreme remedial measure for intractable depression, to reasonable effect (Bridges et al., 1994).

### 1.1.3 Neural circuits implicated in processing facial affect

In accordance with Bruce and Young's (1986) original model of face processing (see Figure 1.3), the neural circuits underlying facial expression processing seem to be distinct (if overlapping) from those directing other aspects of face processing, such as identity recognition. The reason for functionally and anatomically specific systems appears logical, in the sense that it is vital for an individual to be able to recognise transient changes in emotion irrespective of more invariant facial properties such as identity.

Neuropsychological studies in humans documenting inability to identify familiar faces (*'prosopagnosia'*) in the absence of impairment in emotion recognition (Tranel et al., 1988; Shuttleworth et al., 1982), or conversely those reporting intact face recognition in spite of deficient facial expression processing (Young et al., 1995; Adolphs et al., 1994), confirm the anatomically discrete nature of neural areas for identity and expression processing.

Adolphs et al. (1996) examined facial expression perception in 37 patients with different types of brain damage. Patients were asked to rate each expression shown as to the degree of intensity with which it portrayed each of the 6 'basic' emotions. Ratings were compared with those from control subjects to compute recognition accuracy. Right inferior parietal and mesial anterior infracalcarine cortex were delineated as the neural areas most commonly associated with impaired recognition of affect. It is notable though that parietal activation could be a consequence of patients attempting to mimic the expression displayed, with proprioceptive feedback from the facial muscles aiding identification of the emotion and judgements of emotional intensity. Individuals with lesions confined to the left hemisphere did not exhibit any deficits, again highlighting the dominant role of the

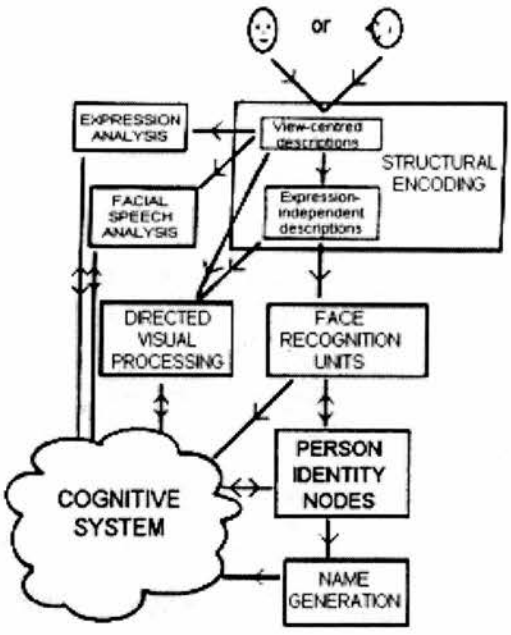


**Figure 1.3: Bruce and Young's (1986) model of face processing, indicating distinct but interconnected substrates mediating different aspects of face perception.**

right hemisphere in processing emotion (see section 1.1.4). Interestingly, all patients demonstrated intact perception of happiness, which could indicate that processing of positive emotion is subserved by different neural structures to those required for perception of negative affect (see section 1.1.4).

Increased activation in right anterior cingulate cortex and bilaterally in the inferior frontal gyrus was revealed with positron emission tomography (PET) imaging in healthy woman during a facial emotion matching task (compared to control tasks) in a study by George et al. (1993). A further functional magnetic resonance imaging (fMRI) study by Sprengelmeyer et al. (1998) also revealed activation of left inferior frontal cortex during passive recognition of different facial expressions (namely, anger, fear and disgust) in healthy subjects. Selective activation

of discrete neural areas was also observed by Sprengelmeyer et al. (1998) for different emotions, e.g., insula cortex for disgust (see section 1.1.5), implicating a multi-stage process in perception of emotion. Inferior, medial and orbital frontal regions therefore appear to play a general role in emotion processing, with reciprocal projections to specific neural substrates activated selectively for certain emotions.

Interestingly, preliminary reports have indicated abnormal activation (decrease) of prefrontal and frontal areas in mood disorders (Bench et al., 1993, 1992; Dolan et al., 1992), which could imply that similar circuitry is involved in both perception and experience of emotion.

### 1.1.4 Hemispheric lateralisation

A great deal of research has implicated dominance of the right cerebral hemisphere (RH) in processing facial expressions. Left visual field (LVF) superiority for facial expression recognition has been repeatedly demonstrated (Strauss & Moscovitch, 1981; Ley & Bryden, 1979; Campbell, 1978), indicating involvement of RH due to contralateral projections. The right hemisphere does not appear to have sole monopoly over all face processing tasks though, with facial speech preferentially processed by the left hemisphere (Burt & Perrett, 1997). In addition, LVF superiority for facial expression processing could merely reflect the general involvement of RH in visual processing of faces.

Studies have also revealed the left hemiface (controlled by RH) to be both more intense and more animated in expression of emotion than the right side of the face (Borod et al., 1983; Sackeim & Gur, 1978). Indeed RH involvement in analysis of expression could be associated with RH production of expression. Gallese and Goldman (1998) found groups of cells - 'mirror neurons' - in monkey premotor

cortex that respond both during execution of a particular action by the monkey himself and also during the observation of the same action performed by another individual.

Investigation of intact and impaired abilities in individuals with unilateral brain damage has confirmed the importance of the right hemisphere in expression processing, with difficulties in both perception and expression of emotion apparent in patients with RH damage in contrast to relatively intact performance in LH damaged individuals (Adolphs et al., 1996; Borod et al., 1986; Etcoff, 1984; Cicone et al., 1980; DeKosky et al., 1980). Interestingly, in the Borod et al. (1986) study, recognition of negative emotions was more impaired in RH damaged subjects than recognition of positive emotions. Recognition errors also tended to be more inappropriate than those made by LH damaged or normal subjects, for example 'disgust' was misidentified as 'jerky', rather than 'anger'.

Several studies have also indicated contribution of the left hemisphere in emotion processing (Stone et al., 1996; Davidson & Sutton, 1995; George et al., 1993; Young et al., 1993). Relatively intact ability shown for expression perception tasks presented to the right visual field of split brain patients (Stone et al., 1996) suggests a role for the left hemisphere in processing expression, even if the majority of function is normally executed by the right hemisphere. Indeed, patients who exhibited the most severe impairment in expression perception in a study by Young et al. (1993) actually had unilateral lesions of the *left* (rather than right) hemisphere.

The possibility has been raised that the left hemisphere might play a role in processing positive emotion, with the right hemisphere controlling perception of negative emotions (Borod et al., 1997, 1986, 1981; Mandal et al., 1991; Reuter-Lorenz et al., 1981; Schwartz et al., 1976). The fact that depressive state has been

tentatively linked with left prefrontal dysfunction (Bench et al., 1993, 1992; Dolan et al., 1992) corroborates both the idea of the left hemisphere's role in processing positive emotions and Davidson and Sutton's (1995) hypothesis that a function of left prefrontal cortex might be to inhibit negative affect.

### 1.1.5 Discrete neural substrates for specific emotions

The belief that discrete neural substrates mediate perception of specific facial expressions receives some confirmation from functional imaging and neuropsychological research, following findings of circumscribed brain activation during perception of certain emotions and discoveries of selective deficits after brain damage.

Confirmation that discrete neural areas mediate perception of different emotions essentially requires the presence of a double dissociation, with the finding of a selective impairment for one emotion in spite of intact recognition of another and the opposite pattern, in individuals with different forms of brain damage. Such a double dissociation has been shown for recognition of fear and disgust in different patient groups.

Impaired recognition of fearful expressions resulting from amygdala damage has been well-documented (Calder et al., 1996a; Young et al., 1995; Adolphs et al., 1994). Patients' ability to recognise static photographs (of morphed images in the Calder et al., 1996a study) from the standard Ekman and Friesen facial affect series (1976) was examined and a differentially severe impairment in perception of fear was discovered, with recognition of the other 5 'basic' affects (Ekman, 1992) remaining virtually intact. (Recognition of anger was also affected but not to the same extent as fear.) Ability to discriminate gaze direction was also impaired.

Contrasting results were presented however by Hamann et al. (1996) who reported intact perception of fear in two patients with amygdalotomy sustained late in life. Adolphs et al. (1996) gave a possible explanation for the differential effect of amygdala damage. They believed that at an early stage in development, the human child establishes the association between others' expressions of fear and his/her own experience of that emotion, which requires integration of information from both neural systems coding the perceptual attributes of expressions and those enabling retrieval of previously stored knowledge about emotion. Adolphs et al. (1996) believe the amygdala and neocortical regions in the right hemisphere, respectively, might mediate these functions. If damage occurs early on, connections from the amygdala might not have had sufficient time to develop and thus impairment in fear recognition might result. Damage at a later stage will not have the same impact, as other structures can mediate this function.

This developmental hypothesis by Adolphs et al. (1996) is only one interpretation of course and it is perhaps worth noting that direction of emotional change and dominance showed considerable variation after amygdalectomy in monkeys. Although most monkeys became more submissive and avoided even affiliative interactions with conspecifics, a few became more hostile and aggressive and their dominance ranking actually increased in the social group. The variation in performance observed in humans following amygdala damage might therefore require further study.

Impairment in emotion processing in the amygdala has been demonstrated not to be restricted to the visual domain. In a study by Scott et al. (1997), a patient with bilateral amygdala damage was shown to exhibit problems in auditory recognition of fear, i.e., could not grasp the intonation of fear in a voice, even though

her hearing was normal. As with her deficit in visual perception of emotion, she also had additional moderate problems with recognition of angry voices.

Increased amygdala activation in response to facial expressions of fear has also been demonstrated in control subjects (Phillips et al., 1998, 1997; Morris et al., 1996; Breiter et al., 1996). Extent of response has been shown to be positively correlated with emotional intensity (Morris et al., 1996), although amygdala involvement is still detectable even when emotions are processed subliminally (Morris et al., 1997). Rapid habituation of response to fearful expressions has been shown to occur in the amygdala (Breiter et al., 1996), which could explain why some studies have failed to show amygdala activation for perception of fear (Sprengelmeyer et al., 1998).

A differentially severe deficit in recognition of disgust was found in patients with Huntington's disease (Sprengelmeyer et al., 1997a; 1996). Interestingly, experience of disgust appears to be altered in this patient group too and is perhaps reflected in clinical reports of poor hygiene standards. Although atrophy in Huntington's is usually diffuse, the most commonly implicated brain areas are basal ganglia and frontal regions, such as insular and orbitofrontal cortex. The involvement of such areas in perception of disgust is further suggested by a study of Huntington's disease gene carriers (Gray et al., 1997). Even though these individuals were presymptomatic, disgust recognition was still selectively impaired. The earliest signs of degeneration in Huntington's are in the basal ganglia, indicating that these structures might mediate disgust perception. Inability to identify disgust, in the absence of difficulty categorising other emotions, has also been observed in individuals with obsessive-compulsive disorder (OCD) by Sprengelmeyer et al. (1997b). Fronto-striatal pathology is recognised in OCD, which again provides

support that limbic and frontal areas might subserve processing of disgust-related stimuli.

Indeed, fMRI studies revealed enhanced metabolism in particular parts of the basal ganglia (the right putamen) and the right anterior insular cortex in response to disgusted, but not fearful or neutral, facial expressions in normal subjects (Phillips et al., 1998, 1997).

Blair et al. (1999) attempted to establish neural correlates for perception of sadness and anger using functional imaging techniques. Presentation of sad expressions produced activation in the left amygdala. The neural localisation of perception of sadness to the amygdala is consistent with finding of amygdala dysfunction in psychopaths (Patrick et al., 1994), who exhibit diminished autonomic response to expressions of sadness (Blair et al., 1997). Perception of angry expressions in contrast was associated with orbitofrontal activation. As described earlier, orbitofrontal cortex is believed to be involved in the regulation of behavioural extinction and reversal learning (Rolls, 1996; see section 1.1.2). Blair et al. (1999) noted that the expression of anger is often an attempt to "*...curtail the behaviour of others in situations where social rules or expectations have been violated*", which might explain orbitofrontal activation in response to anger. The fact that the right temporal pole and anterior cingulate cortex responded to both expressions suggests these areas play a more general role in expression processing (see George et al., 1993). A further study by Blair et al. (in press) has shown a selective impairment in recognition of angry expressions following administration of benzodiazepam in normal subjects. Benzodiazepam receptors are known to be concentrated in both the amygdala and frontal cortex (Dennis et al., 1988). As recognition of fear was not impaired following ingestion of diazepam, it seems likely

that the effect of the drug on individuals' ability to identify anger is mediated by frontal circuitry, as suggested by Blair et al.'s (1999) imaging study.

Results from functional imaging studies therefore suggest distinct neural substrates are involved in processing different emotions, i.e., fear, disgust and anger. The fact that a double dissociation has also been revealed in different patient groups for perception of fearful and disgusted expressions further confirms these findings.

## 1.2 ARE FACIAL EXPRESSIONS UNIVERSAL?

Alongside physiological research investigating neural substrates underlying processing of facial expressions of emotion, there has been a variety of studies examining psychological aspects of facial expression perception.

Few dispute the importance of facial signals in conveying emotional information acting to guide others responses and behaviour. Controversy remains though over whether particular facial configurations actually represent specific emotional states that are recognisable universally or whether they just enable inference with regard to how aroused or pleasant the expresser feels.

Darwin (1872) was one of the first to suggest that displays of emotion were 'hardwired'. He suggested that expressions had evolved for adaptive behavioural purposes and were therefore universal. Tomkins (1962) emulated this approach with the belief that facial expressions directly reflected an individual's internal emotional state and as certain fundamental emotions are innate, one could expect cross-cultural recognition of the corresponding facial expressions.

In support of the innate nature of facial expressions, developmental studies have revealed that infants can imitate different facial expressions at a very early age (Field et al., 1982). Differentiation between positive and negative expressions has

been shown at around 3 months old and by 7 months infants are able to distinguish between different negative expressions (Nelson, 1987), suggesting that to some extent at least humans are biologically prepared for emotion awareness. Further evidence that emotional expressions are unlearned comes from their presence in blind and deaf infants who have little opportunity to learn from social reinforcement (Eibl-Eibesfelt, 1970a). Of course such findings do not preclude the occurrence of social learning in emotion comprehension and indeed social referencing, for example learning not to eat foods that a caregiver expresses disgust towards, must occur for complete understanding of some emotions (Campos & Stenberg, 1981).

Cross-cultural research into recognition of facial expressions has been predominately carried out by Ekman and Izard, who are both of the view that there is a set of 'basic' facial expressions that can be categorised universally. In addition to research in Western civilisations, ethnological studies on preliterate cultures have demonstrated that facial expressions of *anger*, *disgust*, *fear*, *happiness*, *sadness*, and to some extent *surprise* can be understood and represent similar underlying emotional states across all populations, even those isolated from media input (Ekman, 1992; Ekman & Friesen, 1971; Ekman et al., 1987; Izard, 1994, 1992).

The fact that some facial expressions are not under voluntary control, such as the 'Duchenne' smile, where the contraction of the *orbicularis oculi* muscle produces wrinkling around the eyes in addition to a smile, suggests that expressions *do* relate to internal emotional states (such as happiness) and can reflect subconscious physiological processes. This is not to suggest that expressions cannot be produced voluntarily too, for example polite smiles in acknowledgement of another person's speech in conversation.

Instead of each expression conveying a specific emotional state, Russell (1980) proposed that an expression reflected the degree of pleasantness or arousal experienced by an individual. His model of affect encapsulated two orthogonal dimensions (pleasant-unpleasant and degree of arousal) with each expression representing a point along these two continua. The fact that selective deficits have been discovered in patients with circumscribed brain damage (see section 1.1.5) does however indicate that expressions do relate to specific emotions.

Further evidence that facial expressions of emotion are interpreted as discrete concepts is provided by psychological work on categorical perception. Categorical perception is a psychophysical phenomenon whereby it has been demonstrated that it is perceptually easier to discriminate between two stimuli in different categories than between two exemplars of the same category, even though the actual physical differences between the two sets of stimuli are equivalent. For example, changes in colour are perceived abruptly across gradual alterations in wavelength. If facial expressions of emotion are recognised along different dimensions of pleasantness and arousal as Russell (1980) has proposed, then categorical perception should not occur for facial expressions and discrimination between two stimuli straddling a so-called category boundary would be no easier than distinguishing between stimuli supposedly within a category. For example, anger and fear are in close spatial proximity in Russell's circumplex model, since they both represent a high degree of arousal and are perceived as fairly unpleasant, and should therefore not be segregated into distinct categories.

However, evidence from studies of categorical perception of facial expressions of emotion has demonstrated that individuals *do* separate facial expressions into distinct categories, which appear to equate with Ekman's 'basic'

emotions, with discrimination *across* categories easier than within (Young et al., 1997; Calder et al., 1996b; Etcoff & Magee, 1992). (It should be noted though that individuals remain able to distinguish between minor changes within a category.) Individuals are extremely sensitive to changes across a category boundary, although there is a trade-off with processing speed as reaction time increases the further away an exemplar is from the prototype (Young et al., 1997).

To examine the phenomenon of categorical perception, Calder et al. (1996b) pioneered a sophisticated method whereby facial expressions of varying emotional intensity were used as stimuli. These interpolated expression images are known as 'morphs'. Etcoff and Magee (1992) were first to use this morphing method, based on an algorithm formulated by Brennan (1985), using line drawings of facial expressions. Morphed images are generated by calculation of the difference in spatial position between corresponding feature points on the two end-point images (e.g., difference in the position between the corner of the left eye on a happy and on a surprised expression). A percentage of this vector difference is then applied to one of the end-point images, which could result in a 30% happy and 70% surprised expression morph, for example.

Although useful in itself, use of 100% veridical expression images in tests of recognition cannot reveal the presence of more subtle deficits in expression perception, such as the percentage of intensity required for detection of an expression. It is this measure of sensitivity that is of predominant interest in this thesis, in examination of possible alterations in facial expression processing in different psychiatric patient groups.

## 1.3 RATIONALE FOR METHODOLGY

The method used here is an extension of the morphing paradigm advanced by Calder et al. (1996b). Instead of presenting static images of morphed expressions, a computer-generated program, developed from algorithms designed by Benson and Perrett (1991), was devised to enable real-time interactive morphing between two endpoint expression images (one of which is always *neutral*) of the same identity. In this respect, the subject views the change in expression like a gradual movie and is then able to manipulate the intensity of the expression to the desired point at which he/she can first reliably identify the emotion, which might correspond to his/her category boundary between expressions. This measure is an attempt to classify individuals' sensitivity threshold to different facial expressions of emotion.

Essentially this method follows the method of adjustment rather than the more intricate psychophysics staircase threshold paradigm (Wetherill & Levitt, 1965). Advantages of this method over the traditional threshold design include reduction in test duration (as fewer trials are required) and the use of more realistic dynamic stimuli (see Eibl-Eibesfelt (1970b) for advantages of dynamic expression images). Psychophysical investigations reveal a cost-benefit trade-off at category boundaries between reaction time and discrimination (see section 1.2). The purpose of this thesis, however, is *not* to study the nature of the cognitive processes underlying categorical perception but to examine any changes in the point at which a category boundary is perceived across different groups of individuals.

### 1.3.1 Forced-choice paradigm

In accordance with Ekman's findings, the facial expressions used in this thesis are the 6 'basic' affects of *anger*, *disgust*, *fear*, *happiness*, *sadness* and

*surprise* (in addition to *neutral*), which are believed to be universally recognisable. Recognition accuracy was assessed in addition to sensitivity level and for this a 6-way forced-choice procedure was used, with subjects required to choose which of 6 emotion labels best described the stimulus displayed. There has been criticism over whether forced-choice methods are valid (Russell, 1994), in the respect that an individual might choose the correct response by default even if not convinced the facial expression suitably reflects that emotion. For example, *sadness* might be correctly selected only because the expression does not suitably fit any of the other categories.

However, the decision to use the forced-choice paradigm in the test of facial expression perception described in this thesis is defensible in several ways. First, the stimuli employed here are taken from the Ekman and Friesen (1976) stimuli set of Pictures of Facial Affect, which have been repeatedly used and demonstrated to be well-recognisable cross-culturally. Second, as Ekman himself has reported, even when subjects are given free choice in classification of different facial expressions, they tend to use the labels according to these 6 standard emotions. Ekman's (1994) rationale for using the forced-choice procedure is that, as well as being a great deal simpler than an open-ended design, the labels used act as an 'abbreviation' *"...that refers to any of a number of different aspects of an emotion – including antecedents, expressions, memories, and consequences"*. In this respect, the label '*disgust*' could equally refer to feeling nauseated, repulsed or could cause the observer of the corresponding expression to assume the expresser had just smelt or tasted something offensive, for example, but the underlying *sense* of the expression would still be understood by different perceivers.

Ekman (1994) also comments that in spite of all the problems associated with this method, studies have still revealed a high degree of accuracy in emotion recognition cross-culturally and even if errors are made, the majority tend to be made in the same direction, e.g., anger misidentified as disgust. This still demonstrates that certain facial expressions are universally recognisable, which is the most important issue, even if the specific verbal labels used are not the best options. The precise labels used are in a sense fairly irrelevant.

## 1.4 EXPERIENCE AND PERCEPTION OF EMOTION

There are several reasons why psychiatric groups – namely depressed and eating disorder patients – were selected as the subject populations of interest in this thesis. Firstly, social problems are well noted in both these conditions and could in part be a consequence of incorrect appraisal of facial expression cues. Misinterpretation of expression (and therefore intention) could cause inappropriate responding in interactions and might ultimately lead to social isolation, with other individuals avoiding future contact.

Secondly, *experience* of emotion is known to be altered in both these conditions and one would assume that if emotional state is altered, so *perception* of emotion might also be affected. Affective disorders, such as depression, are of particular interest in the investigation of facial affect perception as they involve, by definition, alteration of affect or emotional state in the sufferer. Indeed, Hornak et al. (1996) observed a close relationship between altered subjective emotional state and impaired perception of facial expressions in patients with ventral frontal damage (see section 1.1.2). In addition, Huntington's Disease sufferers are reported to have poor personal hygiene standards and are also shown to be unable to recognise facial

expressions of disgust, implicating a general emotional impairment with disgust (Sprengelmeyer et al., 1996). These studies also indicate that similar neural structures might subserve processing of both experiential and perceptual aspects of an emotion.

Finally, sensitivity to different facial expressions has not yet been systematically examined in either of these patient groups. There have been several studies investigating recognition of different facial expressions in depression but results to-date have been inconclusive and methodological flaws have been apparent (see Chapter 3). Facial expression perception does not appear to have been examined at all in patients with eating disorders. The development of this new interactive testing method will enable elucidation of not only the presence of any severe impairment in expression recognition but will also allow examination of more subtle differences in *interpretation* of others facial expressions. Results should inform whether or not these patients' social difficulties could be linked to how they perceive facial expressions of affect in other individuals.

# CHAPTER 2: DEPRESSION

Before examining the results of the current studies of facial expression perception in individuals suffering from different degrees of depressive state, this chapter aims to provide a general overview into the condition of depression, describing symptomatology, putative causal factors and then focusing in greater detail on interpersonal difficulties experienced by depressives.

## 2.1 INTRODUCTION

Depression is an affective disorder, which by definition indicates that sufferers have altered experience of mood. An example of symptom constellations endured by depressed individuals would be: negative mood state, loss of interest, apathy, anhedonia and feelings of guilt and anxiety. In addition, there are often associated insidious somatic symptoms, for example disturbed sleeping and eating patterns. Low self-esteem and lack of motivation (i.e., difficulties with response initiation) are also prominent features of this debilitating condition. Depressed individuals tend also to experience problems in the social sphere – for example, possessing few social supports and having difficulty interacting with others, resulting in many becoming withdrawn and isolated.

A distinction has been made (Mendels & Cochran, 1968) between two different types of depression – endogenous (biological) and exogenous (psychological). The latter is believed to be precipitated by a major life event or stressor and is usually not as chronic or severe as the former, which is thought to be genetic. Treatment patterns are also different, with antidepressants considered more effective for endogenous types and therapy more expedient for exogenous

depression. However, such a distinction has been brought into question with incidence of depression in relatives of individuals diagnosed as suffering from endogenous depression being no greater than in those with the exogenous form (Andreasen et al., 1986). It is now therefore believed that the distinction might relate more closely to the continuum of mild (exogenous) to severe (endogenous) forms of depression.

Depression is a fairly prevalent condition - an estimated 5 percent of the general population being affected in any one year (Myers et al., 1984) and about a 10 percent chance for any one individual to experience an episode of clinical depression during his or her lifetime (Robins et al., 1984). In general, prognosis is relatively poor for depression, with only around one in five patients experiencing full recovery. About the same proportion will however go on to develop a more chronic depressive condition. Depression appears to be more prevalent in females than males (about a 2:1 ratio, Nolen-Hoeksema & Girgus, 1994) with approximately 1 in 8 women being afflicted every year, of which a substantial proportion of cases are chronic (i.e., have a duration of more than one year), Brown (1996). This sex difference might be in part accounted for by decreased symptom reporting in males due to lack of conformity with traditional male stereotypes (Hammen & Peters, 1978; see section 5.5). Vulnerability to premenstrual tension (of which depression is a major symptom) might also augment the likelihood of depression incidence in females.

In addition to the suffering of the individual, the annual cost to the Welfare State with regards to workforce depletion and treatment is significant. Continued research into depression in relation to causes and contributing factors is therefore vital, particularly as knowledge to-date remains fairly limited.

## 2.2 PHARMACOLOGY

5-HT (serotonin) dysfunction has been implicated as a biological basis for depression. The normal growth hormone response to an intravenous infusion of *tryptophan*[1] is considerably reduced in depressed individuals, implying serotonergic impairment since 5-HT antagonists block this response (Deakin et al., 1990). Antidepressant drugs (commonly known as SSRI's - *Selective Serotonin Reuptake Inhibitors*) affect 5-HT neurotransmission by inhibiting reuptake of 5-HT at synapses and therefore increasing the amount of 5-HT in the blood stream. Impairment to 5-HT function is also *state-dependent* in depression, i.e., functioning normalises with clinical recovery.

Deakin (1996) suggests the impairment in 5-HT function in depression could be secondary to abnormal production of cortisol, as patients with an attenuated growth hormone response to tryptophan have greater concentrations of cortisol. He incorporated this into a theory of a possible chain of events which give rise to clinical depression: with *psychosocial adversity* (defined as stressful social situations, i.e., social isolation, difficulties at home) resulting in *increased cortisol concentrations*, which in turn cause *impairment of serotonergic function*, culminating in *depressed affect*. Vulnerability to psychosocial adversity appears to be mediated by self-esteem (Brown & Harris, 1986). As yet, evidence for the link between psychosocial adversity and hypercortisolemia is preliminary, although depressed patients appear to have raised cortisol levels following the experience of a stressful life event (Dolan et al., 1985).

Aggressiveness has been linked with low concentrations of serotonin in cerebral spinal fluid (CSF) in both monkeys and humans (Brown & Linnoila, 1990;

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[1] Tryptophan is a precusor for serotonin.

Raleigh et al., 1986, 1980). Individuals with diminished CSF serotonin levels become overly defensive, irritable and withdrawn in social situations. When depressed patients are treated with the SSRI Prozac (*fluxotine hydrochloride*) they are clinically observed to become much more 'easy-going' and feel more relaxed in social interactions. Similar effects are seen in monkeys treated with serotonin agonists, i.e., increased instances of affiliative behaviour and decreased episodes of destructive aggression. Low levels of the serotonin metabolite 5-HIAA have also been associated with suicidal behaviour in humans (for a review see Brown & Linnoila, 1990).

In addition to knowledge accumulation concerning the pharmacological underpinnings of depression, it is also important to examine psychological aspects of depression to enable a more complete understanding of the condition. An area of particular significance is investigation of the nature of cognitive processing in depression and its possible effects on everyday behaviour and perception.

## 2.3 COGNITIVE THEORIES

There has long been debate as to the role of cognition in emotion processing. Initially extreme views were postulated by Zajonc (1980) and Lazarus (1982); the former asserting cognition had *no* influence on 'automatic' processing occurring in emotion, whereas the latter believed that *all* emotional processes were affected by cognitive mechanisms. It is now widely held that cognitive appraisals *do* play a role in emotion, but these include automatic, non-conscious processes in addition to strategic, intentional ones (for review see Mathews & MacLeod, 1994).

Conscious appraisals of emotional information seem important in depression. Depressives show a cognitive bias in recall of emotionally negative information.

When exposed to sets of emotionally valenced (positive and negative) and neutral stimuli and later unexpectedly asked to recognise these stimuli from distractor items, depressed subjects show biased recall of negative information. However, this effect is influenced by the initial nature of encoding, i.e., preferential recall *only* occurs for negative words that are processed in relation to the perceiver (Denny & Hunt, 1992). In addition, selective processing only appears to occur for stimuli related specifically to depression (Watkins et al., 1992) as opposed to all negatively valenced stimuli (for example, words relating to physical threat). A negative self-schema could therefore be operating in depressives' cognitive processing, resulting in the tendency for them to preferentially process negative emotional information related to themselves, but not negative information in general. Biased recall of negatively valenced information has also been elicited in normal subjects following depressed mood-induction (Perrig & Perrig, 1988; Teasdale & Russell, 1983), implying that cognitive bias in depression cannot purely be accounted for by automatic processes, as recall is not dependent on mood state.

Further evidence that selective encoding of negatively valenced information is not automatic in depression (unlike anxiety) is provided by studies using interference paradigms. An interference paradigm is when a subject is required to perform a certain cognitive task while trying to ignore salient information. Mogg et al. (1993) presented depressed subjects with a modified version of the Stroop (1938) task, in which subjects were required to name, as quickly as possible, the colours in which words on a sheet were presented, whilst simultaneously ignoring the meaning of the words. Words were presented very briefly and followed by a pattern mask obscuring the stimulus, preventing conscious detection of the word. Depressives did not exhibit delayed colour-naming latency for subliminally presented negatively

valenced words. The bias does *not* operate outside of awareness therefore, suggesting the involvement of more intentional processes. Rather than invariant, automatic effects being present in depression then, it appears that alterations in normal information processing are characterised by preferential recall of self-rated negative information occurring at a more elaborate, interpretative stage of processing.

Several prominent theories attempt to account for these differences in cognitive processing in depressed compared to non-depressed individuals. A summary of each is given in the following three sections.

### 2.3.1 Cognitive distortions

Probably the most influential theory regarding cognitive processing in depression is that proposed by Beck in 1967. Beck's 'self-blame' model was essentially derived from his therapeutic experience with depressed patients. He suggested three cognitive mechanisms through which depression could arise - the cognitive triad, schemata and cognitive distortions.

The cognitive triad involves 3 aspects of negative thinking believed to be characteristic of depressives – negative thoughts about oneself, one's current situation and the future. Depressed individuals tend to regard most experiences as negative, even when equally plausible positive interpretations are available. In Beck's experience, depressives blame such apparently negative outcomes on their own lack of ability and worthlessness. The belief that all future occurrences will be similarly aversive as a consequence of their own personal defects is immutable.

Schemas are '*stable, organised representations of past experience*' and are used by individuals to analyse and interpret incoming stimuli. These schemas

become dysfunctional in depressives, whereby negative conclusions are drawn from a situation when more appropriate positive, or at least neutral, interpretation(s) are present.

Similarly, distorted thought processes and logical errors systematically arise. Depressives frequently overgeneralise, with one small oversight on their behalf making them draw conclusions as to their general inadequacy and worthlessness as a person. Likewise they will selectively focus on the negative impact of a minor criticism while ignoring any positive feedback from others. The tendency to feel personal responsibility for bad outcomes (when in fact the depressed individual has no bearing on the event whatsoever) is also common. Some researchers have noted, however, that depressed individuals negative self-perceptions can be accurate whereas non-depressed individuals can exhibit inappropriate self-enhancing perceptions (see section 2.3.3).

The practice of cognitive therapy (Beck et al., 1979) stemmed from Beck's (1967, 1987) theory. Cognitive therapy aims to identify and correct the distorted thought processes characteristic of depression and encourage individuals to attempt situations which they believe are outwith their ability. In addition to countering negative thoughts and feelings, behavioural techniques such as assertiveness training and increasing activity levels are taught in treatment. Cognitive therapy (in connection with behaviour therapy and often in combination with drug treatments) has generally been highly successful in helping the recovery process in depression (Teasdale et al., 1984; Kovacs et al., 1981; Rush et al., 1977) and reducing likelihood of remission (Evans et al., 1992; Blackburn et al., 1986; Simons et al., 1985).

### 2.3.2 Learned helplessness

Around the same time as the emergence of Beck's theory, Seligman (1974) defined the theory of learned helplessness in depression. He believed that depressives viewed events as uncontrollable and therefore they felt they would be unable to avoid similar negative consequences in the future. The basis for this theory came from experimental evidence from animals. Motivational deficits were induced in dogs following prior experience of uncontrollable random presentation of shock (for review see Rosenhan & Seligman, 1989). When later able to escape receiving shock, the dogs became passive and made no attempt to flee. The authors attributed this behaviour to the previous uncontrollable experience with shock, with voluntary escape responses not being initiated as they had previously been unsuccessful. Similar patterns of behaviour were observed in humans not only with loud noise as an aversive uncontrollable stimulus but also using unsolvable cognitive tests (Hiroto & Seligman, 1975; Roth & Kubal, 1975). The uncontrollable presentation of an aversive stimulus causes an individual to lose the contingency relationship between voluntary response and outcome (avoidance of aversive stimulus) and so in future presentations of such a stimulus they are not motivated to respond. In the case of depressives, this helplessness is more pervasive and generalises across many different situations.

A study conducted by Abramson et al. (1978a) examined the role of expectancy on learned helplessness in depressives. Performance on tasks where outcome was related to either skill or chance did not affect depressed individuals' expectation of their future ability in similar tasks. For example, if they succeeded on a task of skill, their expectation that they would succeed on a future task did not change. In addition, their expectation regarding future performance also remained

unchanged if they failed. In contrast, outcome on skill related tasks (but not tasks governed by chance) *did* affect the future expectancies of both non-depressed subjects and schizophrenic patients, with successful task completion being related to greater confidence in future achievement and failure resulting in lowered expectancies. Again it would appear as though depressed individuals have lost the idea of contingency between their own behaviour and the outcome of any situation.

More recently Benassi et al. (1988) conducted a meta-analysis of studies examining the relationship between locus of control and depression. The concept of locus of control relates to the beliefs an individual holds regarding the controllability of different outcomes in his/her life. For example, if an individual believes outcomes to be beyond his/her personal control, he/she is said to have an external locus of control, whereas internal locus of control relates to the belief that situational outcomes are within one's control. Benassi et al.'s (1988) analysis found support for the learned helplessness model, i.e., that attributions concerning an external locus of control are commonly formed in depression.

However a paradox is apparent between Seligman's theory of learned helplessness in depression and Beck's theory of distorted perception. The idea of assuming no control over events and therefore feeling helpless does not equate to the self-blame for aversive events commented on by Beck.

Abramson et al. (1978b) partially addressed this issue by reformulating the learned helplessness theory to include *attributions* as a mediating mechanism by which lack of control could result in failure to learn response-outcome contingencies in turn affecting future perceptions. The type of attributions made following either success or failure is crucial and affects how future situations will be construed.

Abramson et al. determined three dimensions along which attributions can function: *internal-external*, *stable-unstable* and *global-specific*. The *internal-external* dimension refers to whether individuals believe failure or success of events to be due to their own ability (or lack of) or due to the situational constraints themselves. The degree of *stability* attributed to an event is also important, i.e., is the cause of such a positive or negative outcome likely to be permanent or transient and therefore will it persist on future occasions. *Global* or *specific* attributions affect whether or not an individual believes the cause of the current failure or success is due to a characteristic that is likely to produce the same outcome in only a small number of similar situations or across a wide range of different occasions.

Depressed individuals are believed to attribute negative events to internal, stable and global causes but successful outcomes to external, unstable and specific factors, leading to the persistence of depressive symptoms. The lack of contingency anticipated between their response and the outcome of a situation results in a pervasive and lasting feeling of helplessness.

### 2.3.3 Depressive-realism

Alloy and Abramson (1979) suggested another theory that contrasted considerably from Beck's view of cognitive processing in depression. Instead of depressed individuals having distorted ideas and views about themselves and their environment, Alloy and Abramson concluded from their study that depressives were in fact very astute in their perceptions and held quite realistic views of their own abilities. Non-depressed individuals were conversely 'at fault' for exaggerating their own potential.

They asked subjects to judge the amount of contingency between their response (button pressing) and the appearance of a green light. Degree of control was varied across several groups from no control to 75% control (i.e., 75% contingency between button pressing and the light appearing). Subjects who scored higher for depressive symptomatology were found to be more accurate at judging the correct level of contingency between their response and the outcome (regardless of actual level of control) than were non-depressed subjects who tended to overestimate the amount of control they had, particularly in the condition of no control.

Similar findings were reported in a study by Lewinsohn et al. (1980) investigating how depressed and non-depressed individuals rated their own and other's social skill in group interactions. The authors wanted to determine whether depressives rated themselves more negatively than others judged them or whether their negative self-perception was in fact quite accurate and reflected their actual social ability with respect to other individuals' perceptions. Self-ratings in addition to observer-ratings (both from other individuals in the interaction and from independent onlookers) were collected from depressed patients, non-depressed psychiatric patients and controls following group interactions in an attempt to address whether the negative self-perceptions present in depression are illusory or not. Although depressed individuals rated themselves as less socially skilful than non-depressed subjects did, there was much less discrepancy between their self-ratings and others-ratings than with non-depressed individuals, who tended to overrate their social ability.

These findings imply that depressives can be fairly *realistic* in their appraisal of their own social ability (which both they and others view as not particularly competent), whereas non-depressed individuals tend to view themselves in a more

positive light than is actually perceived by others. Such self-enhancement might act as an emotion regulation process in normal individuals in that attention is concentrated on positive, as opposed to negative, aspects of one's own personality and perhaps the personality of others, in turn helping a positive emotional state to be maintained. The concept that unrealistic self-perception is a crucial factor in depression must therefore be seriously questioned following such results.

More recent findings suggest that the idea of depressive-realism needs to be slightly modified. Wood et al. (1998) demonstrated that depressed patients lack confidence in their responses – particularly when they are correct. The authors examined face recognition memory in both depressed patients and non-depressed controls and also ratings of confidence subjects gave themselves in each response. Although both groups performed equally well on the task, depressed individuals were much less confident in their responses than control subjects when they were actually correct but expressed the same degree of confidence as controls when they were wrong. These results do not support the idea of depressive-realism in its extreme form. Instead these results suggest that although depressives are quite accurate at identifying when they are incorrect, they are not so proficient at appraising when they are right. Wood et al. also looked to see whether these effects were still present in a subclinical population (i.e., individuals who are suffering some degree of depressive affect but who have not been clinically diagnosed), by comparing performance of dysphoric and non-dysphoric students. (Dysphoria was assessed according to score on the Beck Depression Inventory[2]; Beck et al., 1961.) However, here no differences in self-confidence ratings were found between the two

[2] See Appendix 4.1 and section 4.2.2 for further description of the Beck Depression Inventory (BDI)

groups leading the authors to caution the use of subclinical populations in the formulation of models of clinical depression (see section 4.7.1).

### 2.3.4 Are negative cognitions a causal factor for depression?

Lewinsohn et al. (1981) emphasize that although there is evidence of the presence of negative cognitions in depression, it is not clear *which direction* the causal relationship between negative cognitions and depression takes.

They conducted a longitudinal study in a large sample (around one thousand participants) from the general population in an attempt to examine the direction of causality more systematically. Various cognitive measures and depression status were assessed at time 1 and then the presence/absence of depression was re-evaluated around 8 months later (time 2). Results demonstrated that the cognitive style of individuals who became depressed by time 2, but who were not classed as depressed at time 1, did not differ from that of subjects who remained non-depressed for the course of the study.

Such patterns of negative thinking (known to be associated with depressive state) do not therefore appear to act as a causal factor for depression. The idea that negative cognitions are a *consequence* (rather than an antecedent) of depression was instead corroborated by the data. Indeed, such 'depressive thinking styles' seem to improve with recovery, in that individuals who (although they were not currently depressed) had reported suffering from previous episodes of depression were shown not to differ in their cognitions from individuals who had never been depressed. However, individuals who reported more negative cognitions concomitant with depression were more likely to have a poorer outcome than those reporting fewer depression-related cognitions. Before the role of negative cognitions in depression

can be conclusively established, a further replication of such a study using a similar prospective, longitudinal design would be required.

### 2.3.5 Critical review of evidence for cognitive theories

Coyne and Gotlib (1983) criticised the extensive reliance on cognitive models as an explanation of psychological aspects of depression. They presented a review of the evidence both supporting and refuting the two major cognitive models (outlined in sections 2.3.1 and 2.3.2).

For example, a criticism is made of the evidence so frequently cited in support of Beck's cognitive model that depressed individuals' hold distorted negative views of their own ability. Other research has indicated both that such lowered self-evaluations are not necessarily specific to depression and indeed also that such negative self-perceptions might indeed not be distortions but in fact reality (see Coyne & Gotlib, 1983 and section 2.3.3 for references).

The authors also comment on the literature related to contingency learning in depression. Although the learned helplessness model (see section 2.3.2) posits that depressives believe their response and the outcome of a situation are independent, there is also evidence to suggest that depressives are in fact more able to judge contingency relationships than non-depressed individuals (Alloy & Abramson, 1979; see section 2.3.3). In addition, Coyne and Gotlib note that contingency judgements are not commonly made in daily life and results indicating lack of perceived contingency in depressives could instead perhaps be a consequence of experimental instruction.

Evidence supporting preferential recall of negatively valenced information in depression is also equivocal. Many of the studies conducted have used fairly

contrived laboratory tests and also feedback given by the experimenter with regards to performance (for example) is not necessarily accurate and might be discrepant with the participants own belief of his/her performance - both of which could affect results.

Similarly, evaluation of the available evidence on attribution processes in depression has not by any means conclusively supported the theory that depressives attribute failure to internal, stable and global factors. An additional problem of most of the work in this area is that either unrealistic laboratory situations or hypothetical situations have been used as the basis on which individuals must form attributions. For an accurate understanding of the attribution process, an attempt must be made to use situations that are currently stressful to the participant that will therefore possess much greater salience for the individual.

Coyne and Gotlib (1983) stress that greater focus on external, environmental factors surrounding cognitive processing in depression is required, rather than sole concentration on internal thought-related factors.

## 2.4 INTERPERSONAL ASPECT

A seminal paper by Coyne in 1976 highlighted the importance of the *interpersonal* aspect of depression, indicating that depressive state is likely to be perpetuated by aversive social interactions between depressives and other individuals.

He put forward the idea that a self-perpetuating cycle could arise in depressed individuals' interactions. Negative cognitions experienced by the depressed individual would result in him/her seeking excessive amounts of support and reassurance, which in turn might lead to others to respond aversively towards the

depressive and ultimately reject him/her, which would merely serve to aggravate the depressive's negative self-evaluation and could result in him/her withdrawing from future interactions. In sum, depressives seem to create (by their interpersonal behaviour) the very depressogenic environment they are trying to avoid (i.e., negative reactions from others and unfavorable evaluations from them) which in turn helps perpetuate their depressive state.

A scale to assess interpersonal sensitivity in depressives - the Interpersonal Sensitivity Measure (IPSM; devised by Boyce & Parker, 1989) - was used in a study by Boyce et al. (1992) in an attempt to predict the effect of this trait on depression course. Factors already known to predict poor outcome are lack of social support (Krantz & Moos, 1988) and high levels of neuroticism.

The IPSM attempts to tap personality characteristics relating to depression along several dimensions, entitled *interpersonal awareness*, *need for approval*, *separation anxiety*, *timidity* and *fragile inner-self*, all of which relate to interactional skills. High scores on the IPSM (taken 20 weeks after the initial assessment) were found to relate to poor outcome at 52 weeks. Scores on the subscales *interpersonal awareness*, *separation anxiety* and *fragile inner-self* were particularly predictive of one-year depression course. Depressive state is liable to be maintained by increased awareness of and sensitivity to negative responses from others, e.g., enhanced sensitivity to angry expressions. Indeed depressives tend to perceive others as less friendly towards them than do controls (Hoehn-Hyde et al., 1982). In addition, reluctance to speak about oneself for fear of ridicule (as assessed by the *fragile inner-self* subscale) could present a difficulty for depressives, particularly in therapeutic settings when available help cannot be properly exploited. A prospective

study would however be needed to examine more clearly the relative causal influence of vulnerability factors on outcome.

### 2.4.1 How do depressives respond to others?

Coyne's (1976a) influential paper stresses the crucial role of such interpersonal behaviour in the maintenance of depression. He believes that how other people react and respond to depressives affects how the depressed individual perceives him/herself and consequently whether or not social isolation and in turn exacerbation of depressive affect might ensue.

Several studies have examined behaviour during depressed individuals' interactions and produced varying results. For example, Youngren and Lewinsohn (1980) examined both subjective self-reports and observer ratings of depressed patients' interpersonal skills in experimentally constructed dyadic and group situations. Although there were no *observable* differences in their verbal and non-verbal behaviour compared to controls, the depressed participants rated themselves as feeling less comfortable, being less assertive and both giving and receiving fewer positive responses than their non-depressed partners or group members. The depressed individuals were noted to speak more softly and engage in less eye contact than controls but, as non-depressed psychiatric participants exhibited similar behaviours, this effect was unlikely to be due to depression alone. Depressives also held a more negative perspective of their own ability in social situations. In addition, both independent observers and other (non-depressed) participants rated the interpersonal skills of the depressed individuals fairly negatively, but this was only significant for the group situation.

There were several problematic methodological aspects to this study – most notably the fact that the interactions were contrived and not necessary realistic to everyday situations. Previous research in more naturalistic settings (Libet & Lewinsohn, 1973) has also indicated social skill deficits in depressed individuals though. It would appear that depressed individuals *do* experience social difficulties, which in turn seems to induce negative reactions from others.

Another study examined differences between depressed and non-depressed individuals' behaviour according to response style enacted by another individual in an experimentally contrived dyadic interaction (Blumberg & Hokanson, 1983). Confederates were instructed before the interaction to act in one of three ways - in a *critical-competitive*, *supportive-co-operative*, or *helpless-dependent* manner. (Subjects were obviously unaware that the other individual in the interaction was a confederate and believed them to be a fellow participant.) University students comprised the subject sample and individuals were classed as depressed or non-depressed according to their scores on the Beck Depression Inventory (BDI). On conversational measures, depressed subjects' speech was reported to have more negative content and less neutral content (i.e., the subjects were less skilful at maintaining conversation, for example filling gaps with inconsequential comments or platitudes) than that of non-depressed subjects. Although there were few significant differences between depressed and non-depressed subjects' evaluation of the other individual depending on the type of role he/she portrayed, there were patterns in the data suggesting that depressives might respond differently in various interactions depending on situational constraints. For example, depressed subjects were more disparaging and became more irritated when conversing with the confederate enacting the *critical-competitive* role than non-depressed participants.

Conversely, when interacting with the *helpless-dependent* confederate (who appears vulnerable and submissive), depressed subjects were also more self-devaluating. Use of a clinical (rather than subclinical) sample and a more naturalistic situation might help extend these findings.

Related studies have demonstrated that the power role assumed in an interaction can play an important modulating role on depressives' interpersonal behaviour. Depressed individuals display more passive and helpless behaviour when in low power roles compared to non-depressed individuals and more exploitative and aggressive behaviour when in high power roles (for example, Hokanson et al., 1980).

Following telephone conversations with female depressed patients, non-depressed female students were shown to feel significantly more depressed, anxious and hostile than non-depressed students who had interacted with either control subjects or non-depressed psychiatric patients (Coyne, 1976b). The manner by which such behaviour is elicited in others remains unclear however, due to little obvious observed verbal or non-verbal differences in depressed and non-depressed individuals social behaviour. It is possible that increased conversational focus on the depressed individual and associated personal information, which was demonstrated in Coyne's (1976b) study, might be viewed by others as inappropriate and might contribute to induction of negative mood. Coyne believes that depressed affect could be perpetuated in such situations due to the apparent discrepancy between statements of support and reassurance given by others and the rejecting behaviour displayed towards the depressed person. The more rejected and insecure a depressed individual feels, the more probable he/she will desperately seek some support, resulting in a vicious circle of depressive cognition and problematic social interactions.

Rather than seeking favourable feedback (i.e., support and reassurance), Giesler et al. (1996) believe that depressives actually solicit negative evaluation from others in that it equates to their own self-perception and allows security in the knowledge that their own appraisals are realistic. In their study, depressed individuals chose to receive negative feedback about themselves in preference to positive feedback, whereas non-depressed individuals with high self-esteem chose positive feedback and those with low self-esteem (also non-depressed) selected both positive and negative feedback. Giesler et al. (1996) comment on research demonstrating that individuals tend to seek information that confirms their own self-perceptions and in this respect the depressives can be said to be engaging in normal behaviour here. Unfortunately, the upshot of such behaviour is that it will merely serve to sustain the depressive episode by ingraining such negative self-views in the depressed individual's psyche.

### 2.4.2 Responses of others to depressives

Interestingly, Gotlib and Robinson (1982) found differences in how *other people* responded to depressed individuals during dyadic interactions. In their study, individuals who interacted with depressed participants subsequently felt no more depressed and stated they were no less likely to engage in future social contact with that particular individual than did subjects who conversed with non-depressed individuals. However, videotaped recordings of the interactions revealed that individuals who had conversed with a depressed subject had engaged in more negative behaviour (both verbal and non-verbal): their contribution to the conversation had contained more negative content; they had made fewer supportive comments; they had smiled more infrequently and had facially appeared less aroused

and involved. All the subjects in this study were female students and subjects were classed as 'depressed' if they scored 9 or more on the BDI. The generalisation of these results to clinical depression (as opposed to general psychopathology) and to mixed-sex interactions therefore remains unclear. Nonetheless, if *even* 'mildly depressed' students can induce such a noticeable behaviour change in others, it logically appears *more* - rather than less - likely that a similar aversive response would be elicited in others by clinically depressed individuals, although this is still to be systematically elucidated. Unlike the Youngren and Lewinsohn (1980) study, the behaviour of the depressed subject was not rated here, so any clues as to the potential causes of such response modification cannot be gleaned.

Howes and Hokanson (1979) also studied individuals' responses depending on whether they were interacting with confederates portraying 'depressed', 'physically ill' or 'normal' roles. Individuals were observed to express more direct negative comments and engage in more silence when conversing with a 'depressed' partner. The 'depressed' confederates were also rated as possessing more negative traits and subjects were less likely to desire future contact with them than with other 'non-depressed' partners. Comments of support were also directed at the 'depressed' confederates (to a similar level as with 'physically ill' confederates) which adds credence to Coyne's (1976a) hypothesis that the contradictory nature of sympathetic and rejecting responses directed at depressives heightens their social difficulties.

Cumulative evidence from these studies does indicate that depressed individuals *do* unintentionally procure aversive reactions from others, which in turn is liable to result in their withdrawal from social situations and maintain any self-directed negative cognitive schema (Giesler et al., 1996). Coyne suggests that these social problems could be a consequence of the depressed person not possessing

sufficient additional social skills required to overcome the aversive responses often directed at him/her, rather than deficient social ability *per se* as proposed by Libet and Lewinsohn (1973).

Remedial treatment to address such difficulties – although potentially vital - will be ineffectual unless the underlying cause can be determined. Whether interpersonal problems are secondary symptoms or possible contributing factors in the onset of depression is another rudimentary issue that should be addressed in future research.

#### *2.4.2.1 Criticism from others*

Criticism from others is a very important aspect of expressed emotion (EE) – studies have shown that depressives who have relatives that score high on EE are significantly more likely to relapse (Uehara et al., 1996; Hooley et al., 1986). However, assessment of EE requires a long interview and a trained examiner so it is crucial to find other more economical predictors of relapse. Hooley and Teasdale (1989) conducted a study to examine how well EE, marital distress and perceived criticism (PC) predicted relapse in a sample of married clinically depressed patients. Both EE and marital distress predicted relapse equally well but PC from a spouse was the single best predictor of relapse.

Obviously it is uncertain how well PC relates to actual criticism – it is possible some depressives are just extremely sensitive to mild forms of criticism. The actual nature of the mediating relationship between PC and relapse also remains unclear and requires more research. Treatment interventions could focus more on the relationship between the depressed individual and his/her partner in an attempt to address this problem.

### 2.4.3 Interactions with strangers versus partners

Whether criticism from strangers is equally significant in depression maintenance is uncertain. Coyne (1985) stressed the importance of focusing on interactions with strangers, in addition to intimates, in this research. If depressive behaviours are present and negative reactions are still induced in others in spite of the depressed-stranger pairing having experienced no previous social contact (and thus no previous negative experiences), then such findings are similarly meaningful in contributing to our understanding of depression maintenance.

Observation of the actual behaviours involved in support giving and receiving (as opposed to questionnaire methods) facilitates better understanding of social support interactions in depression. Hale et al. (1997) examined such support behaviours in videotaped dyadic interactions between depressed patients and their partners and also between patients and non-depressed strangers. In light of Segrin and Abramson's (1994) suggestion that involvement behaviours might mediate rejection responses (see section 2.4.4), Hale et al. predicted that lack of emotional involvement in depressed individual's interactions with others might be associated with unfavourable outcome. Examples of high involvement behaviours are long speech duration, head nods, and encouragement. Low involvement behaviours in contrast include long periods of active listening and reduced rates of speech and encouragement.

The expectation that the stranger would exhibit more involvement behaviours towards the patient than the partner would was validated in the findings. Similarly patients appeared more eager, spoke more and made more effort speaking when interacting with a stranger as opposed to their partner. Partners tended to participate in more active listening, were less encouraging and spoke less than strangers did

when interacting with the patient. The best predictor of remission was amount of speech displayed by the partner – the fewer speech behaviours that occurred, the less likely the patient would recover, i.e., the depressed individual would perceive he or she was getting less support, the less verbally reinforcing his/her partner was.

A possible interpretation of these findings is that the increased involvement seen in patient-stranger interactions reflects the early stages of relationship processes in depression, when the other individual provides the necessary reassurance and support demanded by the depressive. As the relationship progresses (as in the patient-partner interaction) the degree of involvement decreases and less support is elicited from the other individual who begins to withdraw from the interaction. An alternative explanation that such processes reflect natural socially polite behaviour (i.e., individuals being more polite to strangers and concealing socially undesirable personality characteristics) cannot be ruled out though, as this study did not have control pairings of a non-depressed individual interacting with both a stranger and his/her partner. This study does further indicate the importance of social support behaviours to the course of depression and encourages the use of such direct ethological assessment in elucidation of underlying depressogenic processes in social relationships.

Hale et al. (1998) took this research one stage further and looked specifically at how *partners* of depressed patients (as opposed to strangers) interpreted schematic facial expressions. Using the same paradigm as Bouhuys et al. (1996) (see section 3.1.3), they asked depressed patients, their partners and age-matched non-depressed controls to rate schematic faces (see Figure 3.1) as to the degree to which each appeared *angry, disgusted, afraid, happy, inviting, sad, rejecting* and *surprised*. Both

depressed patients and their partners rated the faces as appearing *less* happy and inviting (i.e., positive emotions) than the controls. Both depressives and partners differed in response from the control group but did not differ from each other. In addition there was a slight tendency for both depressed individuals and their partners to perceive the faces as displaying more negative emotions than the controls, but this effect did not achieve significance. Replication of such a study seems crucial, as fewer positive appraisals of behaviour on both the depressed individual and the partner's behalf could not only result in decreased likelihood of recovery for the patient, but also the partners could themselves become at risk for future depressive episodes.

### 2.4.4 Explanation of why interactions become aversive

Segrin and Abramson attempted in their 1994 paper to establish a framework around which the interpersonal problems in depression could be more easily understood. To fully comprehend the difficulties surrounding social behaviour in depressives, the authors believed that two complementary research areas needed to be considered: that concerning social skill deficits in depressives heralded by Lewinsohn (1974; see section 2.4.1) and also evidence regarding how other individuals respond to depressives (see Coyne, 1976a and section 2.4.2). As interactions by definition involve at least one other individual in addition to the depressed person, the latter's behaviour can only be properly interpreted once the actions of the former have been taken into account. In spite of a large amount of evidence regarding both the aversive behaviours displayed by depressives in social situations and also the negative reactions consequently induced in other individuals, neither account has yet explained *why* such negative behaviours and rejection are

elicited in others. Focusing on general communication theories, Segrin and Abramson highlighted three ways in which such a vicious cycle might arise in depression.

Firstly, the authors suggested that the general lack of responsiveness exhibited by depressives - in terms of their slowness in response, lack of eye contact, slow speech rate etc. - might contribute to the rejection behaviours exhibited by others towards them. For efficient communication, most individuals expect others to respond to them reasonably enthusiastically, in a manner that is appropriate to the content (and context) of their conversation. (The authors suggest that such expectations might even relate to what were originally biologically adaptive behaviours, i.e., dependency on the response of significant others as infants for food and care.) Depressed individuals are therefore likely to be viewed as incompetent in the manner in which they respond to others due to their lack of apparent interest. Indeed, lack of responsiveness could even be perceived as hostile.

In addition, Segrin and Abramson (1994) suggested that depressives tend to violate assumptions of politeness. The authors described a theory (Brown & Levinson, 1978) suggesting that individuals require both the approval of others and also not to feel imposed on in social interactions. As depressed individuals tend to appear disinterested and detached in interpersonal situations, others might interpret such behaviour as disapproval. Moreover, the tendency of depressives to disclose personal information inappropriately (as a mechanism for gaining reassurance and support) could impose on others an undesired feeling of obligation to help. Again the consequence of such behaviours is the likely withdrawal of others from (and avoidance of future) interactions with the depressed individual.

The third factor, which Segrin and Abramson (1994) believed might contribute to the problems experienced by depressives in social situations, is other individuals' expectation of non-verbal involvement. Non-verbal involvement includes behaviours such as smiling and head nodding frequently in response to the other individual, engaging in eye contact, facial expressiveness and use of gestures, all of which suggest a degree of intimacy, engagement and interest and act as positive reinforcement of the interaction. Lack of such behaviours in depressives has been well-documented, for example, lack of happy facial expressions and eye contact, monotonous speech, long pauses and silences during conversation (Segrin & Abramson, 1994; see also section 2.4.1). The contravention of such normal involvement behaviours in depressives will again probably result in depressives being rejected and avoided by others.

Segrin and Abramson (1994) did stress that such problematic social behaviours are not necessarily unique to depression and some are often present in other psychopathological conditions. Therefore although these interpersonal difficulties might *contribute* to the occurrence of a depressive episode, they cannot be said to be a *sufficient* factor in the etiology of depression. Indeed, although the authors believe that impaired social functioning can act as a vulnerability factor for depression, they also conclude that some individuals might only experience interpersonal problems as a consequence of their depression. For example, depressive symptoms such as apathy, lack of concentration and negative affect might cause an individual who was previously socially proficient to respond slowly, not engage in eye contact, to speak in a monotone and not to be facially expressive - all of which are indicators of deficient social ability.

Whether or not these factors act as a contributing cause or are merely a later manifestation of depressive symptomatology, once present they are likely to impede the recovery of the depressed individual and extend the duration of the depressive episode, in the manner in which stressful social situations are repeatedly generated. Greater emphasis on interpersonal behaviour must therefore be incorporated into future therapy to both promote recovery and guard against future relapse.

### 2.4.5 Emotional sensitivity in depressives

Sensitivity to emotional cues in depression was assessed in a study by Wexler et al. (1994). The authors stressed the importance of determining emotional processing in depression due to the central role of affective disturbance in this condition. Their aim was to obtain an objective estimate of emotional responsivity in depression, rather than use more subjective self-rating measures.

Mimicry of others facial expressions occurs automatically and subconsciously and can give clues as to emotional response. The recording of such facial muscle activity - EMG (electromyography) - was carried out in this study with activity measured at two points: the *corrugator* (frowning) and the *zygomatic* (smiling) muscles. Activity has been demonstrated to be higher over *both* these regions in depressives during resting states than in non-depressives (Greden et al., 1986), suggesting an increase that is emotionally non-specific. Such an increase is perhaps not surprising, as general muscle tension is known to be enhanced in depressives. Here participants viewed images of posed neutral, sad and happy expressions and corresponding facial activity was measured. Zygomatic activity has been shown to increase when a happy face is viewed and conversely enhanced activity in the corrugator region has been reported during observation of a sad face

(Dimberg & Thunberg, 1998; Dimberg, 1982). It was predicted that the increase in zygomatic activity during presentation of happy facial expressions would not be so pronounced in depressed individuals, although it was unclear whether there would be any differences in corrugator activity between control and depressed participants.

As expected, depressed individuals did not exhibit the selective increase in zygomatic activity when viewing happy expressions. Corrugator activity did not increase specifically when viewing sad faces though, but was at a uniformly high level when viewing all the expressions. It is perhaps relevant to note that both groups of subjects performed equally well on recognition of the emotion presented, suggesting not only that there was no major impairment in identification of different expressions but also that differences in muscle activity were not due to any attention deficits in the depressed group. Attenuated EMG response has also been observed in depressed patients when recalling positive life experiences (i.e., happiness imagery) in an earlier study by Schwartz et al. (1976).

In addition to assessing facial muscle activity, a dichotic verbal listening test was also conducted in this study (on a different group of depressed individuals) to assess whether positively or negatively valenced emotional words were preferentially processed by depressives. Although two words (differing in both emotional content and initial consonant) are actually simultaneously presented, one to each ear, subjects only consciously report hearing one word. Depressed individuals in this study reported hearing less positive and negative words and more neutral words than controls.

Wexler et al. (1994) concluded that depressives showed abnormal responses to emotional stimuli in that they were less responsive to affective cues of varying natures (non-verbal and verbal), whether they were positive or negative in valence,

than non-depressed individuals. The authors believed that such blunting to emotional cues could equate to the sense of emptiness and detachment from the 'real world' experienced by many depressives.

Diminished response of depressives to positively valenced cues has also been reported by Sloan et al. (1997). Subjects were asked to rate slides varying in positive, neutral and negative content as to their affective valence and also as to their representation of arousal (consistent with Russell's model of affect; Russell, 1980). (All slides were matched as best as possible on the arousal dimension pre testing.) Examples of stimuli used for positively valenced pictures were people smiling or a delicious food display. For neutral stimuli, pictures of household objects were shown and examples of negative stimuli were pictures of people crying, or photos of spiders. Expressions made by participants in response to the slides were also recorded and rated independently.

Subjective responses as to the affective valence of the stimuli significantly differed between control and depressed participants only for the positive slides, with depressed patients rating the slides less positively than the controls. In addition, depressives rated the positive stimuli as less arousing than control subjects did. No differences were observed between the two groups in expressive response to positive stimuli, although this could have been due to the fact that the positively valenced stimuli did not succeed in eliciting much facial response from either group. Depressed subjects did exhibit slightly more negative facial expressions, however, in response to the negative stimuli than control subjects did. No differences were shown between depressed and non-depressed participants in either subjective or objective response to neutral stimuli. The authors concluded that depressed patients' decreased perception of positive affect (in the positive stimuli displayed) concurs

with the idea of anhedonia being a crucial factor in depression (Watson et al., 1988). Sloan et al. (1997) suggest that further studies should perhaps be conducted to examine whether loss of pleasurable experience plays an important causative role in the development of depression.

### 2.4.6 Developmental significance

Lundy et al. (1997) raised the importance of facial expression cues in development of effective social communication. Young infants are able to imitate and differentiate between different facial expressions from a very early age (Meltzoff & Moore, 1994; Field et al., 1982). A preference for synchronised facial-vocal expressions also seems to be present from a young age (between 5-7 months; Walker, 1982). As infants are likely to model their learning of emotional expression to some extent on their closest caregiver (normally the mother), the emotional state of this individual (and consequently the expressions they display) is liable to influence the developmental process. Lundy et al. (1997) decided to assess whether any developmental differences were present in facial-vocal affect matching of happiness and sadness between 10-month-old infants whose mothers were experiencing depressive symptoms[3] compared to infants whose mothers were not. The prediction was that infants with depressed mothers would be *less* able to match happy facial expressions with the appropriate vocal expression due both to a lack of emotional responsiveness and increased expression of negative affect in the mother. Such a pattern was present in the results. Infants with mothers experiencing

[3] It should be noted that the assessment of depressive state here was only contingent on the mother scoring over a cut-off point on the CES-D (Centre for Epidemiological Studies–Depression Index) scale as to how she'd felt over the previous week. Mothers were therefore not necessarily clinically depressed but just suffering from some degree of depressive symptomatology.

depression also spent more time looking at the sad faces presented and less at the happy faces than did infants with non-depressed mothers.

Longitudinal data is required before any conclusive statements can be made regarding the effects of the mood state of the primary caregiver on an infant's social development. In addition, maternal-infant behaviour would need to be recorded prior to the test session to assess whether actual differences between depressed and non-depressed mother's expressive behaviour existed. However, it is possible that such an increased focus on negative emotional cues and decreased response to positively valenced aspects of the environment might contribute to future depressive episodes at later stages in life if such a pattern of learning is instilled at an early age.

The aspect of social behaviour of particular interest in this thesis is facial expression perception. It is clear from this chapter that depressives experience difficulty in social situations, which could exacerbate negative self-directed cognitions. Although it is unclear what is inducing aversive responses in others, it is possible that misinterpretation of social signals, namely facial expressions, contributes to interpersonal problems in depressives. Indeed, studies have revealed that depressives exhibit differential sensitivity to emotional cues compared to non-depressed individuals (section 2.4.5). Examination of sensitivity to different facial expressions is the focus of the experimental chapters in this thesis. Before reporting results of such empirical studies on depressives (Chapters 4 and 5), the next chapter will provide a synopsis of previous research examining facial affect perception in depressed populations.

# CHAPTER 3: FACIAL EXPRESSION PERCEPTION IN DEPRESSION

## 3.1 INTRODUCTION

Facial expression perception *"offers promise as an investigational tool in the study of affective disorders ... because of its potential role in interpreting and determining social behaviour"* (Rubinow & Post, 1992).

The previous section in Chapter 2 described the nature of interpersonal difficulties experienced by depressives and commented on the putative contributing role of impaired processing of others facial expressions. This chapter highlights research conducted to-date on facial expression perception in depression, including studies on subclinical populations of individuals experiencing high levels of depressive symptomatology in addition to clinical groups.

### 3.1.1 No apparent deficit

Until fairly recently, most studies investigating facial expression recognition in psychiatric groups have focused on schizophrenic patients (Archer et al., 1994, 1992; Gessler et al., 1989). However, depressives have often been used as a psychiatric comparison group in such investigations.

Archer et al. (1992) conducted such a study to examine face processing in schizophrenics and depressives, using three different measures: facial expression recognition, face recognition and unfamiliar face matching. In the expression recognition task, 6 different emotions were used with stimuli taken from the Ekman and Friesen (1976) 'Pictures of Facial Affect' series. Subjects were presented with two images on each trial and required to identify which of the two faces was

expressing a target emotion, e.g., *'which face is sad?'*. It was reported that the performance of the depressed group on the facial expression recognition task was *not* noticeably impaired compared to controls and indeed their performance was similar to control subjects across all tasks. In contrast, schizophrenics were found to be generally impaired on all face processing measures.

Other studies have come to a similar conclusion. Gessler et al. (1989) found that control subjects and depressed patients performance did not differ on a task assessing facial emotion judgements. The stimuli used were newspaper photographs of spontaneous facial expressions, which had been consistently rated by another group of subjects to represent either happy or sad facial expressions. Subjects in the experiment were therefore required to identify whether the stimulus shown was depicting a happy or sad facial expression. The fairly straightforward forced-choice paradigm here ('happy or sad') could explain the lack of any deficit seen in depressives, due to ceiling effects. (For example, it is possible that highly accurate recognition scores could result even if an individual was only able to identify happiness and was therefore classifying non-recognisable emotions as sadness, by default.)

### 3.1.2 A generalised deficit

Other studies have demonstrated what appears to be an unspecific, generalised deficit in facial expression perception in depressed subjects, in the sense that the perception of all expressions is equally impaired, although the ability to recognise face identity remains intact.

Persad and Polivy (1993) examined the performance of both clinically and subclinically depressed individuals in comparison with control groups on an emotion

perception task. Students had to score 10 or over on the BDI to be regarded as mildly depressed (the subclinical group) and 9 or lower to be included in the non-psychiatric control group. In addition to meeting DSM-III criterion for major depression, depressed patients had to score 22 or over on the BDI to be included in the depressed patient group. There was an additional control group made up of non-depressed psychiatric patients, in order that differences in performance resulting from hospitalisation or medication could be ruled out. The control psychiatric group all had to have scores of 21 or lower[1] on the BDI and have no previous history or symptomatology of depression.

Subjects were required to label which emotion was being portrayed in a set of faces (Ekman & Friesen, 1976) and then rate both their behavioural and emotional responses to such expressions. For example, the subjects were asked the degree to which they would avoid a particular facial expression and how fearful an expression made them feel, respectively. The number of errors made in recognising the emotions was significantly higher for the two depressed groups compared to the non-clinical control group (BDI<10) and non-depressed psychiatric patient group (BDI<22), but errors were *not* specific to any one emotion. Both groups of depressed subjects were also shown to have less adaptive responses to the expressions - i.e., they rated themselves as more depressed in response to the expressions and also reported that they would be more likely to avoid all the facial expressions. This type of response could be maladaptive as depressed individuals might try to avoid social interactions and if they *do* engage in contact, they are likely to respond inappropriately to others' facial signals and focus on the negative impact of an

[1] A higher BDI cut-off score is allowed for non-depressed psychiatric patients due to the presence of some overlapping symptoms due to their condition, e.g., extreme tiredness, but not depression *per se*.

expression. In turn this is liable to heighten depressive state and feelings of discomfort, which will merely serve to increase withdrawal from social situations.

It is interesting that few differences were noted in performance between the two depressed subject groups, implying that it is possible to see disruption in expression coding even in quite moderate cases of depression. This suggests that differences in depressive state are *quantitative* rather than qualitative, which affects the degree of confidence with which conclusions can be drawn from studies investigating facial expression processing in non-clinical depressed populations.

The conclusion reached by Persad and Polivy (1993) was that this malfunction in decoding and response to facial expressions is highly likely to be contributing to the social problems inherent in depression, by amplifying the difficulties experienced by the depressed individual in social situations.

#### *3.1.2.1 Uncertainty in processing facial affect?*

Cooley and Nowicki (1989) observed a reduction in the speed with which depressives processed all facial expressions. They tested ability to perform a same-different discrimination task for facial expressions in individuals experiencing depressed affect using a reaction-time paradigm. Level of depression was assessed using the BDI, with scores of 10 or greater resulting in inclusion in the depressed group. Subjects were required to state whether two simultaneously presented images (using the 6 basic emotions from Ekman & Friesen, 1976) were the same or different as quickly as possible. Depressed subjects showed a slower reaction time in facial expression discrimination than non-depressed subjects, although they did not make any more errors. Their performance on a word discrimination task was not impaired, however, with equivalent reaction times shown by depressives and controls.

The authors concluded that depressed individuals difficulty might therefore lie in the fact that they cannot 'keep up' with the numerous non-verbal facial cues that are generated in social interactions. They believed that reduced processing speed (specific to non-verbal cues), rather than an impairment in expression recognition *per se*, could account for the deficit in emotion processing seen in depressives. It is possible such long reaction times relate to a depressed individual's uncertainty or lack of confidence in his/her own judgement[2] of the emotions expressed by another individual and it is notable that the reduction in processing seen here seems to be specific to facial expression perception. Slowness in processing did not appear to be specific to depressed subjects though, as a further 'disturbed' control group, who had low BDI scores, but high scores on at least one of the other Minnesota Multiphasic Personality Inventory (MMPI) scales, also exhibited similar problems on the facial emotion discrimination task.

### 3.1.3 Selective deficits

Bouhuys et al. (1995) attempted to assess the effect of depressed mood on expression recognition using a mood induction procedure (MIP) on normal subjects.

A set of 12 schematic faces designed by Cüceloglu (1970), which differed only in mouth shape and eyebrow position, were modified for use in this study (see Figure 3.1). These faces were rated in a preliminary study as to the degree with which they represented 8 different emotions (Bouhuys et al., 1995). In addition to the six *'basic'* emotions - *happiness, fear, anger, disgust, surprise, sadness* (as defined by Ekman & Oster, 1979), *invitation* and *rejection* were included as they relate to how people appraise others and thus might be more pertinent to someone

[2] The presence of a conservative response bias in depressives has also been demonstrated by Corwin et al. (1990) in a recognition memory task.

experiencing depression. The expression(s) perceived in each face was established according to inter-rater agreement. Six of the twelve schematic faces were described as *'clear'*, in that either positive or negative emotion was rated as more dominant, and the other six faces were *'ambiguous'* in that positive and negative emotions were equally portrayed. Within these subdivisions, faces were also distinguished by the intensity of emotion being expressed (3 intensive and 3 less intensive in each group).

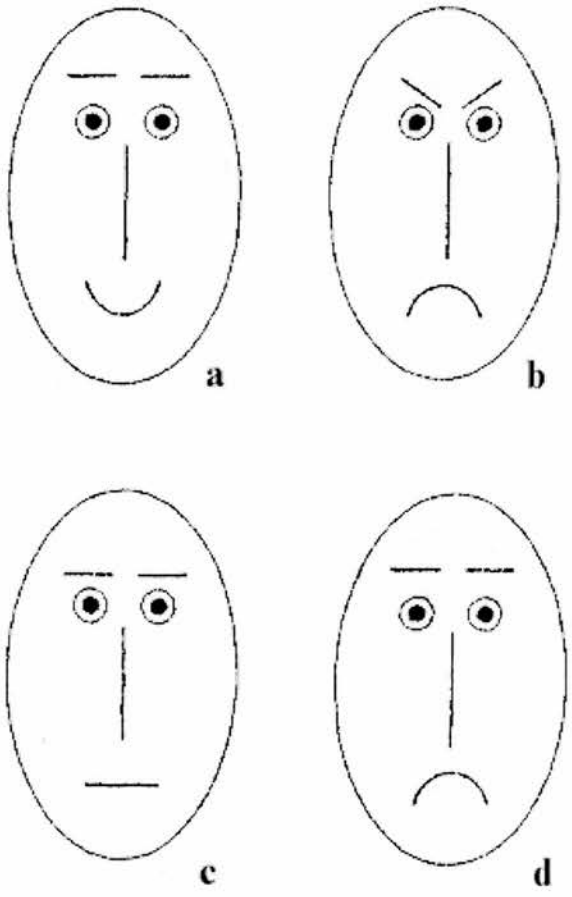


**Figure 3.1: Examples of the schematic faces used by Bouhuys et al. (1995, 1996): a and b represent 'clear' emotions, c and d 'ambiguous' emotions. a and c are examples of emotions rated as less intensive, whereas b and d are more intensive (not to scale).**

In the second part of the study, another set of normal subjects was subjected to two different musical MIPs - one that induced depressed mood and the other elated mood[3] - on separate sessions. Mood state was assessed before, during and after subjects rated the expressions. It was anticipated that perception of sadness and rejection might be affected by depressed mood, as these emotions are more relevant experientially in depression.

Results showed that subjects who had shown substantial mood changes subsequent to the MIP also exhibited differences in their judgements of expression. Following the depressed MIP, these subjects became more perceptive to the amount of rejection or sadness seen in a face, specifically in the ambiguous faces portraying less intense emotions, i.e., they would rate sadness and rejection as being more applicable in describing the faces than they had before. In addition, subjects whose mood had been influenced by the depressed MIP saw less invitation and happiness in the clear faces. Bouhuys et al.'s (1995) prediction that the negative bias in perception would be more pronounced for ambiguous faces was therefore validated by the results. Previous studies, using pictures of emotionally valenced stimuli rather than faces, have also demonstrated that mood state has a greater influence on an individual's judgement of 'ambiguous' rather than 'clear' pictures (Isen & Shalker, 1982).

These results suggest that individuals experiencing depressed affect are more likely to interpret expressions perceived by others to be ambiguous as negatively valenced. Similar findings have been reported in a clinically depressed group by Mandal and Bhattacharya (1985), in that depressed patients found identification of

[3] Each piece of music lasts approximately 7 minutes and mood is believed to remain altered for around 5 minutes (see Albersnagel, 1988).

sad faces easier than happy faces (from Ekman and Friesen, 1976) and were also inclined to label expressions they found difficult to identify as sadness.

Replication of such a study in a clinically depressed population is crucial before any conclusions can be reached regarding facial expression processing in depression, although the results are suggestive of a differential impairment in emotion recognition in depressives. Indeed, the fact that differences in perception can be observed *even* with such transient changes in mood (induced by MIP) has to emphasise the significant role affective state plays in perception and interpretation of emotion.

These results contrast with Persad and Polivy's (1993) finding of a generalised deficit in facial expression recognition in depression. Bouhuys et al.'s (1995) study indicates that the impairment in facial expression processing in depression is more circumscribed, in that only certain emotions were affected. The negative bias shown was only relative to specific emotions (happiness/invitation and sadness/rejection) and was not present for all negative emotions (i.e., disgust, fear or anger). However, the use of schematic faces might have prevented the portrayal of all emotions unambiguously. For example, Bouhuys's faces only included one nose type (straight line), which causes problems for the typical cue to disgust - a wrinkled up nose.

Cooley and Nowicki's (1989) hypothesis, that depressed individuals are merely slower to decode facial expressions but do not actually differ in their ability to identify them compared to non-depressed individuals, does not receive support from Bouhuys et al.'s (1995) study. Instead, it would appear that depressed individuals have a tendency to exhibit a negative bias in classification of some expressions.

The presence of a negative bias in rating of happy, neutral and sad facial expressions has also been demonstrated in a group of clinically depressed patients by Gur et al. (1992). Subjects were shown photos of actors posing a happy, neutral or sad expression[4] and were asked to rate the degree to which each image represented a happy, neutral or sad expression on a 7-point scale, (e.g., scoring '1' meant *very happy*, scoring '4' represented *neutral*, and '7' meant *very sad*). Depressed patients were inclined to rate the faces as being more negatively valenced than control subjects, i.e., there was a tendency for happy faces to be perceived as neutral and neutral faces as sad, indicating a general negative bias in perception of positive and neutral emotions. In spite of this, accuracy at identifying expressions (as happy, neutral or sad, regardless of emotional intensity) was equally high for both controls and depressives. Gur and colleagues stressed the importance of taking several indices of performance, in that selective deficits can be highlighted in one measure (e.g., bias), whereas seem apparently absent in another (e.g., percent correct).

These results seem reasonably intuitive, as one might expect interpretation of emotionally valenced non-verbal cues to be in accordance with depressive mood state, i.e., viewing expressions more negatively than non-depressed individuals would. The fact that such a negative bias has been demonstrated with different methods, in both clinically depressed patients and non-clinical individuals experiencing transiently induced depressive mood state (Bouhuys et al., 1995), indicates misinterpretation of emotional cues in this manner could have a serious impact on a depressed individual's interpersonal relations and could further enhance and maintain depressive state. It would be useful in future to examine whether this bias is restricted specifically to perception of sadness or whether it arises with other

[4] These stimuli were developed by Erwin et al. (1992) to make two discrimination tests - *happy-neutral* and *neutral-sad*.

negative emotions, although Bouhuys et al.'s (1995) findings indicate possible selectivity. These studies indicate that in addition to difficulty in *expression* of affect, depressives also have some impairment in *perception* of emotional expressions.

Rubinow and Post (1992) also examined facial expression perception in depressives, using a facial expression matching task and a control test of verbal expression matching. Depressed patients exhibited a selective deficit in facial expression matching compared to controls, with difficulties shown in matching photos of sad, happy and interested expressions. Depressives performed as well as controls in the verbal expression matching task though, indicating the facial expression deficit was not due to motivational problems. Rubinow and Post compared the impaired performance of the depressed patients in their study with that of patients with brain damage in the right hemisphere, who have been shown to manifest similar problems in facial affect matching (Kolb & Taylor, 1981).

Rubinow and Post (1992) believed that the selective deficit in perception of facial affect demonstrated in depressives could create behavioural difficulties due to inappropriate responding, concluding that depressives were unable *"to employ external and internal emotional cues to modify behaviour and alter self-perceptions"*.

### 3.1.4 Relation of facial expression perception to outcome

The relation between affect discrimination and persistence of depressive symptomatology was examined in a further study by Bouhuys et al. (1996). A prospective design was implemented with clinically depressed patients required to rate schematic facial expressions (Bouhuys et al., 1995; see Figure 3.1) on three

separate occasions: at admission and both 6 and 30 weeks after initial administration. Depression severity was clinically assessed by DSM-III-R criterion and further at every test session with the BDI. The aim was to establish whether rating of facial expressions demonstrated at admission would correlate in any way with prognosis over the test period, i.e., whether predictions could be made regarding persistence of depressive symptomatology.

Correlations revealed that depressives who judged faces previously rated as ambiguous (see Bouhuys et al., 1995) as expressing *less* sadness, anger or rejection at the initial test session were *more* likely to have an unfavourable prognosis, regardless of the initial severity of their depression. Intensity ratings for sadness and rejection demonstrated at admission did not appear to change over the course of the study, which led the authors to propose that perception of sadness in a face might be trait-like, i.e., stable. (It should perhaps be noted that sensitivity to sadness did not correlate with depression severity.)

These results contrast with the prediction that perception might be congruent with mood, i.e., that depressives exhibit a negative bias in expression perception involving an *increase* in sensitivity for certain negative emotions as reported in other studies (Bouhuys et al., 1995; Gur et al., 1992). In other words, one might expect a depressed person to view the world in a depressed manner and therefore be more perceptive to negative aspects of the environment. Hyposensitivity (decreased sensitivity) to expressions of sadness, rejection and anger, demonstrated here in patients experiencing poorer outcome, could perhaps indicate a defence mechanism employed by more severely depressed individuals in a subconscious attempt to block out feelings of ostracism. Rejection by others will nevertheless persist, owing to disinterest in the negative outlook exhibited by depressed individuals, which is likely

to result in maintenance of depressive symptoms. However, this is mere speculation and further research is necessary to determine both the presence and direction of a possible negative interpretative bias in facial expression perception in depression.

Examination of facial expression perception in both non-clinical individuals experiencing mild levels of depressive symptomatology and a clinically depressed group using more ecologically valid stimuli (rather than schematic images) is documented in Chapters 4 and 5 respectively.

## 3.2 CONCLUSIONS

Although some studies have not observed impaired perception of affect in depressives (see section 3.1.1) and others have revealed a general deficit in recognition of all expressions (see section 3.1.2), more recent studies employing more advanced empirical methods (specifically those involving rating of emotional intensity in addition to basic recognition measures) indicate that depressives interpret facial expressions in a different manner to non-depressed individuals. In particular, it would appear that perception of both happy and sad expressions is affected in some way (Bouhuys et al., 1996, 1995; Gur et al., 1992; Rubinow & Post, 1992). The importance of further research to examine the exact nature of any difficulties is evident, due to the likelihood that such misinterpretation will affect the quality of depressed individuals' social interactions and might consequently result in them feeling rejected and cause them to withdraw from interpersonal situations.

The following chapters will concentrate on studies formulated in an attempt to investigate the nature of the facial expression deficit in depression and whether any impairment relates only to clinical forms of the disorder or whether alterations are also present in milder conditions.

# CHAPTER 4: SUBCLINICAL DEPRESSION (STUDY 1)

## 4.1 INTRODUCTION

The aim of this exploratory study was to investigate whether facial expression perception varied in any systematic way according to affective state in a non-psychiatric population. An incorporated objective was to assess whether any such changes could be detected using a novel interactive facial expression processing test (see section 1.3).

As suggested in previous chapters, current mood state is likely to interact with interpersonal variables and could influence interpretation of others' behaviour and therefore how one responds in social interactions. It is meaningful to identify whether such processes arise (and the nature of them) in the general population as these factors could be playing a significant role (either causal or at least sustaining) in the social problems experienced by many individuals suffering from psychiatric conditions. As facial expressions are highly salient cues guiding people's behaviour and interactions, it is important to elucidate what factors influence expression interpretation and particularly how these might relate to individuals with affective disorders, in an attempt ultimately to alleviate some of the social difficulties inherent in many such syndromes.

The affective states that seem of particular relevance to the study of emotion are depression and anxiety, in that experience of emotion is altered in these conditions so one might expect perception of emotion to be similarly influenced. In addition, social problems are readily observable in many individuals suffering from such disorders (Brown & Moran, 1994; Coryell et al., 1993; Boyce et al., 1992; Borkovec et al., 1998). Before examining potential differences in a clinical

population however, it seems wise to establish first whether any trends are present in a non-psychiatric population experiencing varying levels of depressive and anxious symptomatology. There is of course much debate over whether milder subclinical forms of depression and anxiety equate to clinical syndromes or whether they merely represent high levels of negative affect and distress and are qualitatively distinct (for discussion see section 4.7.1). The frequently used clinical questionnaires, the Beck Depression Inventory (BDI; Beck et al., 1961) and the State-Trait Anxiety Inventory (STAI; Spielberger et al., 1983) were taken as estimates of depression and anxiety states respectively. Although elevated scores on these measures are unlikely to equate clinical conditions, for the purposes of this study the working assumption was that they at least tap to some extent some of the vulnerability factors for that disorder.

### 4.1.1 Predictions for individuals experiencing depressed mood

In the light of findings from previous research (mentioned in Chapter 3), some preliminary predictions can be made as to how sensitivity for different facial expressions will differ between subjects scoring low and relatively high on the BDI.

Evidence from studies investigating both cognitive processing in depression and experiments on facial affect recognition would suggest that depressives exhibit a selective encoding bias for emotionally negative information. One might predict therefore in the present study that individuals scoring high on depression will be more sensitive to negative emotions compared to non-depressed subjects. What is unclear, however, from previous research is whether this bias will be present across *all* negative emotions, or whether it will just be specific to some. The majority of evidence available would seem to suggest that only *certain* emotions might be

differentially processed in depression. In particular, it seems likely that perception of sadness would be particularly susceptible to the effects of depressive mood state. What is unclear however, is the direction of the relationship, i.e., would depressed individuals find it easier to detect sadness in a face (which seems more intuitive) or would they be less likely to perceive it than non-depressed individuals? At present, a case could be made for either direction, with some studies indicating enhanced perception of sadness (Bouhuys et al., 1995; Gur et al., 1992; Rubinow & Post, 1992; see section 3.1.3) and another reporting hyposensitivity to sadness (Bouhuys et al., 1996; see section 3.1.4) in depressives.

It is also possible that perception of anger might be altered in individuals experiencing depressed affect, due to concern that others might become irritated or annoyed with them or worry that others might disapprove of them.

The inclusion of anhedonia and lack of pleasurable experience as a symptom of depression might imply that sensitivity to happy expressions would correspondingly decrease in individuals experiencing depressed affect. A depressed person might therefore require a greater intensity of a happy expression, compared to an individual low in depression, to perceive the emotion of happiness.

### 4.1.2 Predictions for individuals experiencing anxious mood

The fact that emotionally negative information has been shown to be preferentially processed in anxious individuals (Maidenberg et al., 1996; Mogg et al., 1993) would lead to the prediction that individuals high in anxiety would orient more rapidly to negative or threatening facial expressions than individuals low in anxiety. Sensitivity to threatening emotions such as anger or fear (which alert an individual to immediate danger) might therefore be particularly enhanced in individuals exhibiting

high levels of trait anxiety and thus these subjects might be able to detect these emotions at low intensity levels.

Alternatively, it is possible that a more general sensitivity to all negative emotions might be apparent, for example, anxious subjects might be more sensitive to expressions of disgust and sadness (in addition to anger and fear) than non-anxious subjects.

It is possible that categorisation errors might also occur, in that positive emotions could be interpreted as negatively valenced, or expressions of disgust or sadness could be identified as negative, but misinterpreted as one of the expressions more relevant to anxiety, i.e., anger or fear.

## 4.2 METHODS

### 4.2.1 Participants

73 students ranging in age from 18-28 with a mean age of 21.4 years (SE=0.2) - 39 females (average age 21.6yrs, SE=0.3) and 34 males (average age 21.3yrs, SE=0.4) - from the University of St Andrews participated in the study. (82 subjects participated in total, but data from 9 subjects had to be discarded - 6 because overall percent correct recognition of expressions did not exceed 70% thus rendering sensitivity data unreliable and 3 due to incomplete questionnaire data.) All subjects gave informed consent to participate in the study.

All subjects underwent exactly the same testing procedure, as there were no experimental manipulations.

The Ethics Committee for the School of Psychology, University of St Andrews gave ethical clearance for this study.

### 4.2.2 Materials

Stimuli for the facial expressions were taken from the Ekman and Friesen (1976) Pictures of Facial Affect Series. The expressions used were *anger*, *disgust*, *fear*, *happiness*, *sadness*, *surprise* and also *neutral* (see Figure 4.1). Nine different identities were used (four male - em, jj, pe, wf; five female - mf, mo, nr, pf, sw), each portraying two different expressions. These stimuli were manually delineated by 179 feature-points that define the shape of all important facial features (Rowland & Perrett, 1995; see also Calder, et al., 1996b; see Figure 4.2). For example the shape and position of the upper eyelid is marked by five delineation points. The stimuli were presented computer-graphically. Subjects were seated approximately 50 cms from the computer screen.

The Beck Depression Inventory was used to measure depressive symptomatology (see Appendix 4.1). The BDI is made up of twelve questions assessing an individual's depressive state, for example how discouraged they feel about the future, or how critical they are of themselves for their mistakes. It has been frequently used to assess depression both at a clinical level and in the normal population. It is valuable in that it is both brief and straightforward to administer. It is also useful in the estimation of extent of depressive symptomatology experienced by an individual - i.e., whether the depression is of a mild-moderate (scoring between 10-18), moderate-severe (19-29) or extremely severe (30-63) nature.

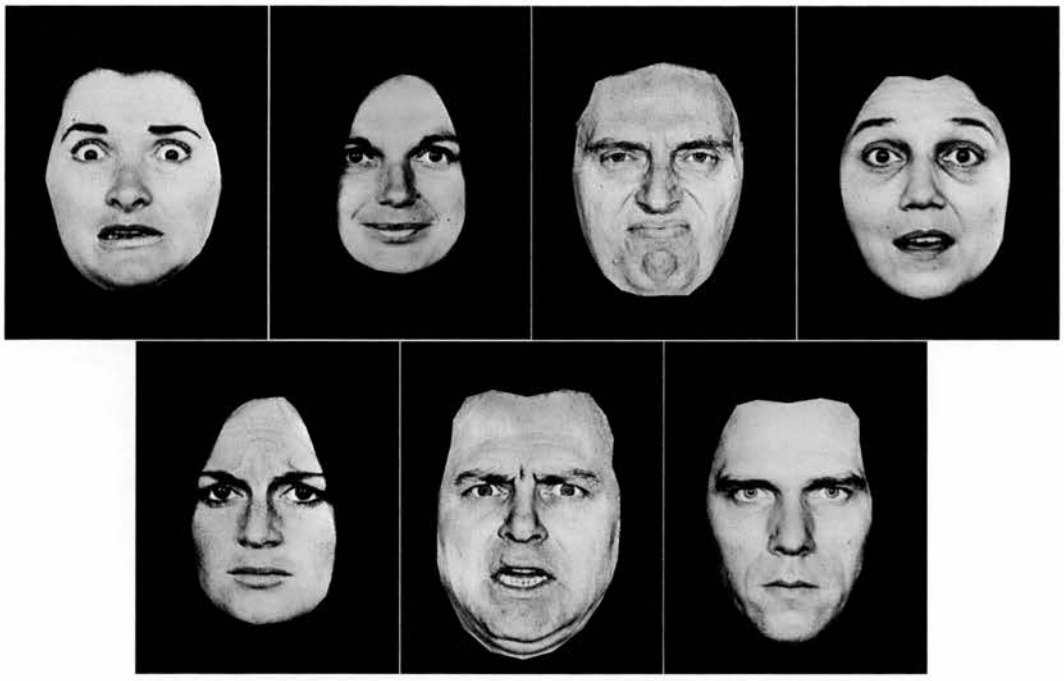

**Figure 4.1: An example of each of the 6 different expressions used plus neutral. (Clockwise from top left: *fear, happiness, disgust, surprise, sadness, anger* and *neutral.* The expressions are presented here in their veridical form at 100% intensity.)**

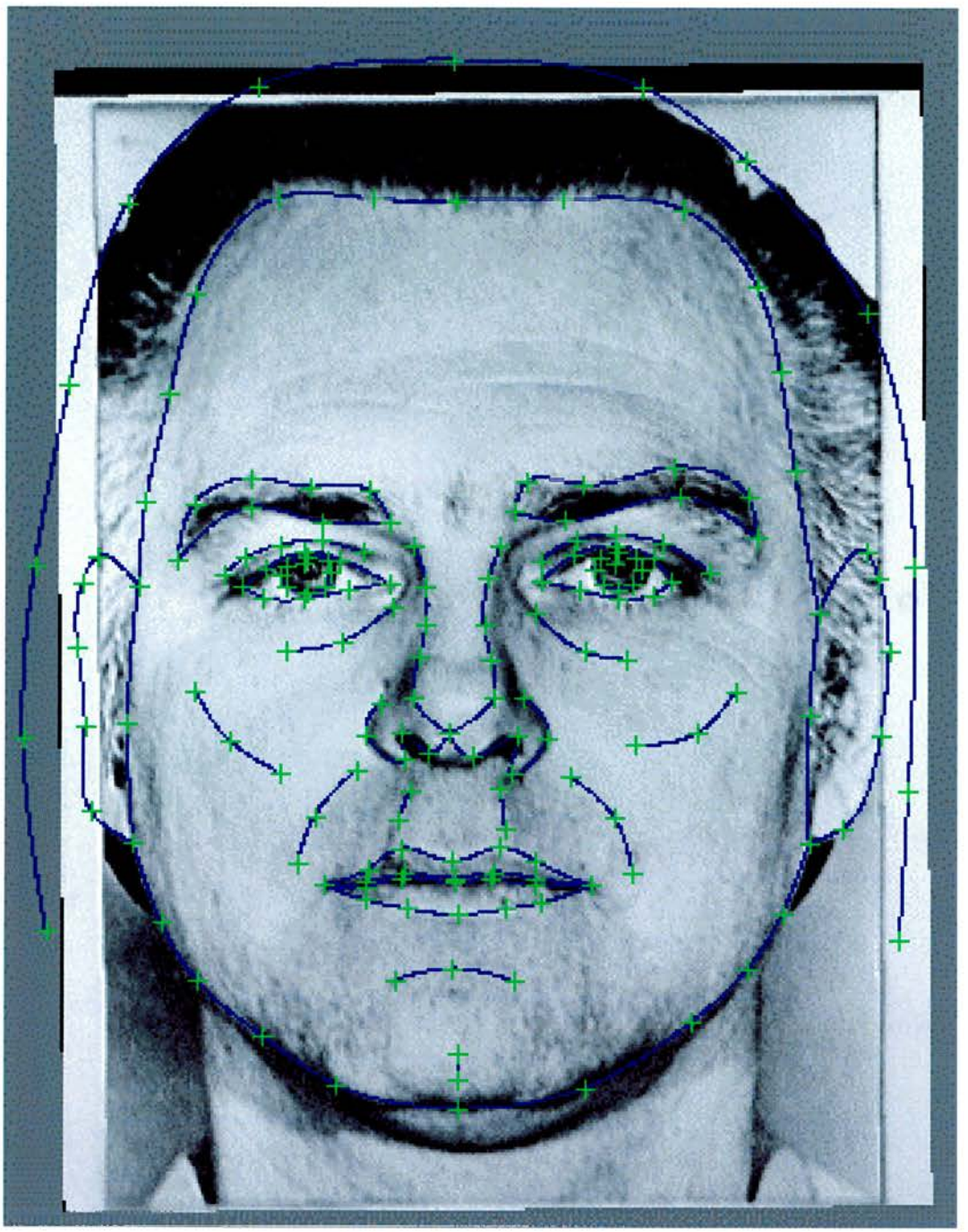

**Figure 4.2: An example of a delineated image with 179 feature points marking the salient facial features and round the hairline. (Points are estimated if parts of the image are occluded, for example the edge of the ears here.)**

The State-Trait Anxiety Inventory was used to assess anxiety levels (see Appendix 4.2). The STAI has been validated across different subject populations. The state scale measures relatively transient degree of anxiety experienced at any particular time, whereas the trait scale attempts to tap a more persistent personality-trait of anxiety proneness. There are twenty different statements in each scale, for example, *'I feel inadequate'*, to which participants respond along a 4-point scale, from *'not at all'* to *'very much so'*. Subjects can score between the range of 20-80, with norms around 35-40 for both state and trait measures.

### 4.2.3 Interactive program

An interactive program was designed, which enabled a continuous dynamic 'morph' to be created between two end-point images - a neutral face (0%) to one of six expressions (100%) (anger, disgust, fear, happiness, sadness or surprise). Movement of the mouse from left to right by the subject resulted in a gradual change in expression in real-time, from 0% (neutral) to 100% (expression) intensity. The continuous morph was generated by calculation of the difference in spatial position between corresponding feature points on the two end-point images (e.g., difference in the position between the corner of the left eye on a happy and a neutral expression). (See Figure 4.3 for an example of a morphed expression.) A percentage of this vector difference was then gradually applied to one of the end-point images in accordance with movement of the mouse. Both dimensions of shape and texture undergo the 0-100% transition. Blending of texture information is computed in a similar fashion to warping of shape. Average feature colour information (greyscale here) is calculated by averaging corresponding pixel values of the individual

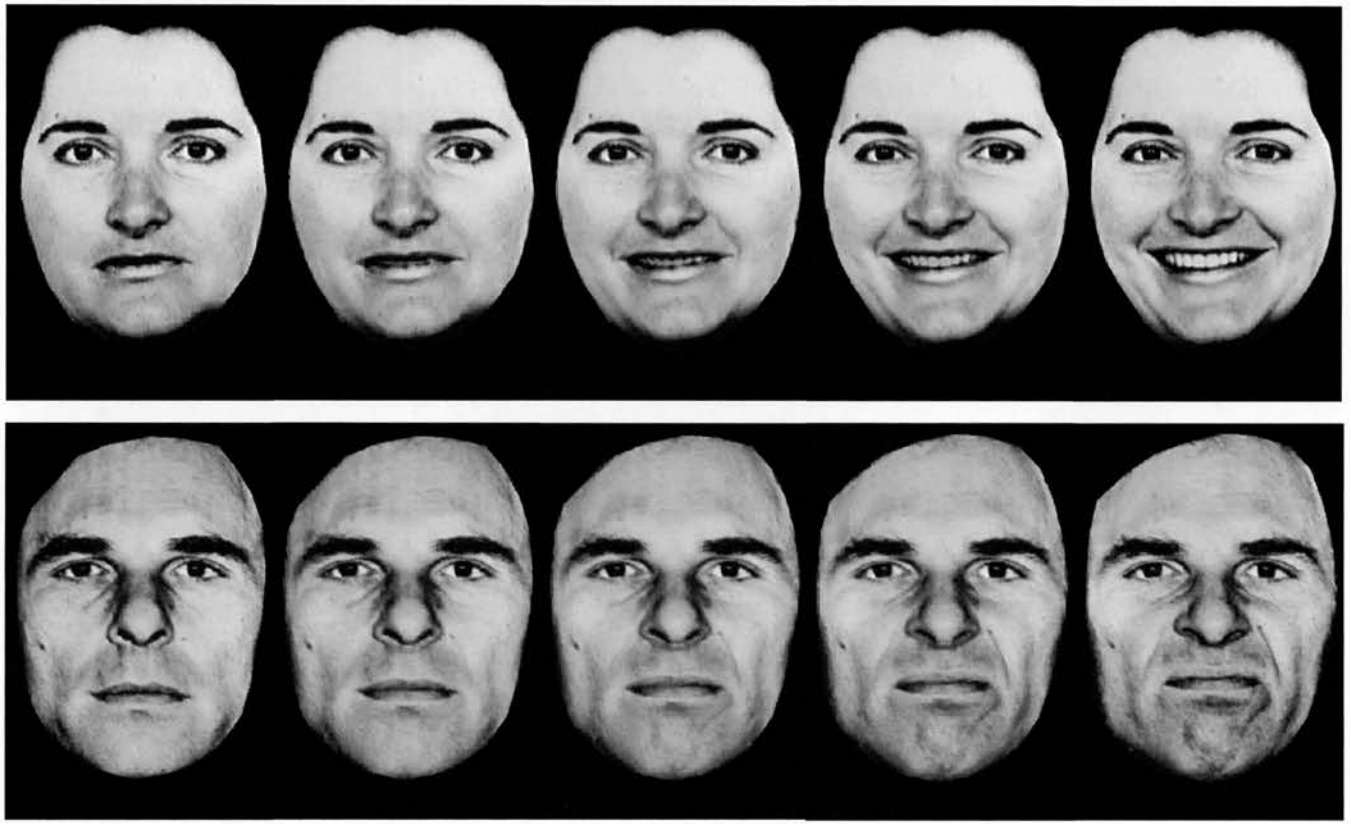

**Figure 4.3: An example of the morphed images used in the interactive test. The top image represents a neutral-happy morph and the bottom image a neutral-disgust morph in 25% steps from 0%-100%.**

expression images. For example, for a 50% neutral-happy morph, shape information from the neutral image and from the happy image is warped to an average shape (50% of the vector difference between neutral and happy end-point images) and the corresponding texture information from each image is blended together to give the average texture of the two images (50% texture information from each expression image).

The interactive morphs were generated on a Silicon Graphics maximum impact indigo$^2$ computer with 4 MB TRAM (texture memory).

### 4.2.4 Procedure

Each subject completed practice trials prior to the actual test block. The practice block constituted 6 trials, with 2 examples of 3 different expressions all portrayed by another Ekman identity (c - female). A prompt with names of the six different expressions (anger, disgust, fear, happiness, sadness and surprise) was placed in front of the keyboard for the duration of the study for the subjects to refer to if necessary. Trial order was completely randomised. The purpose of the practice block was to familiarise the subject with movement of the mouse and to enable understanding of how this directly resulted in change in the expression displayed. It also helped to make the subjects aware of the 6 emotions they were discriminating between.

Both sensitivity (ability to detect expression at varying intensities) and accuracy (percent correct identification of expressions) measures were of interest, so subjects had to perform two tasks in each trial. Subjects were instructed first to move the mouse from left to right to decide which one of the six expressions was being displayed. (When subjects initially clicked the mouse to start each trial, the image

would appear at a random point along the continuum, so they had to move the mouse around to identify the emotion.) Whether the neutral face or the expression was at the left or right of the screen was counterbalanced across trials, in an attempt to prevent a response set bias. (Subjects were aware that one end of the continuum was always a neutral expression.) Following identification, the subject was instructed to find the point along the expression continuum (starting from neutral) where he/she could first reliably classify the emotion e.g., as anger. This assessed a subject's sensitivity to different expressions. Once the subject was satisfied this point had been found, he/she had to press the space bar, at which point the six different expression names were automatically displayed on the screen and the subject then had to click on the expression that had been portrayed. (The image remained on the screen until the choice of expression had been decided at which point the screen cleared and the next trial appeared.) The subjects completed the two blocks of testing consecutively.

There were 18 different continua (three examples of each of the six expressions). Each continuum was repeated four times throughout the course of the experiment. The testing session was thus made up of 72 trials in total. Testing was carried out in two separate blocks of 36 trials (each block containing two examples of each continuum). Presentation order of the continua was randomised within a block.

Prior to the facial expression test, subjects were asked to fill out standard clinical questionnaires assessing mood state and also more stable, trait-like variables (see section 4.2.2). The order of presentation of the questionnaires was constant across subjects. Firstly participants completed the state and then trait version of the STAI. Both state and trait measures were included as it was hoped mood state on day of participation could be recorded and also, more importantly, that the individual's

more stable and enduring frame of mind could be gauged. Having completed both STAI scales, the subject then participated in the main part of the experiment rating facial expressions. After this was completed, the subjects then filled out the BDI.

## 4.3 RESULTS

Analyses of effects associated with BDI and STAI scores were initially examined separately as the assumption was that these measures were tapping different emotional states (albeit mild) and the consequent predictions were distinct.

### 4.3.1 Sample distributions

It was important to assess how well differences in mood had been sampled for across the population. The measures of interest – BDI and STAI-trait scale[1] (see section 4.2.2) - were reasonably sampled for in the current population. (It is possible to score anywhere between 0-64 on the BDI and 20-80 on the STAI-trait scale.) Both measures were however positively skewed (especially the BDI) with a tendency for low scores (see Figure 4.4), as one would expect in a relatively small sample from the normal population – particularly as the selected group consisted mostly of young, healthy students.

[1] Scores on the STAI-trait scale were used in preference to those on the STAI-state scale as these are more likely to indicate general tendency to experience anxious mood state.

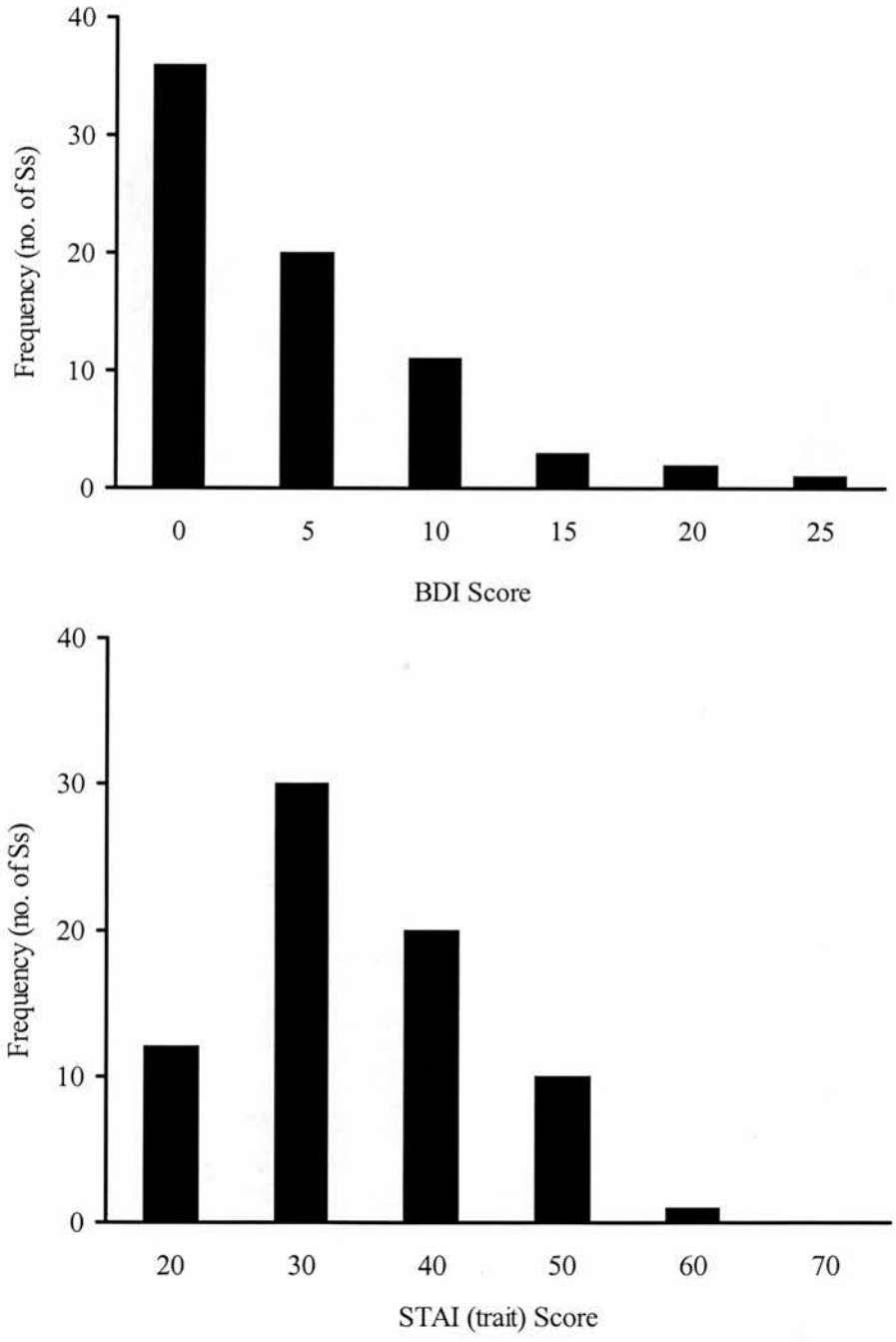


**Figure 4.4: Distribution of mood scores across the sampled population (n=73). The top graph illustrates scoring on the BDI and the bottom graph scoring on the STAI.**

### 4.3.2 Depression-related effects

Subjects were included in the high BDI group if they scored 10 or greater on the BDI (n=19), equating to the cut-off value used by Persad and Polivy (1993) and that defined in the BDI manual for mild-moderate depression (Beck et al., 1961). Scores of 9 or less resulted in inclusion in the low depression group (n=54). Average BDI score for the high BDI group was 14.7 (SE 1.1) compared to 4.1 (SE 0.4) for the low BDI group which was significantly different in a between-subjects t-test (unequal variance), t(21.7)=-9.0, p=0.001.

#### *4.3.2.1 Recognition accuracy*

A 2*2*6 mixed design ANOVA with subject group (low and high BDI) and sex as the between-subjects factors and expression as the within-subjects factor was conducted on the recognition accuracy data (percent correct out of a maximum of 12 trials for each expression).

The main effect for expression was significant, $F_{(3.7,253.2)}$[2]=8.3, p=0.001, implying that some expressions were easier to classify than others for both groups. Comparison of the mean values (see Table 4.1) indicates that subjects in both groups find happy expressions easy to recognise but are not so accurate at classifying sadness. Neither the main effect of group, $F_{(1,69)}$=0.4, p=0.5, nor the main effect of sex, $F_{(1,69)}$=2.0, p=0.2 was significant. In addition the between-subjects interaction of group and sex was non-significant, $F_{(1,69)}$=0.0, p=0.9, as was the 2-way interaction between expression and group, $F_{(3.7,253.2)}$=0.7, p=0.6. The interaction between expression and sex was also non-significant, $F_{(3.7,253.2)}$=2.3, p=0.06, although there was a trend for males to be slightly poorer at recognition of sadness than females.

[2] The Greenhouse-Geisser correction is used throughout as the within-subjects factor (expression) has more than two levels.

The 3-way interaction between expression, group and sex was not significant however, $F_{(3.7,253.2)}=0.7$, $p=0.6$. Therefore, high BDI subjects appear to exhibit no more difficulty in classifying expressions (at their full intensity of 100%) than low BDI subjects do (see Figure 4.5). Equally, subject sex does not seem to significantly influence ability to recognise different expressions (see Figure 4.6).

| | LOW BDI GROUP | | HIGH BDI GROUP | |
|---|---|---|---|---|
| | % correct | SE | % correct | SE |
| ANGER | 92.4 | 1.5 | 92.5 | 2.6 |
| DISGUST | 93.5 | 1.8 | 89.0 | 3.2 |
| FEAR | 89.8 | 1.9 | 92.1 | 3.0 |
| HAPPY | 98.0 | 0.7 | 97.8 | 1.1 |
| SAD | 81.0 | 2.9 | 83.8 | 4.3 |
| SURPRISE | 85.2 | 2.1 | 89.9 | 3.5 |

Table 4.1: A comparison of recognition accuracy scores for low (n=54) and high (n=19) BDI groups.

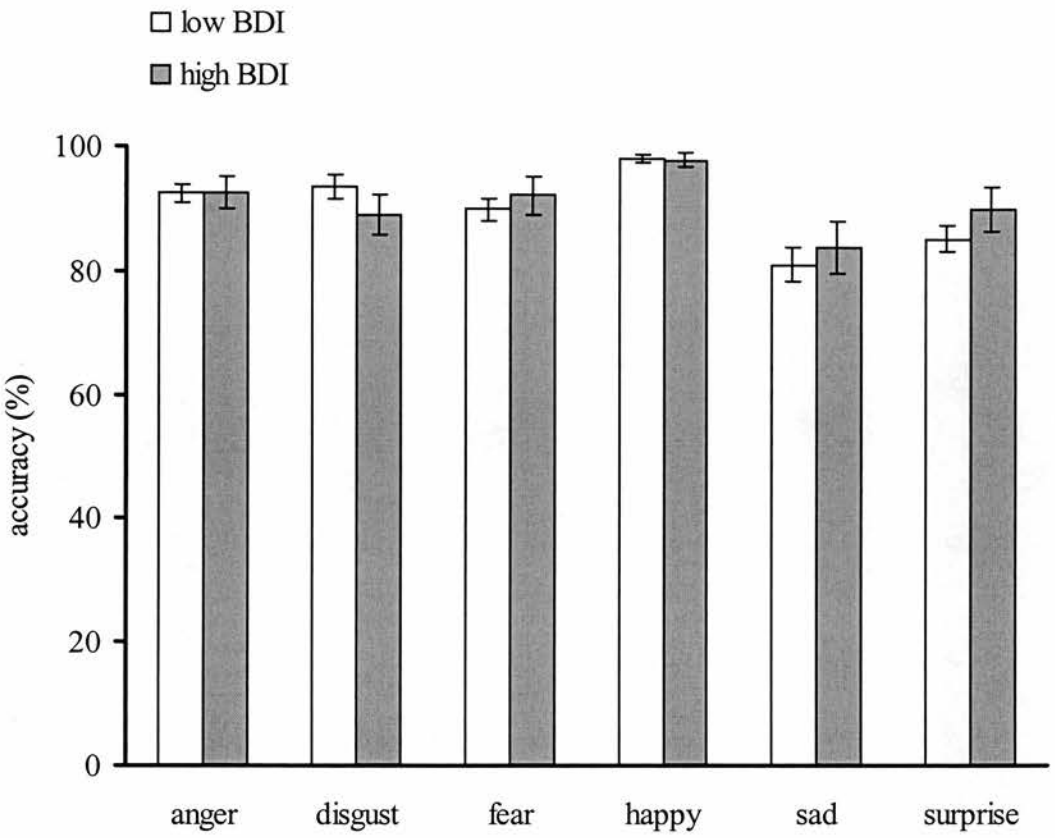


**Figure 4.5: A comparison of recognition accuracy (mean and SE) for the 6 emotions between low (n=54) and high (n=19) BDI groups.**

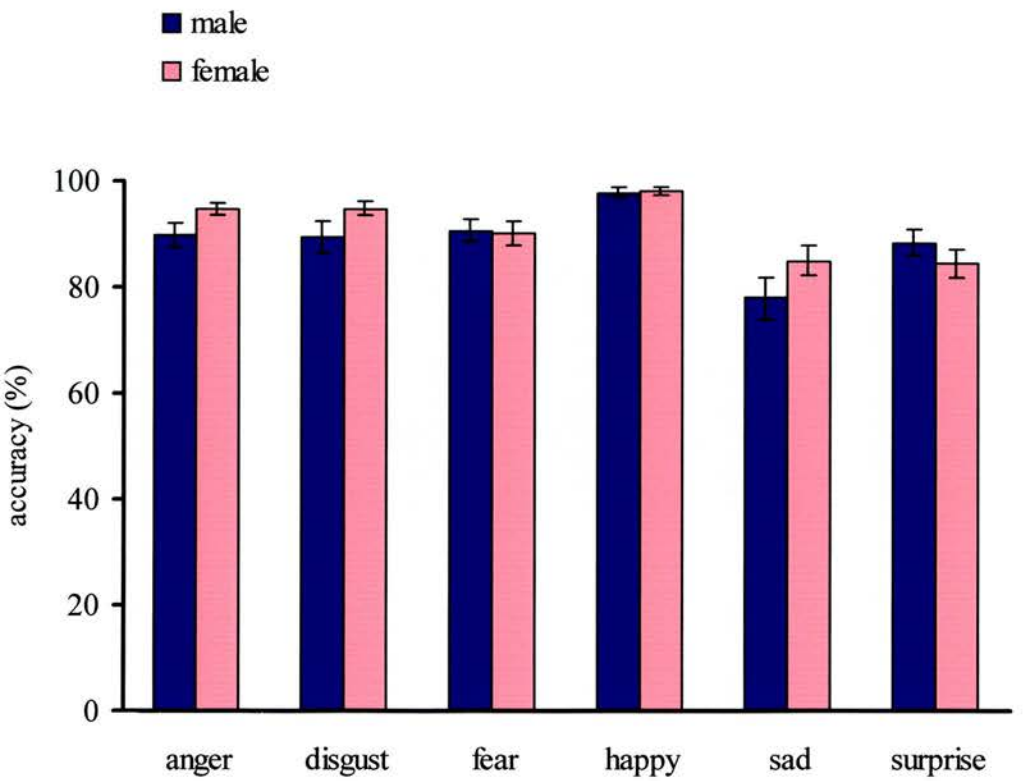


**Figure 4.6: A comparison of recognition accuracy across male (n=34) and female (n=39) subjects.**

More minor differences in error type can be seen when comparing high and low BDI groups (see Table 4.2). Individuals scoring low on the BDI mistake fear for surprise more than the high-scoring group (7.9% cf. 3.5%). High BDI subjects incorrectly classify disgust as anger more often than low BDI subjects (10.5% cf. 6.5%). Differences in error pattern between the two groups do not appear to be pronounced however. (Error pattern was not analysed according to subject sex, as there was neither a main effect of sex in accuracy nor any significant interactions with sex.)

| ACTUAL EMOTION | MISINDENTIFIED AS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | anger | | disgust | | fear | | happy | | sad | | surprise | |
| | ANGER | | | **1.8** | 1.9 | **4.4** | 4.2 | **0.4** | - | - | 0.3 | **0.9** | 1.2 |
| | DISGUST | **10.5** | 6.5 | | | - | 0.3 | - | - | **0.4** | 0.9 | - | - |
| | FEAR | **3.5** | 1.9 | **0.9** | 0.3 | | | - | 0.5 | - | 0.6 | **3.5** | 7.9 |
| | HAPPY | - | 0.2 | **0.4** | 0.3 | **0.9** | 0.8 | | | **0.9** | 0.3 | - | 0.6 |
| | SAD | **3.1** | 4.8 | **2.2** | 2.9 | **10.1** | 9.9 | - | 1.2 | | | **0.9** | 0.5 |
| | SURPRISE | - | 0.8 | **0.4** | - | **8.3** | 11.7 | **0.9** | 2.8 | **0.4** | 0.3 | | |

Table 4.2: Error pattern for high BDI (bold) and low BDI (normal type) groups. The value given is the average percentage of errors made across the group of subjects.

#### *4.3.2.2 Absolute sensitivity*

Sensitivity to an expression was recorded as a score between 0-100%, with 0% representing neutral and 100% representing the 'full' emotion as depicted in the original Ekman series. Trials were discarded from the analysis if subjects had misidentified the expression.

Again a 2*2*6 mixed-design ANOVA was carried out with subject group and subject sex as the between-subjects factors and expression as the within-subjects factor. There was a main effect of expression, $F_{(3.7,256.0)}=17.7$, $p=0.001$, so some emotions could be more easily detected than others for *all* subjects. The order of ease of classification across all subjects was: happiness 52.4%, anger 54.0%, fear 54.4%, disgust 55.3%, surprise 56.3% and sadness 62.8%. The main effect of group was not significant, $F_{(1,69)}=1.6$, $p=0.2$, suggesting subjects with elevated BDI scores are no

more or no less sensitive to all expressions than low scoring subjects (see Figure 4.7). Similarly the main effect of sex was non-significant, $F_{(1,69)}=0.0$, $p=0.9$ as was the between-subjects interaction of group and sex, $F_{(1,69)}=0.0$, $p=0.9$, (see Figure 4.8). The expression by group interaction was also non-significant, $F_{(3.7,256.0)}=2.2$, $p=0.074$, although there was a trend indicating that patterns of sensitivity for different expressions might differ across the two groups. This differential sensitivity can be more appropriately examined in the relative sensitivity analysis (see next section, 4.3.2.3). The interaction of expression and sex was not significant but exhibited a trend, $F_{(3.7,256.0)}=2.2$, $p=0.077$, which is again clearer to analyse using relative sensitivity data (see next section). The 3-way interaction of expression, group and sex was not significant however, $F_{(3.7,256.0)}=1.3$, $p=0.3$.

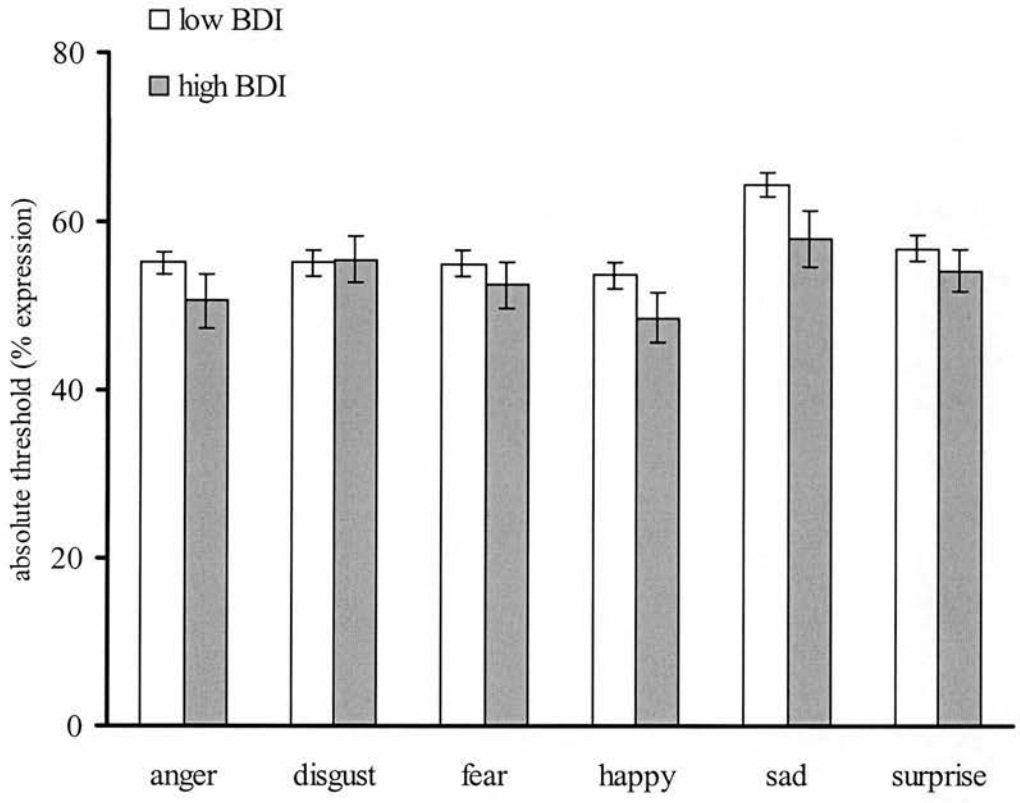


**Figure 4.7: A comparison of absolute sensitivity level (between 0-100%) for the 6 emotions between low (n=54) and high (n=19) BDI groups.**

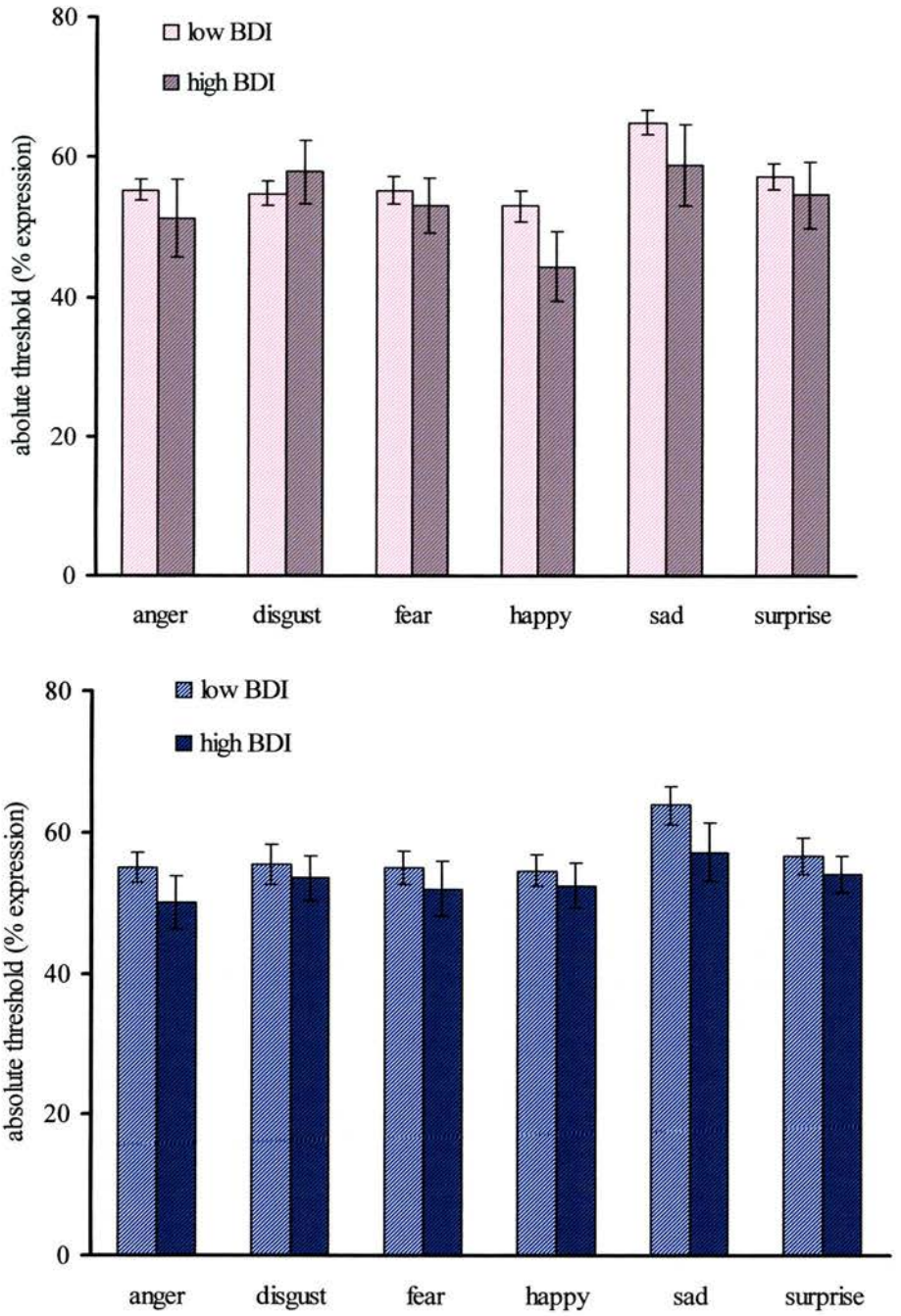


**Figure 4.8: A comparison of absolute sensitivity level (0-100%) in low and high BDI subjects divided by sex. The results for females (low BDI n=30, high BDI n=9) are illustrated in the top graph and for males (low BDI n=24, high BDI n=10) in the lower graph.**

### *4.3.2.3 Relative sensitivity*

Of interest was the possible interaction between expression and group, i.e., would depressed subjects be more sensitive to particular emotions than normal subjects? To examine the difference in the pattern of sensitivity across emotions, an estimate of *relative* sensitivity to each emotion compared to sensitivity to all emotions was calculated for each subject. The contrast measure used was: (a - b)/ b, were a is the mean sensitivity for the expression of interest and b is the overall mean sensitivity to all the emotions. This measure estimates the sensitivity to any one emotion relative to other emotions and takes into account the overall sensitivity level of the subject.

A 2*2*6 mixed-design ANOVA with subject group (low or high BDI score) and subject sex as the between-subjects factors and expression (6 levels) as the within-subjects factor was conducted on the relative sensitivity data. There was a main effect for expression across all subjects, $F_{(3.7,257.8)}=17.3$, $p=0.001$, suggesting that some emotions are relatively easier to detect at lower intensity than others for all subjects. Neither the main effect of group, $F_{(1,69)}=0.1$, $p=0.8$, nor of sex, $F_{(1,69)}=0.7$, $p=0.4$, was significant, which reflects the contrast measure, as the average relative sensitivity to all the emotions for each individual and therefore each group approximates zero. Similarly the between-subjects interaction of group and sex was non-significant, $F_{(1,69)}=0.7$, $p=0.4$. The interaction between expression and group was significant however, $F_{(3.7,257.8)}=3.0$, $p=0.023$. The different pattern of relative sensitivity for low and high BDI subjects across the six expressions is illustrated in Figure 4.9. Comparison of relative means suggests that subjects scoring high for depression on the BDI found it easier to identify sad expressions, but *more difficult*

to detect disgust, relative to other emotions and to less depressed subjects. Post-hoc analysis (Tukey HSD) confirmed that relative sensitivity to disgust was significantly diminished in high compared to low BDI subjects, p=0.04. Relative sensitivity for expressions of anger, fear, happiness and surprise did not differ significantly between low and high BDI subjects. Although non-significant, p=0.09, there was a trend for low and high BDI subjects to differ in relative sensitivity to expressions of sadness. The expression and sex interaction was also significant, $F_{(3.7,257.8)}$=2.9, p=0.025. Post-hoc analysis (Tukey HSD) revealed that male and female subjects differ significantly in their relative sensitivity to happiness (p=0.03), with females being relatively more sensitive than males (see Figure 4.10). The 3-way interaction of expression, group and sex was non-significant however, $F_{(3.7,257.8)}$=1.4, p=0.2, (see Figure 4.11).

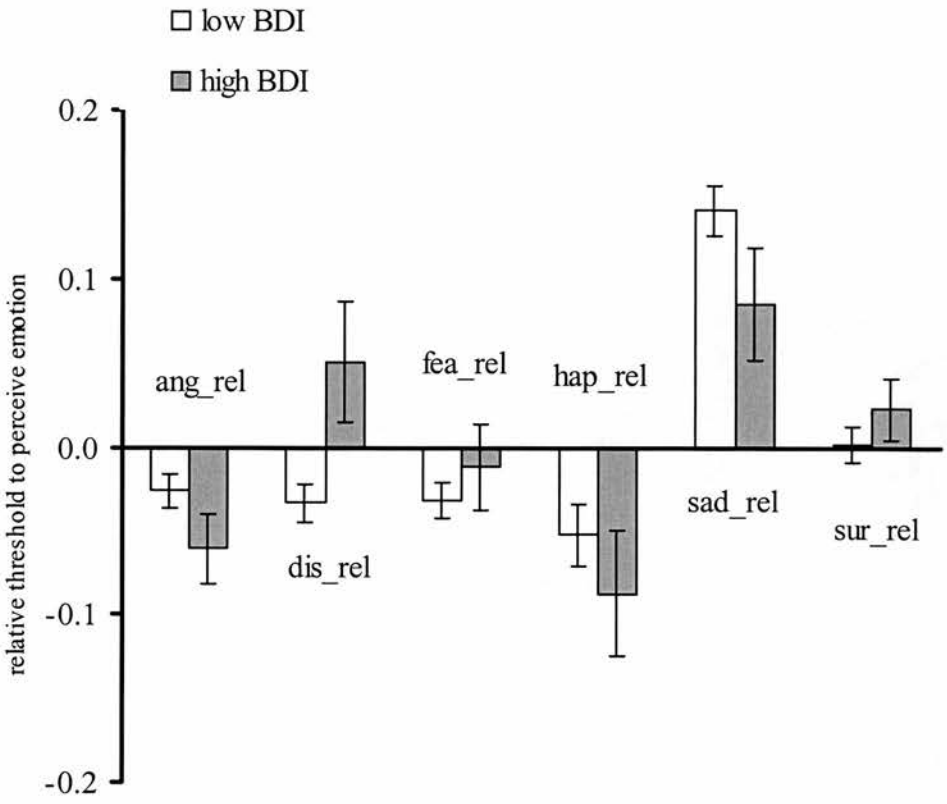


**Figure 4.9: A comparison of pattern of relative sensitivity across the 6 emotions between low (n=54) and high (n=19) BDI groups. A high threshold denotes decreased sensitivity.**

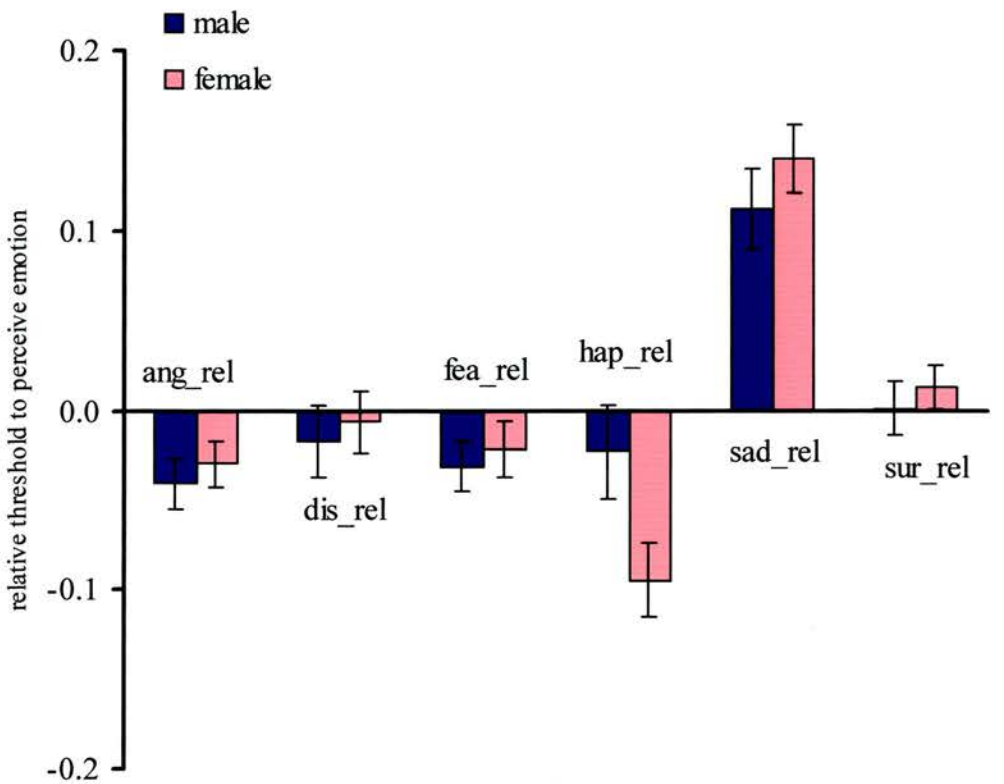


**Figure 4.10: A comparison of pattern of relative sensitivity across the 6 emotions between males (n=34) and females (n=39).**

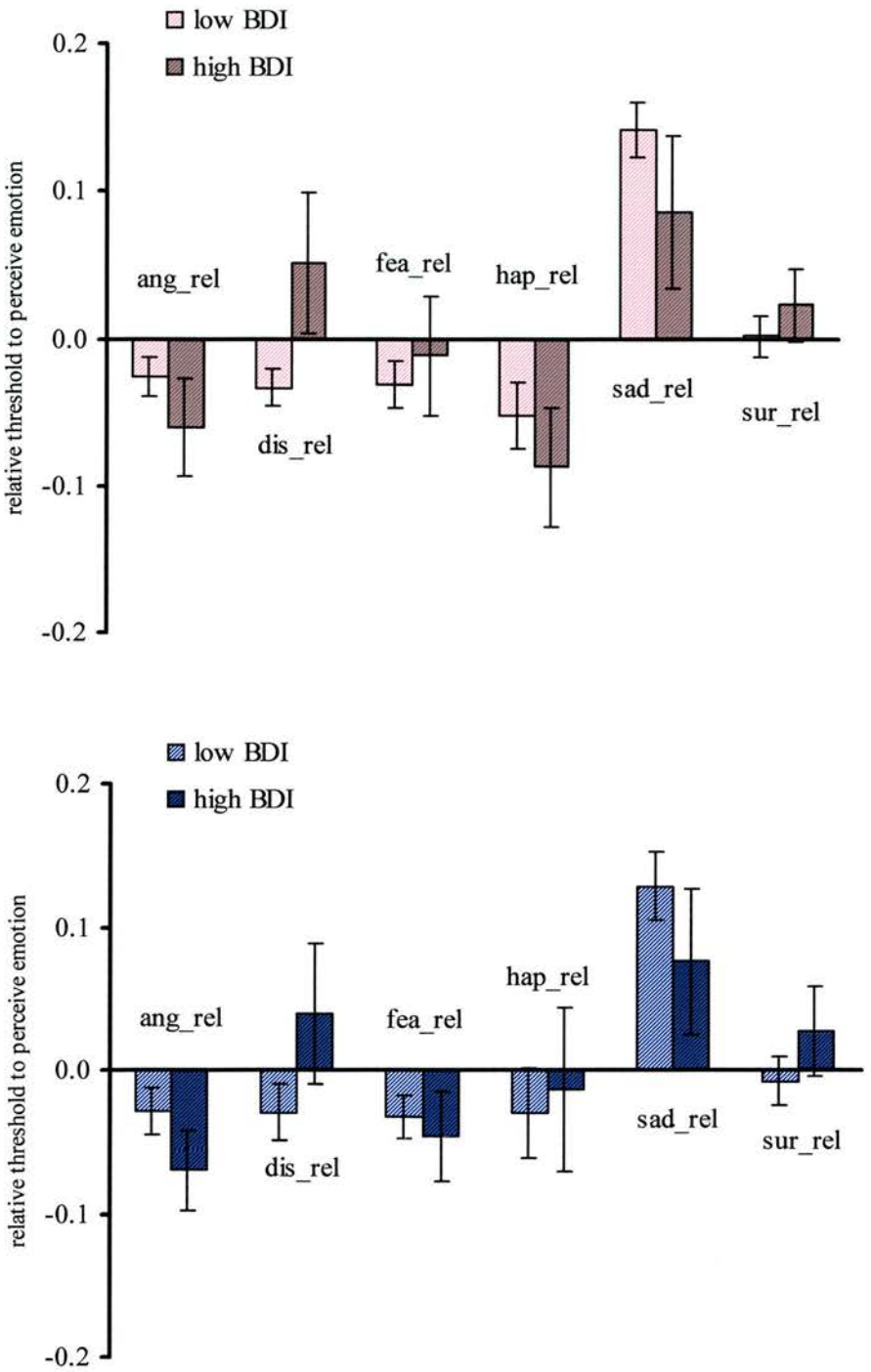


**Figure 4.11: Pattern of relative sensitivity across the six emotions for low and high BDI subjects divided by sex. The results for females (low BDI n=30, high BDI n=9) are illustrated in the top graph and for males (low BDI n=24, high BDI n=10) in the lower graph.**

### 4.3.3 Anxiety-related effects

Subjects were divided into the low or high anxiety group according to scores on the trait version of the STAI, as these should provide a closer estimate of stable individual differences than scores on the state scale. Since there are no standard cut-off values for the STAI-trait, one SD above the normative values (assessed for college students) was used for both male (47.5) and female (50.5) subjects. 14 subjects (8 male, 6 female) exceeded this criterion, giving 59 in the low STAI group and 14 subjects in the high STAI group.

#### *4.3.3.1 Recognition accuracy*

A 2*2*6 mixed-design ANOVA was carried out for low and high STAI subjects, with subject group and subject sex as between-group factors and expression as the within-subjects factor.

There was a main effect of expression, $F_{(3.6,249.4)}=7.7$, $p=0.001$, again showing that some expressions are easier for all subjects to identify than others, e.g., happiness. Main effects were not present for either group, $F_{(1,69)}=0.1$, $p=0.8$, or sex, $F_{(1,69)}=1.0$, $p=0.3$. The between-subjects interaction between group and sex was also non-significant, $F_{(1.69)}=0.0$, $p=0.9$. There was, however, an significant interaction between expression and group, $F_{(3.6,294.4)}=3.4$, $p=0.005$, indicating a different pattern of recognition accuracy across expressions depending on whether subjects were in the low or high STAI group. Particular expressions were therefore more difficult to identify for one group than for the other (see Figure 4.12 and Table 4.3). Post-hoc analysis (Tukey HSD) revealed that subjects scoring high for anxiety found it significantly more difficult to recognise expressions of disgust compared to low anxiety subjects, $p=0.001$, misidentifying it approximately 1 in 5 times. Disgust was

most commonly misidentified as anger by high anxiety subjects (see Table 4.4), which could reflect a slight response bias to see faces as angry or hostile. There were also trends for high STAI subjects to be slightly better at recognising angry expressions than low STAI subjects, p=0.08. In addition, high STAI subjects showed a trend to be worse at identifying happiness than low STAI subjects, p=0.06.

The interaction between expression and sex was non-significant, $F_{(3.6,249.4)}=2.2$, p=0.073, although there was a trend perhaps reflecting that female subjects were better than male subjects at identifying sad expressions. The 3-way interaction between expression, group and sex was not significant however, $F_{(3.6,249.4)}=0.8$, p=0.6.

| | LOW STAI GROUP | | HIGH STAI GROUP | |
|---|---|---|---|---|
| | % correct | SE | % correct | SE |
| ANGER | 91.4 | 1.5 | 97.0 | 1.9 |
| DISGUST | 95.2 | 1.3 | 80.4 | 5.1 |
| FEAR | 89.5 | 1.9 | 94.0 | 1.6 |
| HAPPY | 98.4 | 0.5 | 95.8 | 2.1 |
| SAD | 81.8 | 2.5 | 81.5 | 6.9 |
| SURPRISE | 85.9 | 2.0 | 88.7 | 4.4 |

Table 4.3: A comparison of recognition accuracy scores for low (n=59) and high (n=14) STAI groups.

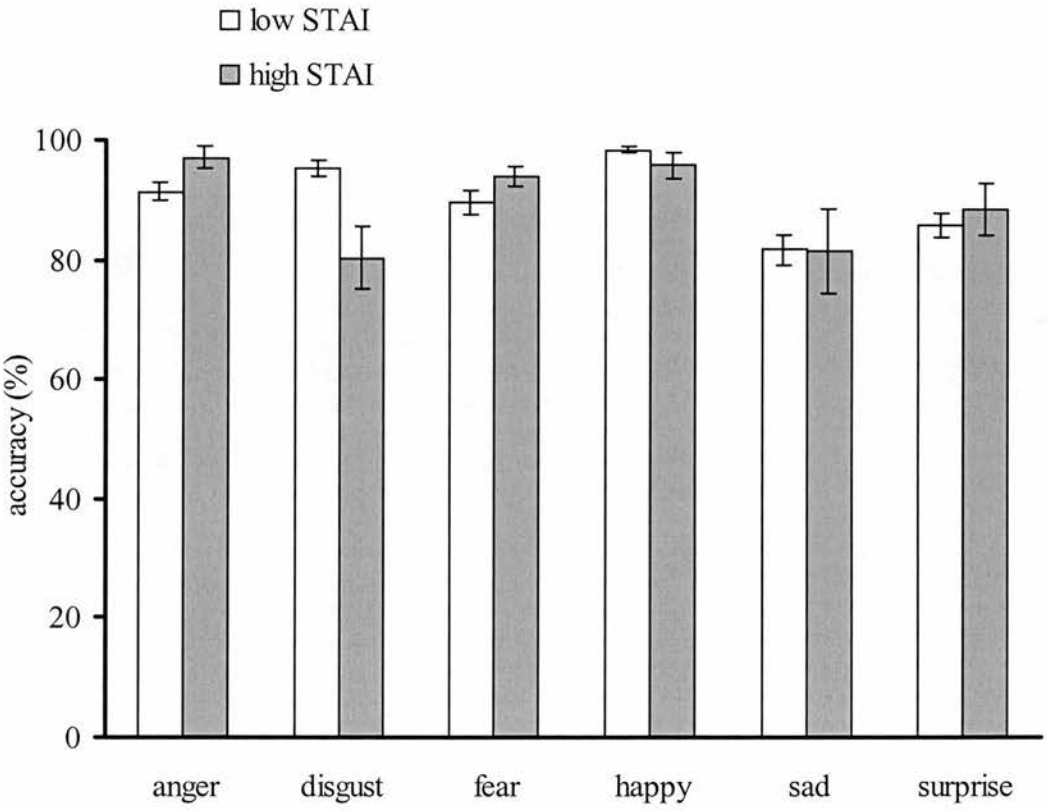


**Figure 4.12: A comparison of recognition accuracy (mean and SE) for the 6 emotions between low (n=59) and high (n=14) STAI groups.**

| ACTUAL EMOTION | MISINDENTIFIED AS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | anger | | disgust | | fear | | happy | | sad | | surprise | |
| | ANGER | | | **0.0** | 2.3 | **3.0** | 4.5 | **0.0** | 0.1 | **0.0** | 0.3 | **0.0** | 1.4 |
| | DISGUST | **19.0** | 4.2 | | | **0.0** | 0.3 | **0.0** | 0.0 | **0.6** | 0.3 | **0.0** | 0.0 |
| | FEAR | **0.6** | 2.5 | **1.2** | 0.3 | | | **0.0** | 0.4 | **0.0** | 0.6 | **4.2** | 6.7 |
| | HAPPY | **0.0** | 0.1 | **0.6** | 0.3 | **2.4** | 0.4 | | | **0.6** | 0.4 | **0.6** | 0.3 |
| | SAD | **3.6** | 4.1 | **3.0** | 3.2 | **11.3** | 9.2 | **0.6** | 1.0 | | | **0.0** | 0.7 |
| | SURPRISE | **0.0** | 0.7 | **0.6** | 0.0 | **10.1** | 11.1 | **0.0** | 2.1 | **0.6** | 0.2 | | |

Table 4.4: Error pattern for high STAI (bold) and low STAI (normal type) groups. The value given is the average percentage of errors made across the group of subjects.

#### *4.3.3.2 Absolute sensitivity*

A 2*2*6 mixed-design ANOVA was carried out with subject group and subject sex as the between-subjects factors and expression as the within-subjects factor. There was a main effect of expression, $F_{(3.7,258.0)}=12.4$, $p=0.001$, with all subjects being able to detect some emotions at lower intensity than others (see section 4.3.2.2). There was also a main effect of group, $F_{(1,69)}=4.5$, $p=0.037$, with low scoring subjects needing all the emotions to be displayed at a greater intensity to first perceive them than individuals with elevated STAI scores (see Figure 4.13), which implies that anxious individuals are generally *more sensitive to facial emotion cues*. Again there was no main effect of sex (see section 4.3.2.2), $F_{(1,69)}=0.2$, $p=0.6$, and also no interaction between anxiety group and subject sex, $F_{(1,69)}=0.5$, $p=0.5$.

Similarly, none of the interactions with the within-subjects factor of expression were significant: expression and group, $F_{(3.7,\ 258.0)}=1.1$, $p=0.4$; expression and sex, $F_{(3.7,258.0)}=1.4$, $p=0.2$; expression, group and sex, $F_{(3.7,\ 258.0)}=0.4$, $p=0.8$.

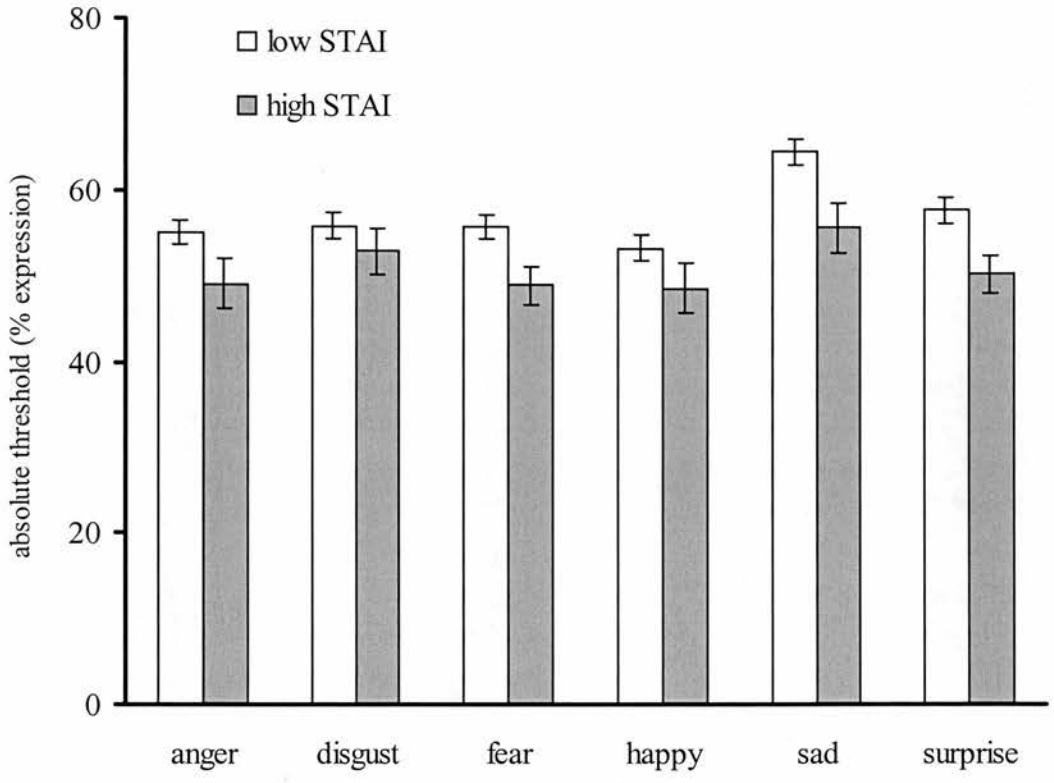


**Figure 4.13: A comparison of absolute sensitivity level (between 0-100%) for the 6 emotions between low (n=59) and high (n=14) STAI groups.**

### *4.3.3.3 Relative sensitivity*

Although the group of high scoring STAI subjects were more sensitive overall to all emotions than the low scoring group, it was unclear whether this enhanced sensitivity was in any way differential across the emotions, i.e., whether high scoring STAI subjects would be particularly sensitive to any one emotion. The relative sensitivity analysis was therefore conducted.

A 2*2*6 mixed-design ANOVA was carried out, again with subject group and sex as between-subjects factors and expression as the within-subjects factor. Neither of the between-subjects factors or their interaction was significant, which was expected due to the contrast measure: main effect of group, $F_{(1,69)}=0.9$, $p=0.3$; main effect of sex, $F_{(1,69)}=0.6$, $p=0.4$; interaction of group and sex, $F_{(1,69)}=0.3$, $p=0.6$. Once again there was a main effect of expression, $F_{(3.8,259.8)}=12.4$, $p=0.001$, with happiness having the lowest relative threshold and sadness the highest for all subjects. Subject group did not interact with expression, $F_{(3.8,259.8)}=0.9$, $p=0.5$, suggesting that subjects with high STAI scores were not differentially more sensitive to any specific emotions (see Figure 4.14) despite being more sensitive overall (see Figure 4.13). Neither the expression and sex, $F_{(3.8,259.8)}=1.9$, $p=0.1$, nor the expression, group and sex, $F_{(3.8,259.8)}=0.4$, $p=0.8$, interactions were significant.

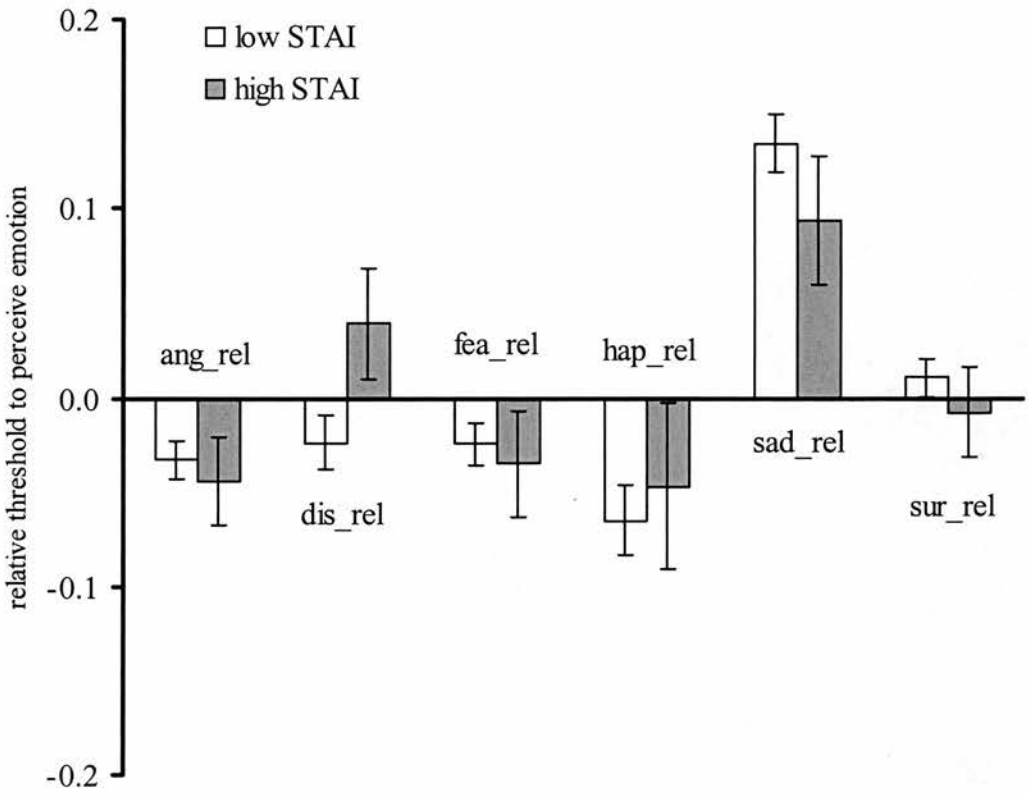


**Figure 4.14: A comparison of pattern of relative sensitivity across the 6 emotions between low (n=59) and high (n=14) STAI groups. A high threshold denotes decreased sensitivity.**

### 4.3.4 Comorbidity of BDI and STAI

Depression and anxiety frequently co-occur in clinical populations, therefore one might predict that a similar comorbidity might be present in a subclinical population. 10 of the 14 subjects in the high STAI group were also in the high BDI group. Pearson's r (one-tailed) showed a highly significant correlation between the two factors, $r(73)=0.6$, $p=0.01$.

Many have questioned whether elevated scores on measures assessing either depression or anxiety in a normal population just represent a high level of negative distress as opposed to milder forms of discrete depression and anxiety disorders. In order to assess the relevance of the comorbidity issue with regard to the current study, additional analysis were conducted with STAI entered as a covariate in the BDI analysis and conversely BDI as the covarying factor in the STAI analysis.

#### *4.3.4.1 Depression analysis with anxiety as a covariate*

When STAI group was entered as a covariate, there were no changes with regard to recognition accuracy results – there was still no difference in ability to identify emotions between low and high BDI groups and no interaction between group and expression. Similarly there was no change in absolute sensitivity findings – again there were no differences between low and high BDI scoring subjects. The relative sensitivity interaction between expression and group also just remains significant ($p=0.05$) with STAI group as a covarying factor. This suggests that differences exhibited in relative sensitivity to specific emotions (i.e., high BDI subjects are relatively less sensitive to expressions of disgust than low BDI subjects and show a tendency to be relatively more sensitive to sadness) cannot be accounted

for by anxiety effects as measured by STAI group. (See Appendix 4.3 for appropriate F values.)

#### *4.3.4.2 Anxiety analysis with depression as a covariate*

The interaction of expression and group for recognition accuracy scores remains significant ($p=0.014$) with BDI as a covariate – individuals in the high STAI group still make significantly more errors in disgust recognition than those in the low STAI group (Tukey HSD, $p=0.001$).

However, the main effect of group for absolute sensitivity (with subjects in the high STAI group more sensitive overall to all the emotions than subjects in the low group) is no longer significant and just exhibits a slight trend ($p=0.1$). BDI group must therefore be accounting for some amount of the variance in absolute sensitivity between STAI groups. As BDI and STAI scores correlate significantly, $r(73)=0.6$, $p<0.01$, it can not be said that anxiety level (as measured by STAI group) affects absolute sensitivity to expressions. There was no change in relative sensitivity with the inclusion of BDI as a covariate. (See Appendix 4.4 for appropriate F values.)

## 4.4 DISCUSSION

### 4.4.1 BDI and depression

Analysis of the data suggests that individual differences in terms of mood state *do* seem to influence facial expression perception.

Results indicate that although individuals with low and high BDI scores do not differ in overall sensitivity to facial expressions, they *do* exhibit differential responses to particular expressions. The precise nature of these sensitivity

differences was revealed by post-hoc analysis, which suggested that individuals with high BDI scores were relatively more insensitive to expressions of disgust, but showed a trend to be slightly more sensitive to expressions of sadness compared to low scoring subjects. It is notable that differences in pattern of relative sensitivity between low and high BDI subjects remained once anxiety level had been controlled for.

Comparison of current findings with previous research on depression must be qualified by the consideration that subjects in the high BDI group here were not necessarily suffering from a mild form of depression, but might have just been experiencing a 'bad day' or slight negative mood. Indeed division of subjects into low and high groups according to a BDI cut-off score is fairly arbitrary, although scores of greater than 9 are supposed to indicate a mild form of depression (Beck et al., 1988) and such cut-offs have been used in other studies (Persad & Polivy, 1993). In addition, BDI is unlikely to tap very transient fluctuations in mood as subjects are instructed to circle which statement best describes how they have been feeling over *'the past week, including today'*. It is probable that the BDI *does* measure some form of depressive mood tendency in a non-clinical population, which (due to the results following the addition of anxiety level as a covariate) is unlikely to merely represent level of general negative distress. In this respect, it is reasonable to comment with caution on the comparative nature of these results with other studies of clinical groups.

The finding of differential pattern of relative sensitivity across high and low scoring BDI groups is in line with the majority of previous research and the predictions made at the outset. It was predicted that depressed individuals might exhibit a bias to preferentially process negatively valenced emotional information,

i.e., show enhanced sensitivity to negative emotions relative to normal individuals. It was not specified at the outset whether this negative bias would be specific to certain negative expressions (as hypothesised by Bouhuys et al., 1995) or if it would be general to all negative emotions. It was thought likely that sensitivity to expressions of sadness would be affected, although evidence has been inconsistent as to the direction of effect on detection of sadness (as illustrated by several experiments in the literature reviewed earlier, see sections 3.1.3 and 3.1.4).

The tendency shown here for individuals with high depression scores to exhibit increased relative sensitivity in perception of sadness is similar to Bouhuys et al.'s (1995) finding of increased ratings of sadness and rejection in schematic faces in a normal population following a depressed mood induction procedure (MIP). In addition, Mandal and Bhattachayra (1985) found enhanced recognition of sad compared to happy faces in a depressed population. The most probable explanation for enhanced sensitivity to sadness as depression score increases is the fact that this emotion is congruent with the subject's depressed mood state - perhaps rendering other individuals' sad expressions more amenable to perception. Maintenance of depressive affect is liable to result from such selective processing, as attention is focused on negatively valenced aspects of the environment, thus heightening feelings of despair and pessimism. It is noticeable, however, from assessment of recognition accuracy that the high depression group did not have a tendency to mislabel other expressions as sadness when viewing the full emotion at 100%, that is they do not exhibit a bias towards seeing all negative expressions as sadness. (Lack of bias for sadness was not due to ceiling effects, as there were some recognition errors, see Tables 4.1 and 4.2.) Such results - with no differences in recognition ability yet differential relative thresholds required to perceive emotions - give further

justification for the use of this interactive test, which is able to assess more than one aspect of perception.

Depression not only encompasses heightened negative affect but also a decrease in positive affect (Watson et al., 1988). Gur et al. (1992) noted that depressed subjects were biased towards seeing expressions as negatively valenced; they misinterpreted happy faces as neutral and neutral faces as sad. The present study also shows a trend for increased sensitivity to sadness but does not confirm the negative bias to rate happy faces as appearing neutral, i.e., exhibiting decreased relative sensitivity to happiness. The reason why the present study does not detect any change in perception of happy expressions in depressive subjects is at present unclear. It is possible that sensitivity might only be altered in more severely depressed individuals, when negative affect is so extreme that positive cues in the environment are no longer readily detectable as they are so discrepant with mood state. It is possible that this aspect of depressive symptomatology (anhedonia) is not ordinarily represented in a subclinical population and is only prevalent in more severe syndromes of depression. In addition, a criticism that has been directed at the BDI is the lack of inclusion of items related to anhedonic mood state (Coyne, 1994) which is a crucial component for a diagnosis of major depression. It is important to note again that the subjects included in the high depression group in this study were not institutionalised and could only be said to be experiencing mild levels of depressive symptomatology or depressed mood state.

Difference in level of depression might account for the discrepancy between the result for sadness found here and that demonstrated by clinically depressed patients in a study by Bouhuys et al. (1996). Bouhuys et al. demonstrated that *decreased* sensitivity to sad expressions correlated with more stable depressive

periods, i.e., patients who were least likely to rate expressions as displaying sadness at the initial time of testing were more likely to have an unfavourable prognosis over the subsequent 7 months compared to patients who did not show this tendency. All patients studied by Bouhuys et al. were clinically depressed and all were receiving medication. It is therefore possible that the apparent change in sensitivity to expressions of sadness across subclinically and clinically depressed individuals could be a function of depression severity. (Another possibility is that alterations in sensitivity to sadness could be due to blunting effects of medication, although this is improbable as any pharmacological effect reducing responsitivity would be likely to affect perception of all emotions, rather than differentially influencing perception of sadness.)

Individuals suffering from mild and relatively transient forms of depression might be initially overly sensitive to negative mood in others, as this equates with experienced mood state. Decreased sensitivity is possible in more severe clinical conditions, because by this stage familiarisation with negative responses from significant others might have occurred. Reassurance seeking is common in depressives and is likely to lead to rejection from others (Joiner, 1999; Coyne, 1976a). If depressed patients are slower to detect negative responses from others, they will continue to misinterpret social situations, which will decrease the likelihood of clinical recovery.

The finding that individuals with increased depression scores were relatively less sensitive to expressions of disgust than those in the low BDI group was unexpected and receives no apparent explanation from the literature. The disgust response is speculated to have originated as a defence mechanism (and thus would

be expected to be related to anxiety and fear responses), to prevent an animal from consuming noxious stimuli (Rozin et al., 1993). There is no obvious reason why either perception or experience of disgust should be affected in depressed individuals. Haidt et al. (1994) developed a scale assessing general sensitivity to disgust, which examines response to various types of disgust elicitors (e.g., food contamination, body deformity). They found that neuroticism correlated positively with experience of disgust, suggesting a link between enhanced arousal and increased sensitivity to disgust. Symptoms of depression, in contrast, include flattened affect and blunted arousal (American Psychiatric Association, 1994: Diagnostic and statistical manual of mental disorders, 4$^{th}$ edition) which might imply a greater intensity of disgust is required to elicit a disgust response from depressed individuals. Steiner et al. (1993) found that depressed individuals' responses to both pleasant and disgusting olfactory stimuli were less pronounced and of a shorter duration than those of control subjects. The authors speculated that this flattening of response could be a result of inhibitory mechanisms acting on primitive areas in the brain stem, which regulate experience of pleasure or repulsion. The relation between experience, expression and perception of disgust, or any emotion, is unclear, although there are hints that perception of emotion can lead to contagious experience of that particular emotion (Hietanen et al., 1998).

Although it remains unclear why perception of disgust might be affected in depressed individuals, there have been interesting findings in other groups of patients with regard to disgust, which are relevant to note.

More severe impairments in the perception of disgust have been demonstrated in patients suffering from Huntington's disease (Sprengelmeyer et al., 1997a, 1996). These patients exhibited a profound deficit in identification of

expressions of disgust and also showed milder concomitant problems with recognition of anger and fear. Clinical observations that Huntington's patients often have poor personal hygiene standards imply that they might also have difficulties experiencing disgust, which suggests that a more general deficit in processing of disgust is present. Interestingly, this selective impairment in the recognition of facial expressions of disgust has also been reported in Huntington's disease gene carriers (Gray et al., 1997), even though they do not exhibit any of the clinical symptoms of the disease at that stage of the illness. The earliest pathological signs of Huntington's disease are observed in the basal ganglia, implicating this region in recognition of disgust. Fronto-striatal abnormalities present in Huntington's disease might also be evident in depression (A. Young, personal communication), which might explain the sensitivity problems exhibited for disgust in individuals with depressed mood. It is also possible that alteration in serotonergic function seen in depression (see section 2.2) could affect brain regions associated with disgust regulation. Functional imaging could help elucidate whether metabolism in subcortical regions differs during presentation of facial expressions of disgust in depressed and normal individuals. Before undertaking such a study, it would be useful to determine whether the finding in the present study would be replicated in a sample of clinically depressed patients (see Chapter 5).

A recent fMRI study investigating neural response in normal subjects to neutral, fearful and disgusted facial expression stimuli (Phillips et al., 1997) revealed that selective activation in anterior insular cortex occurred in response to expressions of disgust, which was replicated in a further study (Phillips et al., 1998). These findings tie in with the idea of associative learning in experience and perception of disgust, as the anterior insular cortex is also known to respond to offensive tastes,

implying that the sight of disgust in another is related to personal experience of disgusting stimuli and feelings of repulsion.

Obsessive-compulsive disorder (OCD) patients also demonstrate a selective impairment in recognition of disgust (Sprengelmeyer et al., 1997b). This seems counterintuitive in some respects, in that one might predict that individuals suffering from OCD would be *more* responsive to expressions of disgust since extreme feelings of disgust are symptomatic of many forms of the disorder. A possible explanation for these findings could be the loss of correlation between self-experience of disgust in OCD and perception of others expressions of disgust. OCD patients are liable to be highly disgusted by stimuli that do not elicit disgust in others and thus the association between their own feelings of disgust and visual expressions of disgust in others becomes weakened. It would seem important in future studies to establish whether this deficit is moderated by type of OCD suffered – i.e., whether a disgust impairment only arises in individuals who are suffering from contamination-type anxieties as opposed to those whose OCD is manifested by checking rituals.

Another psychopathological group, which might show a differential pattern of sensitivity to facial expressions of disgust, is bulimic individuals. People suffering from bulimia have a distorted perception of their own figure and resolve feelings of being overweight by vomiting up food ingested in binge episodes. As taste and vomiting are closely related to the core concept of disgust as a rejection response, it is quite possible that perception of disgust might be altered in bulimics. It is of interest that depression and bulimia often co-occur (Willcox & Sattler, 1996). It might also be of significance that serotonergic function is altered in bulimia (Brewerton, 1995; see section 7.3.1). As yet any alteration of disgust perception in

bulimics is entirely speculative, however, and requires empirical substantiation (see Chapter 7).

### 4.4.2 STAI and anxiety

Again, as with the link between BDI and depression, it cannot be explicitly stated here that individuals with elevated STAI scores suffer from an anxiety disorder, just that they might exhibit increased tendencies to feel anxious.

Level of anxiety did appear to exert an influence on accuracy of identification of facial expressions, in that subjects in the high STAI group were significantly worse at recognising disgust than subjects in the low scoring STAI group. On closer inspection of error type, it was noted that disgust was consistently misidentified as anger. These two expressions have been reported to be commonly confused (Ekman & Friesen, 1976; see also Young et al., 1997), but not usually to such a degree. The high rate of correct classification of anger that is demonstrated by high STAI scoring subjects might therefore be a consequence of a slight response bias to perceive expressions as disapproving or threatening and could reflect a tendency for anxious individuals to be overly 'tuned-in' to recognise anger directed at them. (It should be noted though that expressions other than disgust were not misidentified as anger, implying that the high error rate might be something to do with the morphological similarity between these two emotions.) This bias might cause them to misinterpret an expression as anger if it is ambiguous, which could in turn exacerbate feelings of anxiety. These selective differences in disgust recognition remained once BDI group status had been factored out.

The main effect of group that was revealed in the absolute sensitivity analysis, with individuals in the high STAI group being more sensitive to all

emotions than those in the low group, must be viewed with some caution as significance of this finding disappeared once depression had been partialled out as a factor. Level of depression must therefore have been accounting for some of the effect and so it cannot be concluded that change in absolute sensitivity level is due to anxiety tendencies alone. There was a high degree of covariance between anxiety and depression measures, which could in part be a consequence of many of the items on the STAI being highly related to depression (Vredenburg et al., 1993). For example, asking subjects to rate the degree with which the statements *'I feel like a failure'* or *'I am happy'* generally apply to them. It would seem more intuitive, however, that increased sensitivity (due to selective attention) would be found to be present only for *specific* expressions, as opposed to all expressions, in a clinically anxious group.

In the relative sensitivity analysis there were no significant differences between individuals varying in anxiety level though, implying anxious subjects perceive facial expressions in an equivalent manner to non-anxious subjects. Lack of any significant difference between anxiety groups could be a consequence of the relatively lax criterion used for inclusion in the high STAI group, due to the paucity of extreme anxiety scores in this sample. It is possible that expressions that suggest the presence of threat to the perceiver, such as anger and fear, might elicit differential sensitivity in more severely anxious individuals. Support for such a prediction comes from experiments demonstrating interference effects or selective attention for threat-related stimuli in anxious individuals (Maidenberg et al., 1996; Mogg et al., 1993; MacLeod et al., 1986; Mathews & MacLeod, 1985).

In addition, it might be important to differentiate between types of anxiety when examining the psychopathology of facial expression perception. Particularly

relevant to the study of expression processing might be the anxiety disorder social phobia. Fear of criticism from others is the primary concern of the social phobic, which leads to anxiety in social situations and could result in misinterpretation of expression cues. For example, a social phobic could interpret another individual as being extremely angry with him when in fact the other individual is only expressing mild annoyance, thus heightening feelings of anxiety in the phobic. Sensitivity might also be altered for expressions of disgust in social phobics, as disgust can imply disapproval.

In a recognition memory experiment (Lundh & Ost, 1996), subjects rated pictures of facial expressions as to the degree to which they appeared *'critical'* or *'accepting'*. After a brief interval the subjects were unexpectedly asked to participate in a memory task. Although social phobic subjects did not initially encode more of the faces as appearing critical than normal subjects, they *did* exhibit a memory bias for the faces they had judged as critical. Control subjects, in contrast, showed a slight recognition bias for 'accepting' faces. It might be interesting therefore to examine sensitivity to different facial expressions in this patient group.

## 4.5 STUDY CONCLUSIONS

The results have demonstrated that individuals experiencing mildly depressed mood (if not necessarily a mild form of depression) differ in relative sensitivity to expressions compared to individuals not reporting depressed mood. In contrast, level of anxiety as assessed by the STAI did not appear to have an effect on sensitivity in the current study. It is probable that these differences in sensitivity to facial expressions influence the way in which an individual suffering from depressed mood interprets and responds to others in a social environment. If awareness is heightened

towards negative cues (i.e., enhanced sensitivity to expressions of sadness relative to others), then depressed affective state is likely to be maintained which could result in rejection from others and withdrawal from social groups. In addition, if depressive mood state causes an individual to be slower at detecting disgust directed at him/her, such an individual is likely to continue to respond inappropriately and might therefore be avoided in future social interactions.

However, before any inferences can be drawn with regard to current theories of depression, this study must be replicated with a group of clinically depressed patients to establish whether perception of facial affect differs in kind or merely in degree between mildly and severely depressed individuals (see Chapter 5).

## 4.6 METHODOLOGICAL COMMENTS

The benefits of having used the interactive morph technique are clear from the data: even though there were no significant differences across BDI groups in expression identification when the expressions were viewed at 100%, there *were* significant differences in the relative intensity of expression required to classify it as detectable. The use of more than one index of performance (e.g., percent correct, sensitivity) when investigating expression perception in different subject groups was also endorsed by Gur et al. (1992). Difficulties in expression perception seen in individuals suffering from depressed mood do not therefore appear to lie with misidentification of different expressions; rather, these individuals seem to be differentially sensitive to certain emotions, for example they tend to be over-responsive to mild expressions of sadness compared to non-depressed individuals.

It might be useful to compliment this study with a test assessing categorical perception of expressions in depressed individuals. Previous research investigating

how individuals sort stimuli into discrete categories has shown that perceptual boundaries between categories are very abrupt, for example the sudden perceived change in colour across constant changes in wavelength. Sensitivity is enhanced for discrimination between stimuli straddling category boundaries compared to stimuli within any one category, even though the actual physical difference between stimuli is constant. Several research groups (Young et al., 1997; Calder et al., 1996b; Etcoff & Magee, 1992) have demonstrated that discrete categories exist at a perceptual level for facial expressions, which suggests that the perceptual system is 'tuned in' to specific facial feature combinations representing basic expressions. As sensitivity to different expressions is altered in individuals experiencing depressed mood, presumably there will be a corresponding shift in category boundary. It would therefore be interesting to note whether there was a difference in threshold for categorisation between normal and depressed individuals, by presenting static expression morphs under a forced-choice staircase procedure (Wetherill & Levitt, 1965).

## 4.7 INTERIM DISCUSSION

### 4.7.1 Subclinical versus clinical depression

There is a great deal of controversy over the continuous nature of depression and whether so-called 'subclincal' depression is simply a milder form of the illness or whether it encompasses an affection of an altogether different nature, i.e., is just dysphoric mood or general negative affect. Caution must be exercised in attempting to equate mood state of individuals with elevated depression scores on the BDI with clinically depressed individuals as one cannot assume those individuals are just experiencing milder forms of the same syndrome.

Many of the theories and models posited in the literature to-date with regard to depression actually relate to findings from individuals suffering mild levels of psychological distress, which may or may not be valid for clinically diagnosable depression. It is clearly important to establish the relevance of examining depression levels in non-psychiatric populations (with the majority of studies to-date having been conducted on student populations - presumably in part due to accessibility) as the supposed continuity of depression has not by any means been unequivocally determined. Until the nature of depression is more definitely ascertained, findings from such populations must be regarded with much more reservation as a basis for general research and theories on clinical depression.

Coyne (1994) examines at length the potential problems associated with the use of research conducted on non-clinical student populations exhibiting elevated self-report depression scores as a basis for models of clinical depression. He believes that high levels of distress and diagnosable depression are distinct concepts and that the assumption that the former is a milder form of depression makes light of the emotional, psychological and social costs and associated symptoms of severe depression. In such cases, any correlation that is reported between depression and another variable (for example a predicted causal factor such as bereavement) becomes unnaturally inflated and assumes unnecessary importance when individuals with elevated depression self-report scores are used as the sample for depression. Coyne uses the example of poverty as a potential causal factor for depression. Poverty cannot account for much of the variance in predicting occurrence of depression in that there is much higher incidence of poverty than depression in the general population, i.e., most people who are poor do not go on to develop depression although many might experience some distress. Theories proposed on the

basis of such investigations cannot therefore be said to be applicable to clinical depression.

The sampling procedures used in many studies have also been criticised by Coyne (1994) when normal subjects are excluded from analyses, to enable equal subject numbers, on the basis of reporting what could be high levels of negative affect or distress *in the absence of* actual depression. The resulting sample is therefore unrepresentative of the general population. He believes that the extensive costs endured by sufferers of clinical depression are being trivialised by the direct comparison being made between them and students experiencing mild distress. In addition, Coyne reports that depressives themselves *can* differentiate between mild, irritating daily stressors and severe depressive episodes.

Other researchers *do* believe that student populations are valid to study when examining depression, albeit with the caveat that caution must be exercised in generalising from any findings. For example, Seligman (1978), amongst others, warns against researchers underestimating the distress experienced by non-psychiatric individuals (whether equitable to a mild form of depression or not). If subclinical depressive symptomatology is predictive of major depression (which appears to be the case) then it is not only important to examine distress in non-psychiatric populations in its own right but also to help understand more about the nature of depression development. Nolen-Hoeksema et al. (1992) have reported the significant negative effect moderate levels of depression have on both adults and children, with regard to both work and interpersonal ability, the persistence of such episodes and also the high risk factor they pose for the development of major depression. Such results indicate the importance of studying moderate levels of

depression in non-psychiatric populations, regardless of the outcome of the debate as to the continuity of depression.

The importance of studying students in particular is emphasised by Vredenburg et al. (1993) as the transition to University life involves a variety of academic and interpersonal stressors. Perhaps accordingly, a high rate of suicide and suicide ideation is reported in students - 50% higher than in similarly aged non-students, Beck and Young (1978). The link between depression and suicide is well-documented (Lewinsohn et al., 1986; Pokorny, 1964). The student population is as susceptible as the general population to many vulnerability factors for depression, such as low self-esteem - particularly as students are in general an advantaged group and therefore negative self-comparison with others could easily occur in individuals with such tendencies. In addition, investigation of influences on depressive symptomatology in students might facilitate understanding of the development of depression. Sorenson et al. (1991) reported that over half of the depressed individuals they sampled had experienced a depressive episode prior to the age of 25 - a period which approximates the average student age.

Vredenburg et al. (1993) endorsed the use of correlational designs when investigating student populations rather than the more arbitrary division of subjects into non-depressed and depressed groups. This allows an examination of factors influencing *depressive symptomatology*, as opposed to major depressive syndromes. An underlying assumption evident in such a correlational method is that any changes present will differ in degree along a continuum rather than in kind.

Coyne (1994) also warns against the specificity of self-report methods in screening for depression in the general population, in that the degree of self-reported

depression is much greater than the actual incidence (of around 5-10%) as assessed more objectively by trained clinicians. Discrepancy is common between subjective self-reports and objective clinical estimates with regards to many health problems (Watson & Clark, 1984). For example, Hammen (1980) reported that only 5 of 34 student subjects with elevated BDI scores were assessed by a clinician as having probable major depression 2 to 5 weeks later. It is therefore clear that elevated BDI scores do not necessarily equate to diagnosable depression outside a clinical population. Other forms of distress, which do not meet criteria for depression, could account for high scores. Negative affect can bias and exaggerate symptom reporting (Blaney, 1986; Bower, 1981). Conversely, clinician reports are often necessarily based on scant and possibly idiosyncratic examples of an individual's behaviour. In addition there can be inconsistencies both between and within interviewer ratings. Ideally both self-report and independently rated questionnaires should be used when assessing psychiatric state in the general population for purposes of subject classification. Such methods (particularly the use of trained interviewers) are usually impractical however, so the use of more than one self-report measure or the re-administration of the same self-report schedule over a period of time would also be feasible approaches in determining group status.

Vredenburg et al. (1993) and Flett et al. (1997) counter Coyne's (1994) and Gotlib's (1984) suggestion that elevated scores on self-report questionnaires merely represent high negative distress or general psychopathology as opposed to specifically demonstrating depression. They indicate that many of the indices used to examine other constructs, e.g., anxiety, are intrinsically confounded with depression. Vredenburg et al. (1993) claim that the high correlations frequently reported particularly between anxiety and depression self-report measures are often a

consequence of many items on the 'anxiety' questionnaire, e.g., the STAI, explicitly applying to depression as opposed to purely examining levels of anxiety. An example of questions relating to depression being, *'I feel like a failure'* or *'I wish I could be as happy as others seems to be'* and solely to anxiety, *'I feel nervous and restless'*. In addition, anxiety and sociopathy (Rohde et al., 1991) are commonly found to co-exist with major depression in psychiatric populations so in this respect non-psychiatric depressed populations cannot be said to differ. The issue of ensuring specificity of pure depression in a subclinical group then becomes less significant. The addition of non-depressed psychiatric control group would be beneficial to most studies to help resolve whether any results found are purely due to depressive state.

Flett et al. (1997) review evidence for both sides of the continuity debate but unlike Coyne (1994) and Gotlib (1984) they conclude that *"...most relevant literature in the depression field is consistent with the continuity perspective"*. In particular, phenomenological evidence was examined which revealed that substantial impairment (occupational, social) could arise as a consequence of subsyndromal (subclinical) depression. Flett et al. also reviewed literature on etiological continuity, with many studies reporting that subthreshold depression is a significant risk factor for major depression at some future point. Vulnerability also seems to be specific for depression as opposed to others forms of psychopathology (Gotlib et al., 1995). One variable that has been shown to precipitate the development of more severe depression in such individuals is the occurrence of major life events (Brown et al., 1986).

Flett et al. (1997) concluded by recommending that a two-factor model, accounting for both continuities and discontinuities, should be applied to future

research as both quantitative and qualitative differences are likely to be present across varying levels of depression. An additional suggestion was made that a consensus should be reached as to classification of subsyndromal depression, as lack of agreement will necessarily affect comparability of studies and results.

### 4.7.2 Difficulties with BDI use

In spite of the widespread use of the BDI as a brief and straightforward measurement of predisposition to depression, the validity of the BDI has actually only been systematically examined in clinically depressed populations (Beck et al., 1988). Similarly criterion scores used to estimate severity have only been shown to relate to diagnosable depression. Beck himself warned against extrapolation of results from non-clinical student populations with high BDI scores to clinically depressed psychiatric populations.

One difficulty with the BDI involves the tautology of the questions in that high scoring on one item is likely to result in elevated scores on semantically related questions (i.e., some items are rather redundant and might lead to circular response patterns). For example, items 3 and 7 describe failure and disappointment in oneself respectively (see Appendix 4.1). The BDI also focuses primarily on psychological distress whilst neglecting co-occurring somatic symptoms more commonly associated with severe depression. This results in the BDI being weighted in favour of the acquisition of elevated scores even if only mild distress is experienced. Another criticism that can be levelled at the BDI is that it does not really encompass the feelings of apathy and anhedonia, which are often crucial symptoms - in addition to sad mood - in the classification of depression. The importance of symptom duration (which must be greater than 2 weeks to satisfy clinical criteria) is also not

adequately catered for by the BDI as the instructions only ask the individual to comment on how they have been feeling over *'the past week, including today'*.

The BDI has been demonstrated to have both high internal consistency and test-retest reliability. However, Coyne (1994) suggests the former could be a consequence of an individual scoring zero (i.e., not depressed) on a specific item being likely to score zero on most other items. High test-retest reliability in turn could be due to the fact that a large decrease in total score is impossible at retest for individuals initially exhibiting a low total score. Higher scores have been shown to be fairly unstable, with the majority of individuals from a normal population who initially scored highly returning scores below criterion when retested 1-3 weeks later (Zimmerman, 1986; Sacco, 1981; Hammen, 1980). Coyne concludes that the most probable reason for instability of elevated scores in a non-clinical population is the *"... mild and transient nature of much of the distress in a student population"*. Therefore, students who are considered as 'depressed' according to a single BDI estimate are, in the majority of cases, only liable to be suffering from relatively mild and short-lived forms of distress. The causes and consequences of such phases are likely to be different from any possible antecedents and outcomes of depressive episodes. (For example, they are unlikely to be due to parenting difficulties or marital or economic stresses.) Students should not therefore, in his opinion, be used as a comparative sample on which to base clinical models. If elevated scores on the BDI *did* relate directly to depression, the general incidence rates would be significantly greater than are actually the case. Such a statement does not however preclude the occurrence of clinical depression in a small percentage of the student population, but merely suggests that elevated BDI or self-report scores are not a sufficient prerequisite for diagnosis. The high rate of false positives detected by the

BDI is characteristic of most self-report scales (measuring varying constructs) and results in a low predictive pattern. Type of assessment (clinician versus self-rating) therefore plays a crucial role as to the validity of directly comparing subclinical and clinical models of depression.

To ensure more accurate diagnoses of the presence of stable predisposition to depression in students, it is advisable to use more than one measure of depressive symptomatology. For example, the Diagnostic Interview Schedule (DIS) which can be given by lay interviewers or the Inventory to Diagnose Depression (IDD) if a more stringent assessment is required. (Obviously a structured interview administered by a trained clinician would be preferable but this is usually not practically possible). It does not seem expedient to merely increase the cut-off value for mild depression on the BDI in order to divide non-psychiatric individuals into non-depressed and depressed groups more rigorously as such a procedure will diminish the sensitivity of the scale and generate a non-representative sample. In addition, re-administration of the questionnaire on a separate occasion might help prevent against the inclusion of individuals merely experiencing a 'bad day' at time of testing in a depressed group. Fluctuation of BDI scores across time has been demonstrated by Sacco (1981) amongst others (although this could in part be a consequence of memory for individual items). Again, a more stable estimate of the depression construct can be obtained if more than one measure of depression is taken.

Other difficulties arise in classifying individuals on the basis of self-report scores or minimal information. For example, although depression might not be current at time of testing, previous episodes of depression might have occurred so an individual could be incorrectly excluded or equally, individuals might be eliminated

who have a predisposition to depression but have not yet encountered significant negative stressors to trigger the disorder. (The latter is more likely to affect research on young student populations than the former.)

# CHAPTER 5: CLINICAL DEPRESSION (STUDY 2)

## 5.1 INTRODUCTION

Based on previous research (see section 3.1) and data trends present in a subclinical population (see Chapter 4), it seems probable that a depressed individual will process facial expressions of emotion differently from a non-depressed individual. One cannot, however, extrapolate findings directly from subclinical samples and assume similar results will be evident (and stronger) in a clinical population, as it remains unclear whether mild and major depressive syndromes differ in kind or merely in degree (see section 4.7.1). This chapter therefore reports a similar study to that in Chapter 4 using a clinically diagnosed depressed population, in order to investigate more fully the effect of depression on facial expression perception.

There were a couple of notable methodological differences between the two studies. Firstly, 150%-caricatured morphs were used to study clinically depressed patients, as opposed to 100% morphs, to enhance recognition accuracy (see Calder et al., 1997). Using 150%-caricatured endpoints also helped guard against floor effects in sensitivity, which appeared to arise in pilot testing as patients required around 90-100% of the expression for detection (using the veridical 100% image as the endpoint), which would make differentiation in sensitivity across the emotions difficult. In addition, in this study each of the 18 morphed continua was tested twice as opposed to 4 times, which was the case in the previous study. An independent pilot study has revealed that the amount of trial repetition does not appear to effect responses (unpublished findings).

It was predicted that data from the clinically depressed sample would differ quantitatively rather than qualitatively from that of the mildly depressed sample. On

the basis of the findings in the previous subclinical study, it seemed probable that depressed individuals would be relatively more sensitive to sad faces, but less sensitive to disgust than non-depressed individuals. In addition, from the general predictions outlined in section 4.1.1, one would anticipate that sensitivity to happy faces would be altered in a depressed population with the likelihood that depressives would be relatively less sensitive to expressions of happiness in others compared to a non-depressed group. It was also possible that subject sex might influence differences between the depressed groups owing to trends exhibited in the previous study (see section 4.3.2.3).

## 5.2 METHOD

### 5.2.1 Participants

35 individuals clinically diagnosed as suffering from depression constituted the depressed group. This group comprised 19 males and 16 females with an age range of 18-73. Diagnoses ranged from mild depressive episode without somatic syndrome (associated physical symptoms) to severe depressive episode with psychosis (see Appendix 5.1 for diagnostic details). The majority of patients were recruited at an outpatient clinic following psychiatric interview but several were procured from inpatient psychiatric wards. 3 of the patients had intractable depression and were awaiting anterior capsulotomy surgery (see Chapter 6). Patients were considered for inclusion in the study if depression was their primary diagnosis. (1 of the surgical patient's primary diagnosis was Obsessive-Compulsive disorder, but she was also significantly depressed with bipolar disorder.) Participation in the study was entirely voluntary and did not affect the patient's treatment in any way. Pharmacological treatment was not controlled for. (There were 3 drug-free patients,

and 20 were taking only antidepressant medication (15 were taking an SSRI, 3 a Tricyclic, and 2 an SNRI). Of the remainder, 1 patient took SSRI plus low-dose benzodiazepine (Diazepam equivalent of up to 10mg per 24 hours) 3 were taking a combination of an antidepressant and lithium salts, 2 were taking lithium plus low-dose benzodiazepine, and 2 took a combination including an antidepressant plus an atypical antipsychotic. One patient was taking St John's Wort only. See Appendix 5.2 for complete medication details.) 3 patients (2 male, 1 female) were excluded from the analysis on the basis of: English not being the first language (invalidating the mood questionnaire), incomplete data (because the patient became too tired) and the presence of concomitant personality disorder. This gave a final total of 32 subjects (17 male, 15 female) in the clinically depressed group.

45 control subjects of a similar age range (21-68 years) were tested (21 male, 24 female). Subjects satisfied criterion for inclusion in the control group if their score on the Beck Depression Inventory (BDI) was less than 10. A stringent cut-off was used to ensure that individuals were not included if they were suffering from even slight negative affect when completing the study. 13 of the 45 subjects were consequently classified as mildly depressed on the basis of BDI scores of greater than or equal to 10 and were therefore excluded from the control group. The final control group therefore consisted of 32 subjects (17 male, 15 female) with an age range of 21-59 years (see Appendix 5.3 for general subject information).

All subjects gave informed consent to participate in the study.

Ethical approval for this study was obtained from both the Ethics Committee for the School of Psychology, University of St Andrews and also from the Tayside Committee on Medical Research Ethics for the recruitment and testing of control and clinically depressed individuals respectively.

### 5.2.2 Materials

The stimuli used were identical to those in the study in Chapter 4 (see section 4.2.2).

### 5.2.3 Interactive morphs

The software used was identical to that in study 1 (see section 4.2.3) although the program was converted for use on portable PC (to facilitate testing in hospital settings).

Firstly each veridical (100%) expression image was subjected to a 50% shape caricature (enhancing the shape changes already present between 0% and 100% images) which created the 150%[1] expression image. Texture is not warped between 100-150% as this can result in an unrealistic image. Shape therefore undergoes a 150% transform but texture only a 100% change across the 0-150% images. (To illustrate, the original 100% image, which becomes an intermediate morph between 0-150%, remains represented at its original 100% shape difference but only undergoes two thirds change in texture, see Figure 5.1.) This prevents the intensity transition in texture which would result from the creation of two separate morphs from 0-100% and 100-150% which would maintain texture constant at 100% between the 100-150% steps.

[1] 150%-caricatured images were chosen rather than 100% images because pilot testing (on 2 clinically depressed inpatients and 1 schizophrenic inpatient) had revealed that patients required a high percentage of emotion (e.g., around 90%) to first perceive it.

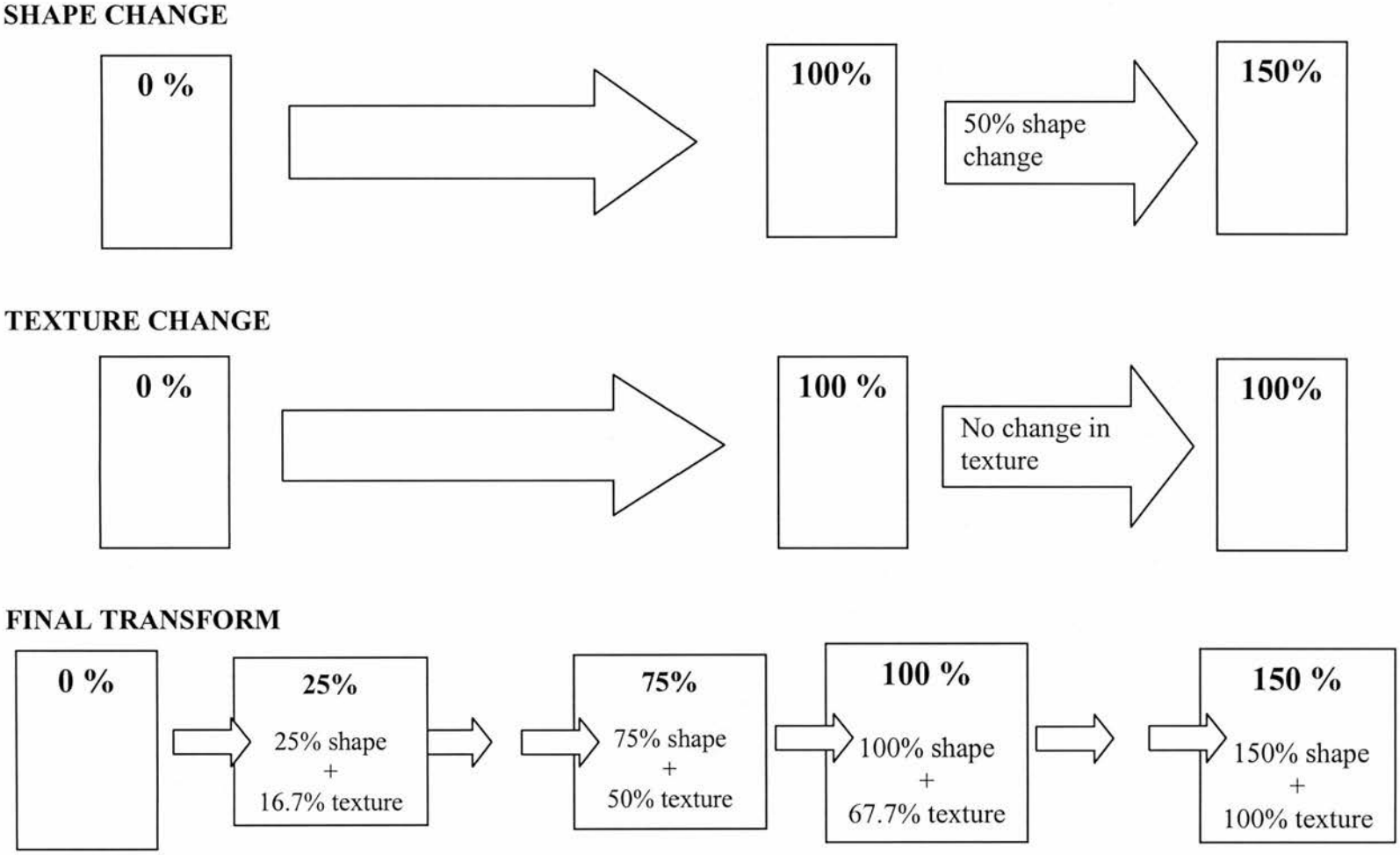


**Figure 5.1: Illustration of shape and texture transformation in the morphed images between the neutral (0%) expression image and the caricatured 150% expression image endpoints.**

A set of 31 individual morph images were created at 5% interval steps from 0% up to the 150%-caricatured endpoint. The morphs were created by taking a percentage of the difference in shape and texture change between the two endpoint images - the neutral (0%) and the caricatured (150%) image. These 31 morphed images were then masked for display in Internet Explorer (see Figure 5.2) as an animated movie. Masking is achieved by replacing the background around the outline of the face with a black mask and prevents unnatural blurring of hair, ears and neck due to the morphing process. The animated movie therefore consisted of 31 sequential frames in 5% steps from 0-150% expression, shown at a constant viewing speed of up to 10 frames per second. (The program was written in Javascript.)

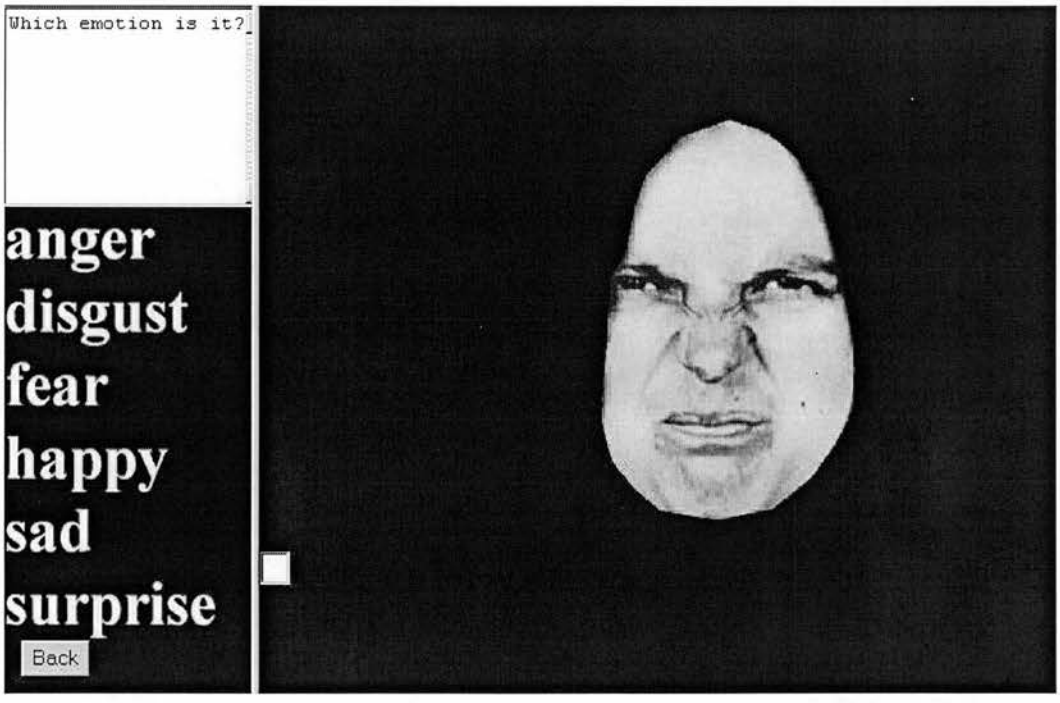


**Figure 5.2: An example of the screen layout shown to subjects. The endpoint of a movie (150%-caricatured expression of disgust) is displayed and the subject is required to choose which of the 6 emotions on the left of the screen corresponds with the expression being portrayed in the right half of the screen. This tests recognition accuracy. (Not to scale.)**

### 5.2.4 Procedure

Following completion of a consent and information form (see Appendix 5.4), subjects were asked to fill out a standard clinical questionnaire - the Beck Depression Inventory (see Appendix 4.1), which assesses an individual's depressive state. All subjects completed the BDI *before* commencing the facial emotion task.

The interactive test was similar to that used in the initial study (see section 4.2.4). Firstly, subjects were shown an animated movie that always commenced with a neutral expression (i.e., all facial muscles relaxed) and gradually morphed into one of six different expressions. (The movie always advanced in this direction, i.e., the 150% expression animating back to the 0% neutral expression was not presented, and subjects were aware that a neutral image would always appear first.) Subjects were asked to identify the expression displayed (at 150%) by selecting the appropriate emotion label (with the cursor) from the choice of six on the computer screen (see Figure 5.2). The experimenter would select the emotion according to the subject's verbal response if the subject had difficulty working the computer mouse (which was fairly common). Subjects were allowed to view the movie as many times as necessary. No feedback was given at any point as to response accuracy.

Following identification, subjects were shown (in practice trials) how to manipulate the amount of expression portrayed (starting from neutral) between 0%-150% by pressing the '>' key to increase the amount of expression displayed and the '<' key to decrease the expression towards neutral, moving frame-by-frame through the images. One key press equated a 5% intensity transition. All subjects *had* to press these keys themselves and were directed to tap the keys lightly to prevent the movie continuing to the end, which occurred if the keys were continually held

down[2]. Subjects were instructed to find the point along the movie where they could first reliably classify the emotion, e.g., as anger. They were encouraged to do this as though they had not previously viewed the expression (as if they did not know what to expect) and were looking at it afresh. This assessed subjects' sensitivity to different expressions. Once subjects were satisfied this point had been found they had to double press the space bar, which terminated the trial and started the next trial.

There were 18 different continua (three examples of each of the six expressions) presented in a pseudo-random order in the test block. The block was then repeated (same stimuli in reverse presentation order). There were therefore 36 trials in total. There were also six practice trials (using the remaining Ekman identity, female - c) to acclimatise subjects to the procedure.

## 5.3 RESULTS

### 5.3.1 Comparison of age and BDI score for the two groups

The average age of the control group (n=32) was 37.2 years (SE 1.5) compared to 43.7 years (SE 2.1) in the clinically depressed group (n=32). A between-subjects t-test (equal variance) revealed a significant difference in age, $t(62)=-2.5$, $p=0.015$, with the depressed group being slightly older. This unbalanced age distribution is in part due to the fact that many of the older control subjects tested had to be excluded as they exceeded 9 on BDI score.

The average control group BDI score was 3.4 (SE 0.5) with a range of 0-9. In contrast the average BDI score in the clinically depressed group was 29.7 (SE 1.5) with a range of 13-49. This difference in BDI scores was shown to be highly

[2] The experimenter observed the subject's progress throughout the test session to ensure this did not occur.

significant using a between-subjects t-test (unequal variance), $t(38.0)^{3}$=-17.1, p=0.001.

(See Appendices 5.1 and 5.3 for details of individual subject's age and BDI score.)

### 5.3.2 Recognition accuracy

A 2*2*6 mixed-design ANOVA, with subject group (control versus clinically depressed) and sex as between-subject factors (2 levels each) and expression as the within-subjects factor (6 levels), was conducted on the recognition accuracy data.

There were no significant between-subjects effects. There was no significant difference, $F_{(1,60)}$=3.2, p=0.08, between the amount of errors made by control and clinically depressed subjects, although there was a trend for clinically depressed patients to make slightly more errors overall (see Figure 5.3). There was also no significant difference in recognition accuracy according to sex of subject, $F_{(1,60)}$=1.3, p=0.3, and the subject group by sex interaction was also non-significant, $F_{(1,60)}$=0.1, p=0.7.

There was a significant effect of expression on accuracy, $F_{(3.5,212.6)}{}^{4}$=10.2, p=0.001, indicating some expressions were easier to identify for all subjects than others (see Figure 5.3). The order of ease of identification was happiness 98.2%, anger 95.1%, disgust 92.7%, sadness 90.4%, fear 87.2% and surprise 83.6%. These figures are comparable to the original identification record of Ekman and Friesen (see Table 5.1), although recognition testing used 150%-caricatured images here as opposed to the veridical 100% images used by Ekman. Fear and surprise were

[3] Corrected degrees of freedom are used in independent t-tests with predicted unequal variance.

commonly confused which accounted for the majority of errors for these expressions. The relatively high error rate for surprise (incorrectly recognised as fear) is predominately due to misidentification of 1 particular face. Depressed subjects also mistook fear for anger (approximately 7% of the time) which did not occur with control subjects. The difference in error pattern between the two groups is illustrated in Table 5.2.

The expression and depression group interaction did not reach significance, $F_{(3.5,212.6)}=0.9$, $p=0.5$, implying that the pattern of recognition accuracy was comparable across the two groups. The expression and sex interaction was also non-significant, $F_{(3.5,212.6)}=1.5$, $p=0.2$, as was the 3-way interaction of expression, sex and depression group, $F_{(3.5,212.6)}=0.4$, $p=0.8$.

| | CURRENT RESULTS | | EKMAN RESULTS | |
|---|---|---|---|---|
| | % correct | SE | % correct | SE |
| ANGER | 95.1 | 1.3 | 92.0 | 8.0 |
| DISGUST | 92.7 | 1.7 | 94.3 | 2.2 |
| FEAR | 87.2 | 2.3 | 86.3 | 3.8 |
| HAPPINESS | 98.2 | 0.7 | 100.0 | 0.0 |
| SADNESS | 90.4 | 2.1 | 92.0 | 1.2 |
| SURPRISE | 83.6 | 1.9 | 92.7 | 1.7 |

Table 5.1: Identification accuracy of the 6 emotions for control and clinically depressed subjects (n=64): a comparison of current results with Ekman & Friesen's norms[5] (1976) (n is not specified, although is referred to as a small group).

[4] N.B. The Greenhouse-Geisser correction is used throughout, as the within-subjects factor has more than 2 levels.

[5] Norms were given for individual expression identities (Ekman & Friesen, 1976), which have been averaged here across the 3 relevant identities used for each expression.

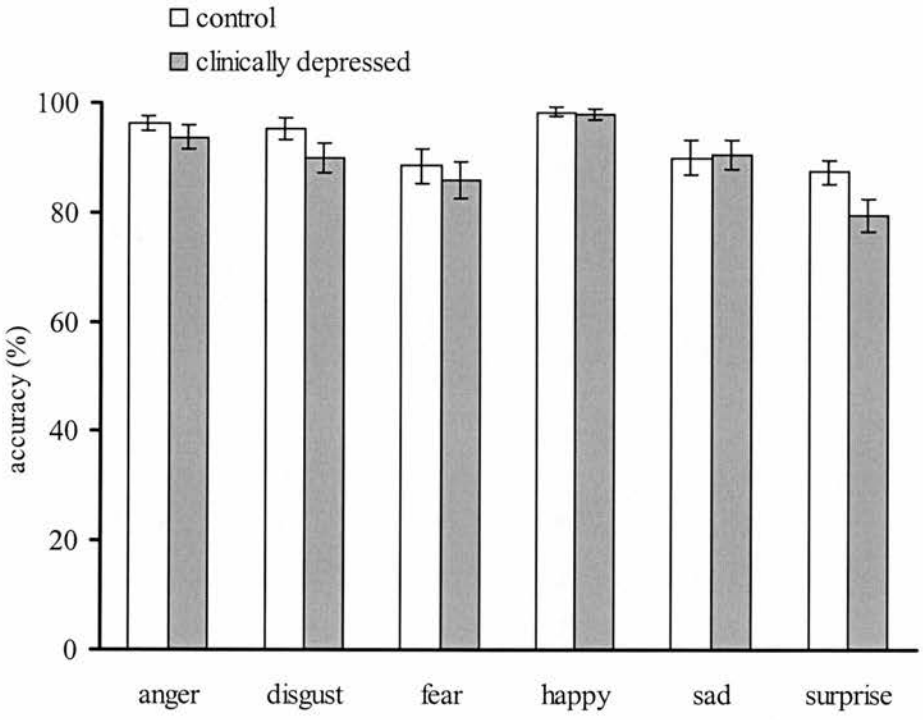


**Figure 5.3: A comparison of recognition accuracy (mean and SE) for the 6 emotions between control and clinically depressed groups (n=32 in each group).**

| ACTUAL EMOTION | MISINDENTIFIED AS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | anger | | disgust | | fear | | happy | | sad | | surprise | |
| | ANGER | | | **2.6** | - | **3.1** | 2.1 | - | - | - | - | **0.5** | 0.5 |
| | DISGUST | **9.9** | 3.6 | | | - | - | - | 1.6 | - | - | - | - |
| | FEAR | **6.8** | 1.0 | - | 0.5 | | | | - | - | - | **7.3** | 9.4 |
| | HAPPY | - | - | - | - | - | - | | | - | - | **2.1** | 0.5 |
| | SAD | **0.5** | - | **2.6** | 3.6 | **5.2** | 5.7 | - | - | | | **1.0** | 0.5 |
| | SURPRISE | **1.0** | - | **0.5** | 0.5 | **16.7** | 12.5 | **2.1** | - | - | - | | |

Table 5.2: Error pattern for clinically depressed (bold) and control (normal type) groups. The value given is the average percentage of errors made across the group of subjects.

### 5.3.3 Absolute sensitivity

As in Chapter 4, trials in which subjects had misidentified the expression were discarded from the sensitivity analysis, as it was important to determine that subjects were using the correct facial cues to detect the appropriate emotion. Firstly the response output, which was the frame number chosen, was converted into percentages (where frame 1 was equal to 0% and frame 31 to 150%). These percentages constituted absolute sensitivity level, which could be between 0%-150%.

Again a 2*2*6 mixed-design ANOVA was conducted with subject sex and group (control versus clinically depressed) as between-subjects factors and expression as the within-subjects factor. Clinically depressed subjects were shown to be considerably less sensitive than control subjects across *all* the emotions with a main effect of group, $F_{(1,60)}=11.5$, $p=0.001$, i.e., clinically depressed subjects required a higher percentage of any expression morph to first detect it as representing a particular emotion (see Figure 5.4). There was no main effect of sex, $F_{(1,60)}=0.7$, $p=0.4$. There was a non-significant trend for an interaction between sex and depression group, $F_{(1,60)}=3.5$, $p=0.067$. Post-hoc (Tukey HSD) tests revealed that male subjects exhibited greater differences in sensitivity according to depression group ($p=0.002$) than female subjects did ($p=0.7$), although the clinically depressed group had a greater absolute threshold than the control group for both sexes (see Figure 5.5).

A highly significant within-subjects main effect of expression was revealed, $F_{(4.0,242.1)}=40.4$, $p=0.001$, showing that some emotions are easier to detect than others for all subjects. Sensitivity to happiness is highest (easiest) at 54.9%, followed by fear at 68.9%, surprise 71.4%, anger 71.8%, disgust 71.9% and finally sadness at 82.3%. The interaction of expression and depression group was significant, $F_{(4.0,242.1)}=2.6$, $p=0.035$, indicating the pattern of sensitivity to particular emotions is different between the two depression groups. The pattern of sensitivity for each group across the emotions can be more easily examined once overall sensitivity level has been controlled for and justifies the relative sensitivity analysis (see section 5.3.4). The interaction of expression and sex was not significant, $F_{(4.0,242.1)}=2.1$, $p=0.085$, although there was a trend for all female subjects to be more sensitive to expressions of sadness than male subjects. The 3-way interaction of expression by

sex by depression group was significant, $F_{(4.0,242.1)}=4.1$, $p=0.003$, (see Figure 5.5). These differences can be more clearly analysed in the relative sensitivity analysis in the following section.

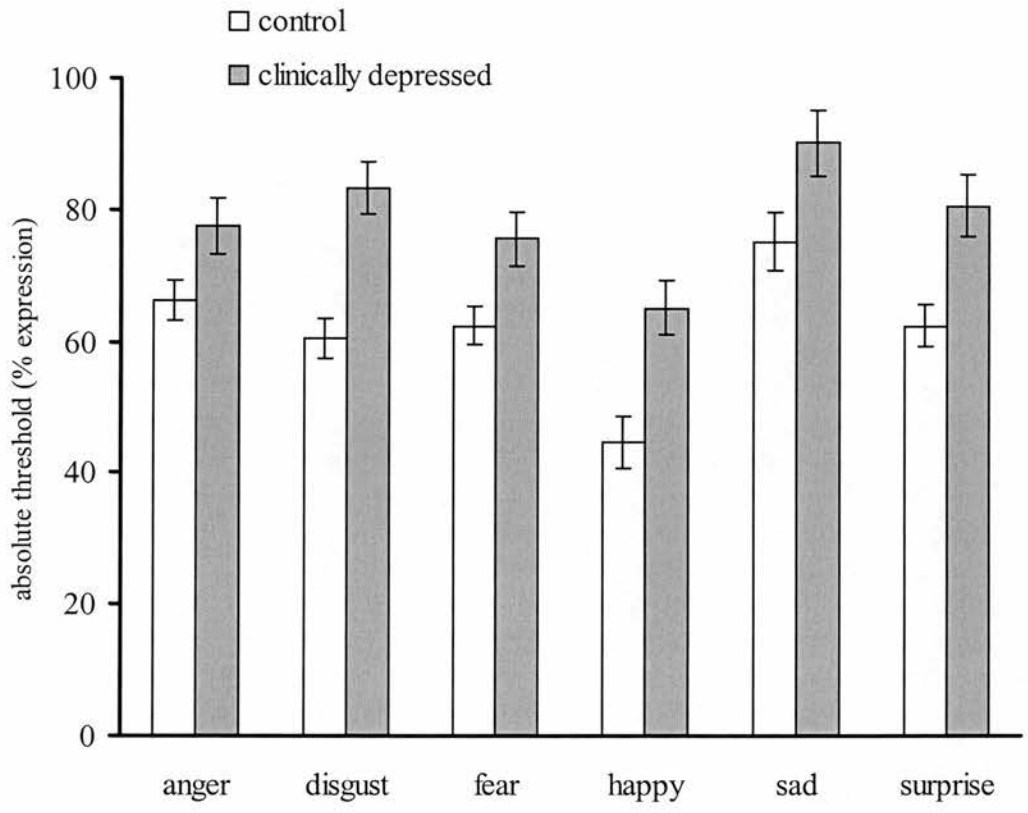


**Figure 5.4: A comparison of absolute sensitivity level (between 0-150%) for the 6 emotions between control and clinically depressed groups (n=32 in each group).**

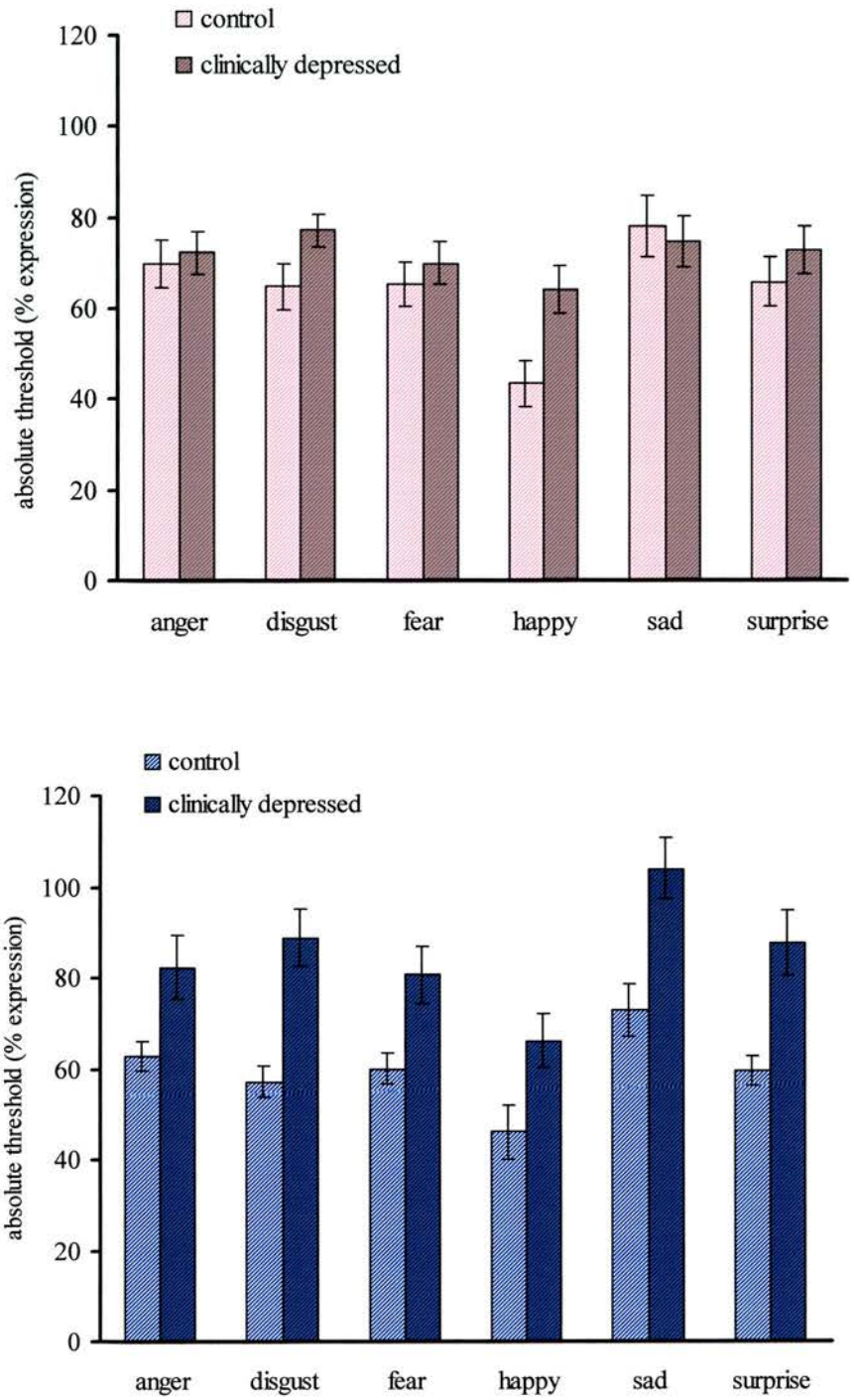


**Figure 5.5: A comparison of absolute sensitivity level (0-150%) in control and clinically depressed subjects divided by sex. The results for females (n=15 in each group) are illustrated in the top graph and for males (n=17 in each group) in the lower graph.**

### 5.3.4 Relative sensitivity

The main experimental question was whether differences in sensitivity would be present across the six different emotions and if these differences would relate to the presence or absence of depression. That is to say, if absolute sensitivity differences were factored out, would there still be differences in the pattern of sensitivity across the six emotions according to depression group? The same contrast measure as that described in section 4.3.2.3 was used to estimate relative sensitivity.

Again a 2*2*6 mixed-design ANOVA was conducted on relative sensitivity.

There was no main effect of subject group, $F_{(1,60)}=1.7$, $p=0.2$, which is expected from the contrast measure used (as the average relative sensitivity to all the emotions for each group should approximate zero). There was also no main effect of sex, $F_{(1,60)}=0.0$, $p=0.9$ and the interaction between depression group and sex was non-significant, $F_{(1,60)}=0.1$, $p=0.8$.

Once again, the within-subjects effect of expression was highly significant, $F_{(4.0,239.8)}=39.3$, $p=0.001$, indicating that all subjects are relatively more sensitive to some expressions compared to other expressions. For example, all subjects find happiness easier to detect than other emotions. There was also a highly significant interaction between expression and depression group, $F_{(4.0,239.8)}=4.8$, $p=0.001$.

Post-hoc (Tukey HSD) tests revealed that control and clinically depressed subjects differ on their relative sensitivity to happy ($p=0.009$), angry ($p=0.001$) and disgusted ($p=0.02$) expressions. Clinically depressed individuals are relatively less sensitive to happy and disgusted expressions compared to controls but are relatively more sensitive to angry faces (see Figure 5.6).

The interaction of expression by sex was not significant, $F_{(4.0,239.8)}=1.7$, $p=0.2$, but there was a trend towards significance for the 3-way interaction of

expression by sex by depression group, $F_{(4.0,239.8)}=2.2$, $p=0.068$. This trend might reflect the difference in relative sensitivity to sadness between female control and clinically depressed subjects and male control and clinically depressed subjects, with female clinically depressed subjects being relatively more sensitive to sadness than female controls and both of the male groups. When divided according to sex, the difference in relative sensitivity pattern for sadness between males and females becomes more apparent (see Figure 5.7). From Figure 5.7 it would appear that females carry the differences revealed between control and clinically depressed subjects in relative sensitivity for happiness and sadness, whereas differences seen for anger and disgust are more evident between male groups. (This trend towards *increased* relative sensitivity to sadness in clinically depressed females is interesting, particularly as sadness is generally the most difficult emotion to detect – presumably due to little morphological change in the face from a neutral expression.) It is notable that differences in *direction* of relative sensitivity between control and clinically depressed groups remain the same for both sexes across all the emotions except sadness.

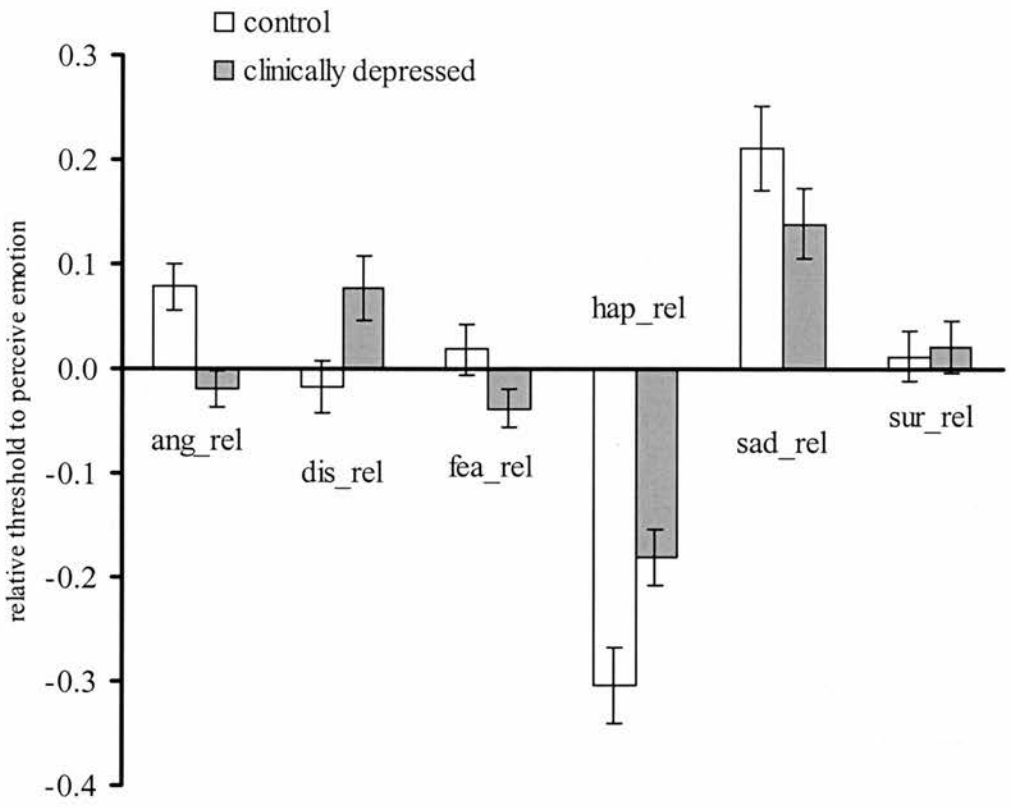


**Figure 5.6: A comparison of pattern of relative sensitivity across the 6 emotions between control and clinically depressed groups (n=32 in each group). A high threshold denotes decreased sensitivity.**

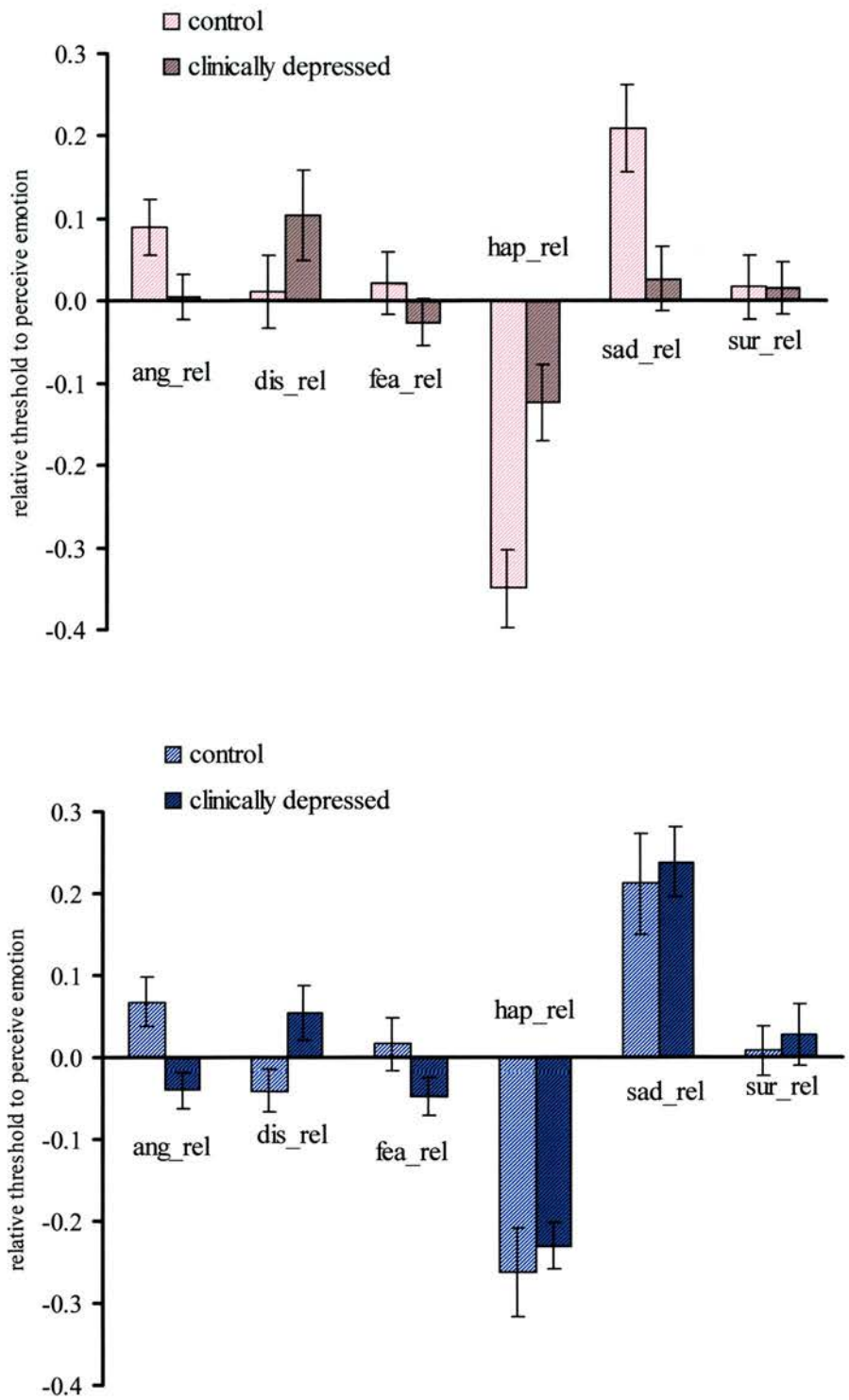


**Figure 5.7: Pattern of relative sensitivity across the 6 emotions for control and clinically depressed groups divided by sex (15 subjects in each female group, top graph; 17 in each male group, lower graph)**

## 5.4 SUBSIDIARY ANALYSIS

13 control subjects (4 male, 9 female) were excluded from the initial data set as their BDI score did not meet the criterion set (≤9). Their data were later entered as a third level of the between-subjects depression factor, in an attempt to establish whether their response pattern approximated that of other control subjects or that of clinically depressed patients. (For simplicity, this group was labelled mild depression; see Appendix 5.5 for general subject information.) Although these subjects were not clinically depressed all their BDI scores were greater than 10 ($\bar{x}$=11.7, SE 0.7). Between-subjects t-tests (unequal variance) revealed a highly significant difference between both control ($\bar{x}$=3.4) and mildly depressed groups BDI scores, t(25.1)=-9.8, p=0.001) and also between mildly and clinically ($\bar{x}$=29.7) depressed groups scores, t(41.0)=-11.2, p=0.001).

Subjects in the mildly depressed group were of a comparative age ($\bar{x}$=44.2, SE 3.4) to those in the clinically depressed group ($\bar{x}$=43.7, SE=2.1), as revealed by a between-subjects t-test (equal variance), t(43)=0.1, p=0.9. There was a slight difference in age between control ($\bar{x}$=37.2) and mildly depressed groups ($\bar{x}$=44.2), t(43)=2.2, p=0.03.

If the mildly depressed group responses approximate those of the clinically depressed subject group, albeit perhaps to a lesser extent, then this would indicate that individuals suffering from subclinical and clinical forms of depression process facial affect in a qualitatively similar manner.

### 5.4.1 Recognition accuracy

A 3*6 mixed-design ANOVA was conducted on the data, with group as the between-subjects factor and expression as the within-subjects factor. Sex was not included as a between-subjects factor here due to the small number of subjects (particularly males) in the mildly depressed group.

Once again a main effect of expression was revealed, $F_{(3.7,274.9)}=10.5$, $p=0.001$, with happiness being the easiest emotion to identify for all subjects. There was neither a main effect of group, $F_{(2,74)}=1.6$, $p=0.2$, nor was there an interaction between group and expression, $F_{(7.4,274.9)}=0.8$, $p=0.6$, suggesting the 3 groups did not significantly differ from one another in ability to identify the emotions using the 6-way forced choice procedure, as can be seen in Figure 5.8.

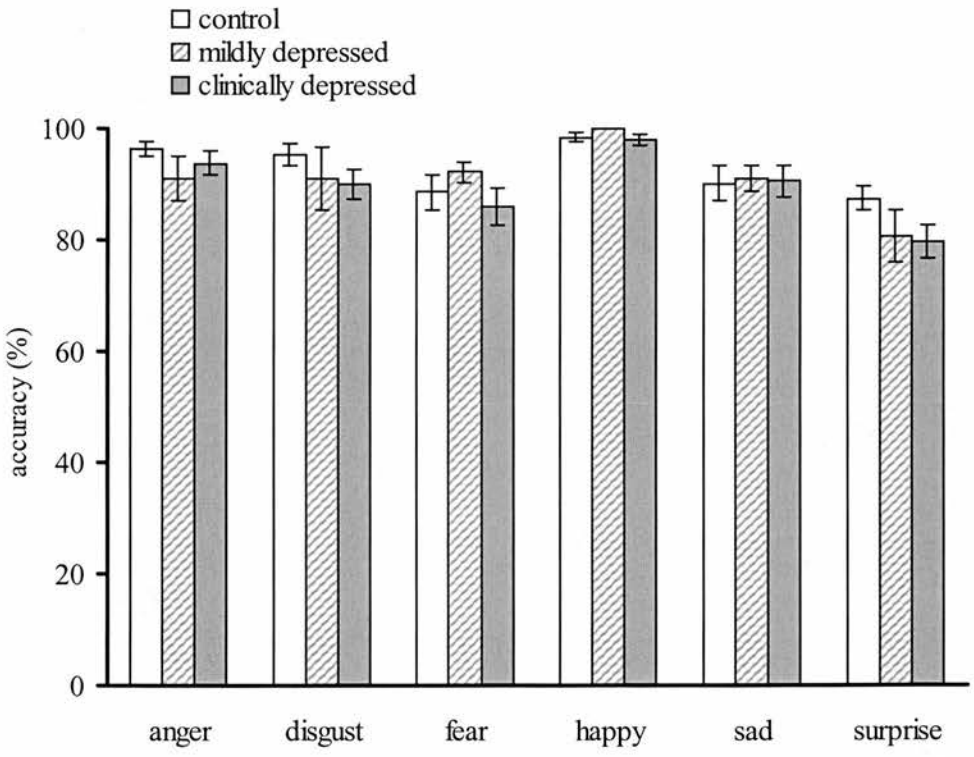


**Figure 5.8: A comparison of recognition accuracy for the 6 emotions between the 3 groups – controls (n=32), mildly depressed (n=13) and clinically depressed (n=32).**

### 5.4.2 Absolute sensitivity

Again a 3*6 mixed-design ANOVA was conducted on the data. There was a main effect of depression, $F_{(2,74)}=5.8$, $p=0.005$, which was analysed with post-hoc tests (Tukey HSD). The control and clinically depressed groups significantly differed in overall sensitivity to the six emotions ($p=0.004$) and there was a trend for the control and mildly depressed group to differ ($p=0.1$), but the mildly and clinically depressed groups did not differ at all from each other ($p=0.9$) (see Figure 5.9). There was also a main effect of expression, $F_{(4.0,294.7)}=28.1$, $p=0.001$. (Again post-hocs revealed that happiness was significantly easier to detect than all other emotions, $p=0.001$, and sadness more difficult than all other emotions, $p<0.05$, for all subjects.) In addition, the interaction between group and expression was significant, $F_{(8.0,294.7)}=2.5$, $p=0.012$. Post-hoc tests (Tukey HSD) revealed that both the mild and clinically depressed groups differed from the control group at the $p<0.05$ significance level only with regard to absolute sensitivity to disgust and to happiness, with the control group being significantly more sensitive to both expressions. Again, differences in pattern of sensitivity are more clearly revealed in the relative sensitivity analysis (section 5.4.3). In addition the control group was significantly more sensitive ($p<0.05$) to both fear and surprise in comparison with the clinically depressed group.

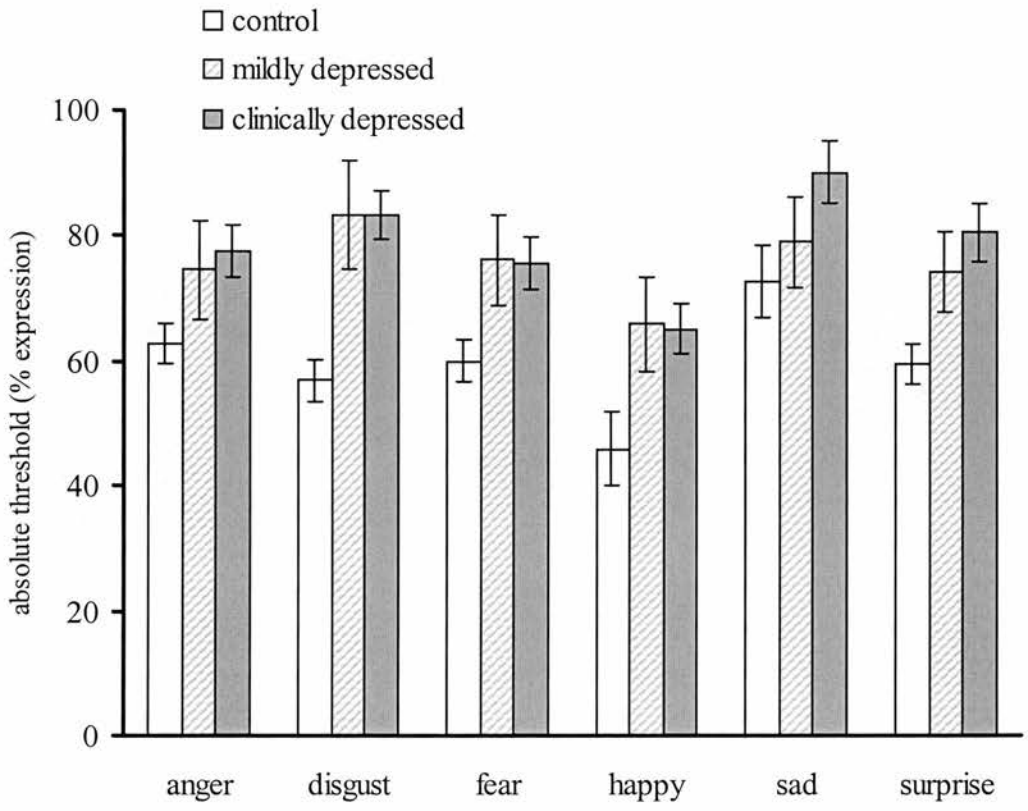


**Figure 5.9: A comparison of absolute sensitivity level (between 0-150%) for the 6 different emotions across the 3 groups – controls (n=32), mildly depressed (n=13) and clinically depressed (n=32).**

### 5.4.3 Relative sensitivity

The same contrast measure (see sections 4.3.2.3 and 5.3.4) was used to compute an individual's relative sensitivity to the different emotions while factoring out absolute sensitivity level.

Relative sensitivity data was subjected to a 3*6 mixed-design ANOVA. This revealed a main effect of expression, $F_{(4.0,296.2)}=27.7$, $p=0.001$, with all subjects exhibiting the lowest threshold once again for detection of happiness. The main effect of group was non-significant, $F_{(2,74)}=1.4$, $p=0.2$, which again is expected when relative sensitivity is compared. The interaction of expression and group was significant, $F_{(8.0,296.2)}=3.4$, $p=0.001$, indicating a different pattern of relative sensitivity across the 6 emotions according to depression group (see Figure 5.10).

Post-hoc tests (Tukey HSD) revealed differences between the groups for expressions of anger and happiness. Both the mildly ($p=0.012$) and clinically ($p=0.002$) depressed groups were significantly more sensitive to angry expressions than the control group. The mild and clinical group did not significantly differ in their sensitivity to anger however ($p=0.95$). For happiness, the control group were significantly more sensitive to happy expressions relative to either the mild ($p=0.025$) or the clinically ($p=0.027$) depressed group and again the mild and clinical group did not differ in sensitivity ($p=0.8$). There were also trends at the $p<0.1$ level for the control group to be relatively more sensitive to expressions of disgust than the clinically depressed group ($p=0.064$). The relative sensitivity of the mildly depressed group to disgust was midway between control and clinically depressed groups (and did not significantly differ from either). There was also a trend for the control and the mildly depressed group to differ in relative sensitivity to sadness with

the mildly depressed group being relatively more sensitive to sad expressions (p=0.058).

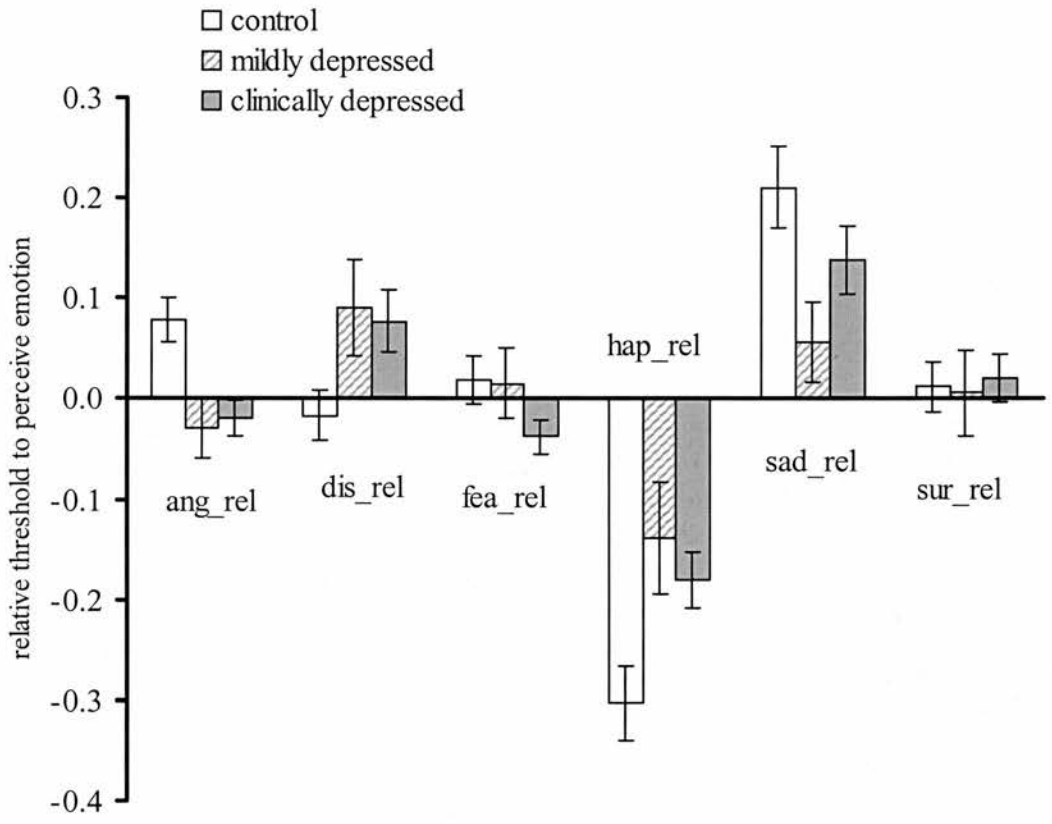


**Figure 5.10: A comparison of relative sensitivity pattern across the 6 different emotions for the 3 groups – controls (n=32), mildly depressed (n=13) and clinically depressed (n=32).**

## 5.5 DISCUSSION

Results from the initial analysis of control (low BDI scores) and clinically depressed groups and also following the inclusion of the mildly depressed group (over cut-off BDI scores but non-clinical) indicate that depressed individuals are generally able to recognise emotions with comparable accuracy to non-depressed individuals. However, depression level does appear to alter interpretation of facial expressions in that both groups of depressed subjects required a greater intensity of each emotion to first perceive it than control subjects did. Non-depressed individuals in this study can therefore be said to have been *more sensitive* overall to changes in facial expression. When pattern of sensitivity across the six different emotions was examined more closely, it was revealed that depressed individuals were relatively less sensitive to happy expressions and to a lesser extent expressions' of disgust compared to control subjects but were relatively more sensitive to angry expressions.

Conclusions that can be drawn from the recognition accuracy data must be cautious at the least as the images used for identification were 150%-caricatured rather than the usual veridical 100% image. The caricaturing should have therefore made identification easier for all subjects (see Calder et al., 1997). There were no between group differences in recognition accuracy but this could be due to ceiling effects. Some errors were present, however, for all groups of subjects, which suggests the test is still valid for testing recognition (in addition to sensitivity) as the caricatured expressions did not result in 100% accuracy. These results lead to the tentative conclusion that any difficulty depressed individuals might have with interpretation of other individuals' expressions is not due to problems in *knowledge* of emotional expressions, i.e., they can identify expressions if presented at sufficient

intensity. Errors made followed the same pattern for all groups, except clinically depressed subjects did exhibit a slight tendency to misidentify fear as anger (in addition to as surprise) and also disgust as anger which was not so apparent in control subjects, although differences did not reach significance.

The absolute sensitivity results demonstrate a clear effect of depression group status on the intensity of expression required to elicit classification of any particular emotion. Depressed individuals (both mildly and clinically depressed) in the current study needed emotions displayed at a greater intensity to be able to identify them. There are several possible conclusions that can be drawn from this. Depressed individuals might be generally overcautious or conservative in their response, perhaps due to a fear of responding incorrectly. Corwin et al. (1990) reported such an effect, with depressed patients exhibiting an abnormally conservative response bias in a recognition memory test. Depressives were shown to be more uncertain of their response and erred on the side of caution (claiming they did not recognise the imagery item from before), even though discrimination ability was not affected (other than in severely depressed individuals). A putative link between response bias and dysregulation of the central noradrenergic system (which is believed to occur in depression; see Schildkraut, 1965) was implicated by this study. The presence of a conservative response bias does not of course account for the differences seen in sensitivity across the emotions between the groups.

Alternatively, it is possible that depressed individuals have become socially 'numb' to an extent – due to a combination of the effect of numerous rejection (either real or apparent) and withdrawal responses of others from them and also their own self-preoccupation (Sakamoto, 1999). Stimuli might therefore need to be more intense to initially engage attention towards external events and then allow sufficient

stimulus processing to occur. Wexler et al. (1994) reached a similar conclusion from results of a study measuring facial muscle response (EMG) in depressives (see section 2.4.5). Their findings suggested that individuals suffering from depression were *"...hyposensitive to emotion-related stimuli in general"*. Medication might also have a mediating effect in that responses might become more blunted (for example, a reduction in processing speed is common with tricyclics; Dr T. Wheeldon, personal communication), although several of the clinically depressed individuals were not receiving any medication. In addition, although medication history was not obtained from control subjects, it seems unlikely that many of the mildly depressed subjects would have been taking medication (particularly as they were informed their data would be used as a control sample and contrasted with data from clinically depressed patients). It is therefore fairly unlikely that medication accounted for the changes exhibited in sensitivity between depressed (both mildly and clinically diagnosed) individuals and control subjects. As the pattern regarding the ease with which the different emotions can be detected differs for depressed and control individuals, it is valid to compare these differences in relative sensitivity while accounting for absolute sensitivity level.

When absolute sensitivity level was controlled for, the relative sensitivity data revealed a significant effect for depressed individuals to be relatively more sensitive to angry faces than control subjects were when taking into account pattern of sensitivity across all emotions. This could reflect a depressed individual's heightened sensitivity to another person's annoyance or disapproval at him/her, which could culminate in the depressed person unnecessarily withdrawing from an interaction. Feelings of pessimism, insecurity and failure are likely to be magnified following apparent expression of anger from others. Another possible reason why

sensitivity to anger is enhanced in depressives could be the fact that *both* depression *and* aggression are associated with dysfunctional serotonergic neurotransmission. Aggressive behaviour (as determined by a laboratory aggression-provoking task) has been shown to be positively correlated with depressive symptomatology in women (Bjork et al., 1997). Increased aggressive behaviour could result in a depressed individual being more sensitive to anger directed at him/her. It would perhaps be useful to address, in any therapeutic treatment, a depressed person's internal emotional experience when he/she believes anger is directed at him/her. This increase in sensitivity is accompanied by a slight decrease in accuracy in that although accuracy of identification (true positives) is equivalent for both control and depressed groups, false positive identification of anger is more common in clinical depressives. However, as only correct identification responses are included in the sensitivity analysis for anger it still remains that depressed individuals are relatively more sensitive to angry faces than non-depressed individuals.

Similar negative social outcomes are liable to arise from changes in the depressed individual's perception of happiness revealed in the current results. Although the depressed group were relatively more sensitive to expressions of happiness than the other emotions examined, they were much less sensitive (in absolute terms) as a group to happy faces than control subjects were. This discrepancy seems to be especially prominent for depressed females. If a greater intensity of happiness needs to be displayed before a depressed individual can process that face - and therefore person - as being happy, one can predict that many of the normally detected nuances of happiness or pleasure will pass by unnoticed. As a consequence, depressed individuals will perceive fewer positive interactions and

experience less positive feedback on their actions than normal. The majority of benefits and positive side effects gained normally from others' happy 'vibes' will elude depressed individuals, which of course is liable to maintain, if not exacerbate, depressive state. This deprivation potentially has both direct and indirect consequences on the depressed individual's feeling of well-being. If the depressed individual believes that his or her company is not enjoyed by others or does not make others happy, then the depressed individual is more likely to feel worthless and negative about him/herself, resulting in increased withdrawal from social interactions. Social isolation will exacerbate negative cognitive schema and therefore depressive symptomatology. People are also likely to withdraw from an interaction with a depressed person if the depressed individual appears to be responding inappropriately towards them and are prone to avoid that individual in the future (Hokanson et al., 1989; Hops et al., 1987; Biglan et al., 1985). For example, a depressed person might be less likely to express positive emotions (either to signal how they feel or in response to others) and might not detect or respond to positive emotions directed at him/her from others.

Another possible cost of such perceptual dampening with regard to detection of facial expressions of happiness is the loss of more direct physiological feedback. Often when interacting with a happy individual, as well as psychologically enhancing one's mood, one will often subconsciously imitate the other individual's expression or at least respond similarly with a smile or positive gesture (Surakka & Hietanen, 1998). Facial EMG activity has been recorded in normal individuals in response to different types of positive and negative affective stimuli. For example, increased activation in the cheek region or zygomatic muscle group (smiling) and the *orbicularis oculi* eye muscle (Duchenne smile) correlates with exposure to positively

valenced affective stimuli, whether the stimuli are facial expressions (Surakka & Hietanen, 1998), vocalisations (Hietanen et al., 1998; Jäncke et al., 1996) or aurally presented words (Wexler et al., 1992). Results have shown that subconscious levels of muscle activity compatible with the affect observed are present, which in turn can induce experience of the affect in question (Hietanen et al., 1998). Contagious effects of smiling (i.e., generation of positive mood from subconscious imitation of others' happy expressions) are less likely to occur in depressives due to decreased perception of happiness in others. Much more intense stimulation will be required for happiness to be perceived by these individuals.

As well as confirming an intuitive prediction that perception of happiness might be altered in depressed individuals (due to extreme negative mood), these results replicate to an extent previous findings of decreased sensitivity to happy faces in individuals with depressed mood state. Following a depressed Mood Induction Procedure (MIP) on normal individuals, Bouhuys et al. (1995) found that individuals rated a set of schematic faces (see Figure 3.1) as appearing *less* happy and inviting (and expressing more sadness and rejection) than they had prior to the MIP. Mandal and Bhattacharya (1985) reported that a clinically depressed group found photos of sadness easier to identify than expressions of happiness. Of course the depressed subjects in the current study had no difficulty *identifying* happiness when at the full intensity of 150% expression, they were just relatively less sensitive to it compared to controls. A negative bias in rating happy expressions has also been revealed in clinically depressed patients (Gur et al., 1992). Patients were shown photographs of actors posing happy, neutral and sad faces and were asked to rate each on a 7-point scale according to the degree with which they expressed each of the 3 emotions. Depressed individuals were more likely to rate happy faces as neutral and neutral

expressions as sad than controls. It is important to note in Gur et al's study that recognition accuracy was not impaired as such in the depressed group inspite of the presence of this bias. A further study revealed a selective deficit in depressed patients' ability to match happy, interested and sad facial expressions, in the absence of any difficulty matching vocal expressions (Rubinow & Post, 1992).

Although there is only a trend in the data (when factoring out sex) for individuals suffering from varying degrees of depression to be relatively more sensitive to sad expressions compared to controls, it is inline with the trend found in study 1 (reported in Chapter 4) and also corresponds to the negative bias exhibited for happiness. Similar findings of increased sensitivity to sadness in individuals experiencing depressed affect have been reported by Gur et al. (1992) and Bouhuys et al. (1995), both using different experimental paradigms. All subjects find sadness more difficult to detect than the other five emotions and require more intensity to first perceive it (probably due to the small structural change in the face from a neutral expression), but this effect is less pronounced in depressed than non-depressed individuals. This trend for relatively increased sensitivity to sadness in clinical depressives is obviously consistent with depressed individuals' negative mood state, which might centre attention towards negative cues from others. As these current results demonstrate, however, the selective attention for negative expressions is discriminative, i.e., only specific to certain emotions, such as sadness and anger[6], but not disgust.

[6] Relative sensitivity to fearful expressions is also in the same direction, i.e., depressed individuals are relatively more sensitive to fear than non-depressed individuals, although this does not achieve significance.

The trend for increased relative sensitivity to sadness becomes much stronger when examining female subjects in isolation. With regards to absolute sensitivity level, female control and clinically depressed individuals first perceive a sad expression at approximately the same intensity. As depressed individuals require more intensity to detect any emotion compared to controls, female depressed subjects are therefore actually quite sensitive to sadness relative to how they perceive other emotions. Interestingly, a recent paper by Bouhuys et al. (1999) has indicated that female depressives are more likely to exhibit a negative bias in perception of affect from schematic faces (i.e., perceive more negative emotion) than male depressives, which in turn seems to have a negative impact on the outcome of their condition.

It is unclear why female depressives seem to carry the sensitivity effects seen for happiness and sadness (see Figure 5.7). It is possible that females are more concerned with whether another individual is being positive or rejecting towards them, whereas males might be more influenced according to whether they perceive disapproval or aggression (anger) from others. Although there is no empirical data as yet to support such hypotheses, in behavioural terms at least, females suffering from depression are more likely than their male counterparts to seek social support, reassurance (i.e., some positive feedback) and help, whereas males are more likely to merely complain of physical symptoms (Dr T. Wheeldon, personal communication). Such behaviour from male depressives might suggest they are less likely than female depressives to disclose their inner feelings for fear of disapproval and scorn from others and are instead stressing the physical complaints associated with their condition. Hammen and Peters (1978) found that individuals acting as depressed were rated as possessing more feminine than masculine traits (e.g., passivity,

helplessness) and also commented on other findings reporting that attributes stereotypically associated with males were more closely related to the idea of a mentally healthy individual than those associated with females. In their study, individuals were more likely to reject depressed than non-depressed actors (with regard to future contact), but this effect was considerably stronger when the actor was of the opposite sex than if he/she was of the same sex. The authors believed this difference could be due to the fact that the presence of depression might be a more salient factor with regard to choosing a possible partner than to forming a friendship with someone of the same sex. The fact that both male and female subjects rated the depressed actors (of both sexes) as having more characteristically female traits led the authors to conclude that males might be less inclined to disclose feelings of depression to others for fear of rejection as such behaviours would be viewed as stereotypically inappropriate. Such a discrepancy between males and females could partly account for the sex difference in incidence of depression reported (i.e., greater prevalence in females).

Differences in sensitivity pattern for disgust between clinically depressed and control groups are striking. Depressed subjects show relatively *increased* sensitivity (in direction if not significance) to all other negative emotions, e.g., anger, sadness and fear, whereas for disgust, *decreased* sensitivity is exhibited. These results correspond with data trends seen in the high BDI subjects in the initial study on a normal population (see section 4.3.2.3 and 4.4.1 for discussion). There is no obvious psychological reason as to why this pattern should occur for disgust in depressed individuals, although other patient groups have also been shown to have difficulties in perception of disgust (see section 4.4.1). It might be of interest in future studies to examine patterns of brain activation in depressed individuals corresponding with

perception of disgust and whether these differ in any manner from those observed in normal individuals.

Corroboration of these findings of altered sensitivity to specific emotions in depressives can be observed in a study by Berenbaum (1992) who investigated the same issue from a contrasting angle, by assessing *expression* (rather than perception) of emotion. He examined types of facial expression exhibited by depressives (and schizophrenics) in response to specific instructions. There were two conditions, with subjects asked on one occasion to imagine smelling or tasting something disgusting and on the other to envisage something wonderful had happened and that they were very happy. For both, subjects were required to display how they felt by posing what they thought was the appropriate facial expression. Depressives were found to display more expressions recognisable as anger or contempt than schizophrenics or controls (in response to the disgust prompt) but fewer expressions rated as happiness (in response to the happiness prompt) than controls. Berenbaum concluded that these expressive patterns exhibited by depressives were likely to "*...reflect their underlying emotional state*", with depressives frequently feeling hostile and exhibiting aggressive behaviours towards others in addition to experiencing anhedonia. Interestingly, in line with the findings of decreased relative sensitivity to disgust in the current study, depressives also appeared to exhibit considerably fewer expressions rated as disgust than controls in the disgust condition, although a statistical comparison is not reported. Comparison of Berenbaum's findings with the results of the current study implies that there is a distinct relationship between expression, experience and perception of emotions.

Much the same pattern of results as demonstrated in clinically depressed patients in the current thesis was present when mildly depressed individuals were included as a third level of the between-subjects depression factor. The responses of this group approximated those of the clinically depressed group rather than those of the control group even though their BDI scores are only slightly higher than the cut-off of 10. This indicates in turn that sensitivity to facial expressions differs in degree according to depression level and adds weight to the general argument that differences in depression are merely quantitative as opposed to qualitative. Persad and Polivy (1993) examined differences in expression recognition and responses to observed expressions across normal and both subclinically and clinically depressed individuals (see section 3.1.2). They arrived at a similar conclusion, as few differences were present between the two depression groups whereas differences in recognition ability and adaptive responses to the expressions were apparent between the control group and both depression groups.

The results - particularly from the control and mildly depressed groups - suggest that the BDI is quite a sensitive measure of individual differences in mood in that it can detect even minor fluctuations in depressed or negative affect in a normal population. These differences in turn corresponded with different interpretation of facial expressions. The interactive facial expression test was therefore equally sensitive to subtle changes in perception between the groups. Such sensitivity is crucial for examination of differences across a 'normal' subclinical population and also to reliably assess and compare results from those suffering from clinically diagnosed affective disorders.

## 5.6 LEVEL OF DEPRESSION AND FACIAL EXPRESSION PERCEPTION

Similar difficulties to those mentioned in section 4.7.1 in classifying control individuals' with high BDI scores apply. These individuals might just be suffering from high negative affect or general distress (with different associated symptoms) rather than a mild form of depression. However, one must remain cautious when dismissing subclinical examples of depressed affect as qualitatively different from clinically diagnosed depressive episodes. It is possible that a differing self-referral rate might account for group classification, i.e., some subclinical sufferers *would* be considered as being clinically depressed if they chose to report their problems to a GP. In addition, previous psychiatric history was not recorded here so it is possible that some of the mildly depressed group might have previously suffered more severe clinically rated episodes of depression and might be predisposed to depression. High recurrence rates are common in depression - over half relapse within a 2-year period (Keller et al., 1984) with 1 in 4 undergoing 6 or more relapses in a lifetime (Angst, 1986). Such high rates are not even moderated by treatment maintenance (Frank et al., 1990), particularly if one relapse has already occurred. Another hypothesis is that negative affect can also be a form of personality trait, rather than a discrete illness, and that individuals exhibiting such a characteristic would possess an enduring, although probably relatively mild, dysphoric mood state, known clinically as *dysthymia* (Rihmer, 1999; Akiskal, 1996, 1994).

The comparison of control, mildly depressed and clinically depressed data appears to be in line with the continuity hypothesis (see section 4.7.1), in that mildly depressed subjects exhibit an almost identical pattern of facial expression perception to the clinically depressed patients (albeit to a lesser degree). Whether this alteration in facial expression perception is merely a symptom or manifestation of depression

or indeed contributes to the persistence of a depressive state (due to decreased social functioning) remains unclear. However, perceptual response does not appear to differ qualitatively between individuals who merely subclinically report experiencing moderately high levels of depressed affect and clinically diagnosed individuals whose daily functioning is severely affected by their illness.

## 5.7 COMMENTS ON METHODOLOGY

Although sad faces were not the most difficult to identify (see Table 5.1), all subjects had the highest threshold for detection of sadness. Such an apparent discrepancy between recognition accuracy and sensitivity is probably a consequence of the small morphological change between neutral and sad expressions, compared to the more substantial alteration of face structure for most other emotions. Sadness will therefore be harder to perceive at lower intensities of the expression, in spite of relatively straightforward recognition ability when viewing the end-point (150%) image.

One criticism that could be levelled is the fact that a 6-way forced-choice procedure was used in the recognition test. Subjects might therefore identify some expressions by a process of elimination rather than giving a free choice. Each Ekman identity only portrayed two different expressions though, so it is unlikely that much comparative information was available to the subject as to which emotion was being represented. (That is to say that if all six emotions had been displayed by one identity, it would have been possible for the subject to deduce the remaining expression – if ambiguous – as a consequence of having already identified the other five. Subjects were prevented from doing this here as insufficient information was available with only two expressions being portrayed by each identity.) As sensitivity

to emotions was the primary concern of the study, it was only important to ascertain that depressed individuals' recognition accuracy (or knowledge of how an emotion was facially expressed) was somewhat equivalent to that of control subjects. There does appear to be a significant amount of evidence (in particular from neuropsychological and cross-cultural studies) endorsing the idea that there are discrete emotion concepts and emotion is not simply identified as variations along continuous independent dimensions of arousal and pleasantness (see sections 1.1.5 and 1.2). The images used here have been used in a variety of tests under different conditions and recognition has been found to be relatively reliable. In this respect, it is reasonable to assume that the current test has assessed some degree of facial expression recognition ability.

# CHAPTER 6: EFFECTS OF REMEDIAL SURGERY ON TREATMENT-RESISTANT DEPRESSIVES (STUDY 3)

## 6.1 INTRODUCTION

The benefits and ethics of psychosurgery have long been debated. Over the past 60 years the frontal lobe has been the main site for surgical treatment of affective disorders. Disconnection of frontal cortex from other cortical and subcortical structures is believed to reduce its inhibitory role, which might be overactive in psychiatric patients. (For example, disinhibited behaviour is apparent in the well-documented historical story of the railroad worker, Phineaus Gage, who severely damaged his frontal lobe in an accident.) Procedures have progressed from complete frontal lobotomies to more refined selective stereotactic lesions of fronto-thalamic or fronto-cingulate fibres (Malizia, 1997). Psychosurgery is utilised as a last resort for individuals with medically intractable depression who have not responded to several different rigorous programs of treatment (pharmacological, psychological and also often electroconvulsive therapy). Approximately 15-20% of patients are considered as treatment-resistant (Burrows et al., 1994), i.e., euthymia (normal mood) has not been achieved or maintained following treatment, but only a minority of these will satisfy criteria for psychosurgery. The explicit aim of psychosurgery is to alter the behaviour and affective state of the individual such that it becomes more normal. Kiloh (1986) reported that a marked improvement in condition is observed after surgery in approximately 63% of patients.

The surgery conducted on the patients in the following study was restricted to connecting fibres in the anterior capsule, which disconnects the orbitofrontal cortex from the mediodorsal nucleus and the septum and hippocampus (see Figure 6.1).

Tailarach et al. (1949) were the first to perform such lesions and reported treatment efficacy with patients suffering from anorexia nervosa. Later studies comparing outcome in a larger number of patients demonstrated that the greatest effects of the treatment were obtained for individuals suffering from obsessive-compulsive disorder (OCD) (Bingley et al., 1977; Herner, 1961). Anterior capsulotomy surgery is still commonly used for treatment-resistant OCD patients, in addition to individuals with affective disorders, due to the lack of associated surgical complications and low rate of side effects combined with the relatively high rate (~60%) of post-surgical improvement in condition (Hodgkiss et al., 1995; Ballantine et al., 1987; Bailey et al., 1973). (However, it is notable that not all individuals respond to psychosurgical treatment.)

Assessment of post-surgical social functioning has been particularly focused on due to the often deleterious personality changes associated with earlier, cruder procedures. In the majority of recent cases involving more refined surgical intervention, improvement was shown with scores progressing towards population norms on personality and social competence scales (Mindus & Nyman, 1991). In addition, no differences in global IQ have been shown due to surgical treatment (Kartsounis et al., 1991; Bingley et al., 1997). Stereotactic subcaudate tractotomy (SST) surgery or anterior cingulotomy is also carried out for treatment of refractory affective disorders. This procedure involves selective lesioning of the white matter in the medial and posterior area of the frontal lobe.

This study of stereotactic surgery of frontal fibre systems was essentially unplanned and the opportunity of such a longitudinal investigation arose in the

course of work for study 2 (see Chapter 5) carried out in the Psychiatry Department in Ninewells Hospital and Medical School.

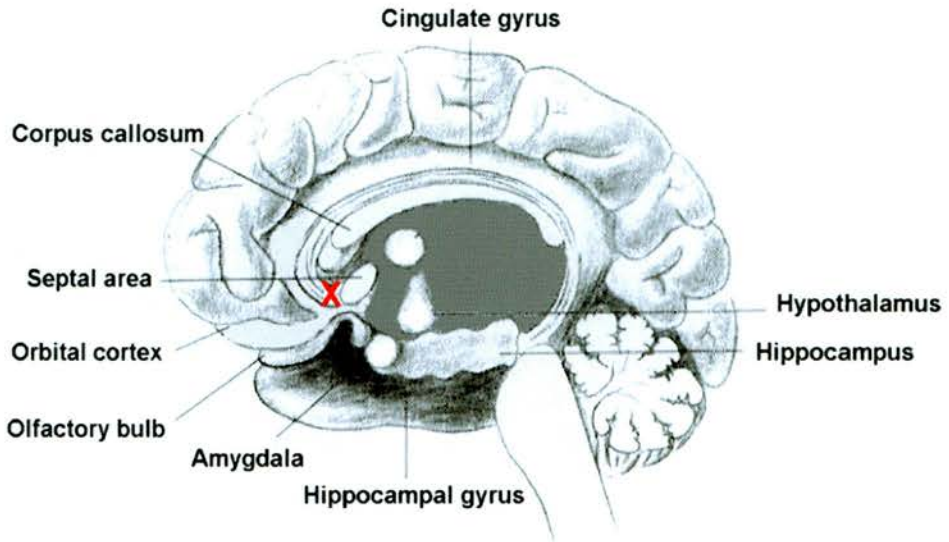


**Figure 6.1: A diagram to illustrate the psychosurgical procedure of anterior capsulotomy. Fibres in the anterior capsule are lesioned (see cross), disconnecting neural pathways between orbitofrontal cortex and the thalamus, hippocampus and septum.**

Two of the three patients admitted for anterior capsulotomy surgery in the current study were suffering from bipolar affective disorder with a current severe depressive episode. The primary diagnosis of the third patient was OCD but she also had a severe secondary depression. A close relationship between OCD and depression (particularly with obsessional thoughts) is common (ICD-10 Classification of Mental and Behavioural Disorders, World Health Organisation, 1992).

The data from the pre surgery test sessions of the three patients were also included as part of the clinically depressed group in study 2. The comparison of

interest here however was the examination of the effects of the surgical treatment on facial expression perception (presumably in part as a function of change in depressive state), as opposed to contrasting results with control subject data. A within-subjects design was therefore implemented.

## 6.2 METHOD

### 6.2.1 Participants

3 patients, 2 male and 1 female, participated in the study. Patient 1 was male, aged 55, with an ICD-10 diagnosis (F31.4)[1] of bipolar affective disorder with a current depressive episode without psychotic symptoms. Patient 2 was male, aged 49, also with an ICD-10 classification of F31.4. Patient 3 was female, aged 44, with an ICD-10 diagnosis of obsessive-compulsive disorder (F42.2) but was also suffering from severe depression. Patients were approached by the consultant in charge as to participation in the study, which was entirely voluntary and did not affect their treatment in any way. Pharmacological treatment was not controlled for. (See Appendix 6.1 for complete details of patient medication.)

All subjects gave informed consent to participate in the study.

### 6.2.2 Materials and procedure

The stimuli and interactive morph software used were identical to those in study 2 (see sections 5.2.2 and 5.2.3). The procedure was also equivalent except subjects were tested on two separate occasions: once before surgical intervention (patient 1, ~48 hours before; patient 2, ~49 hours; patient 3, ~48 hours) and again post surgery (patient 1, 3 days after; patient 2, 12 days; patient 3, 8 days).

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[1] ICD-10 is the classification system used by the World Health Organisation for mental and behavioural disorders.

Initially it was hoped that number of days between each test session and surgery could be equivalent. Although this was possible with patient 1, patient 2 was experiencing confusion and head pain (as a consequence of the surgical procedure), had difficulty maintaining attention and was therefore unable to complete testing 3 days post surgery. He was re-tested over a week later, once an improvement in his attentional state had been clinically reported. Patient 3 was re-tested 8 days post surgery due to practical difficulties. The time of day at which patients were tested was kept constant across test sessions to control for any possible diurnal mood or attention fluctuations, although this was not entirely possible with patient 3 due to other appointment and interview sessions in the hospital. (Patients 1 and 2 were seen between 11am-12pm on both sessions and patient 3 at 12pm on session 1 and 9.40am on session 2.)

Patients were not required to complete all 6 practice trials on the second test session (assuming they recalled the aim of the task sufficiently after a couple of trials). Otherwise testing procedure was the same, with patients filling out the BDI prior to completing both parts of the facial expression test.

## 6.3 RESULTS

### 6.3.1 Comparison of age across the three patients

The average age of the patients was 49.3 years (SE 3.2). See Table 6.1 for the age of individual patient's.

### 6.3.2 Comparison of BDI scores across the two sessions

Average BDI score pre surgery was 40.3 (SE 4.9) compared to a post surgery average of 21.0 (SE 8.3). A paired t-test (one-tailed) revealed that this difference was significant, $t(2)=5.3$, $p=0.017$, suggesting that remedial surgery did succeed in decreasing depressed affect to some degree. (See Table 6.1 for individual scores.)

| | Age | BDI (PRE) | BDI (POST) |
|---|---|---|---|
| Patient 1 (male) | 55 | 40 | 17 |
| Patient 2 (male) | 49 | 49 | 37 |
| Patient 3 (female) | 44 | 32 | 9 |

Table 6.1: Individual patient's age and pre and post surgery BDI scores.

### 6.3.3 Recognition accuracy

A 2*6 repeated-measures ANOVA was conducted on the recognition accuracy data, with session (pre versus post surgery) and expression (6 levels) as within-subjects factors.

There was a main effect of expression on accuracy, $F_{(5,10)}=5.1$, $p=0.014$, as some expressions were more difficult to identify than others. In general though, recognition accuracy for all emotions was very high. There was also a main effect of session, $F_{(5,10)}=30.3$, $p=0.031$, indicating an overall increase in errors after surgery. Both main effects were qualified, however, by the significant interaction of expression and session, $F_{(5,10)}=3.8$, $p=0.036$, which reflects the dramatic decrease in correct recognition of fear post surgery from 95% to 55% (see Figure 6.2). Post-hoc tests (Tukey-HSD) revealed a significant difference in accuracy in identifying fear

according to session ($p=0.017$). (N.B. 55% is still shown to be considerably above chance when tested for binomial distribution, $p<0.001$, which would be 16.7% in a 6-way forced choice.)

| | PRE SURGERY | | POST SURGERY | |
|---|---|---|---|---|
| | % correct | SE | % correct | SE |
| ANGER | 100.0 | 0.0 | 100.0 | 0.0 |
| DISGUST | 100.0 | 0.0 | 100.0 | 0.0 |
| FEAR | 94.4 | 5.6 | 55.6 | 5.6 |
| HAPPY | 100.0 | 0.0 | 100.0 | 0.0 |
| SAD | 100.0 | 0.0 | 94.4 | 5.6 |
| SURPRISE | 100.0 | 0.0 | 83.3 | 16.7 |

Table 6.2: Comparison of recognition accuracy scores for the 6 emotions within-subjects (n=3) across test sessions (pre and post surgery).

In spite of the small number of total trials for each expression per session (3*2), it is notable from the means (see Table 6.3) that fear recognition drops in all three patients and is not merely a consequence of poor response by one individual patient. (The variance across the 3 patients remains the same pre and post surgery for fear identification, see Table 6.2 – SE=5.6 for pre and post)

| | PRE | POST |
|---|---|---|
| Patient 1 | 100.0 | 50.0 |
| Patient 2 | 100.0 | 66.7 |
| Patient 3 | 83.3 | 50.0 |

Table 6.3: Individual patient's recognition accuracy (percent correct) for fear both pre and post surgery. (Each score is the total correct identifications from a possible 6.)

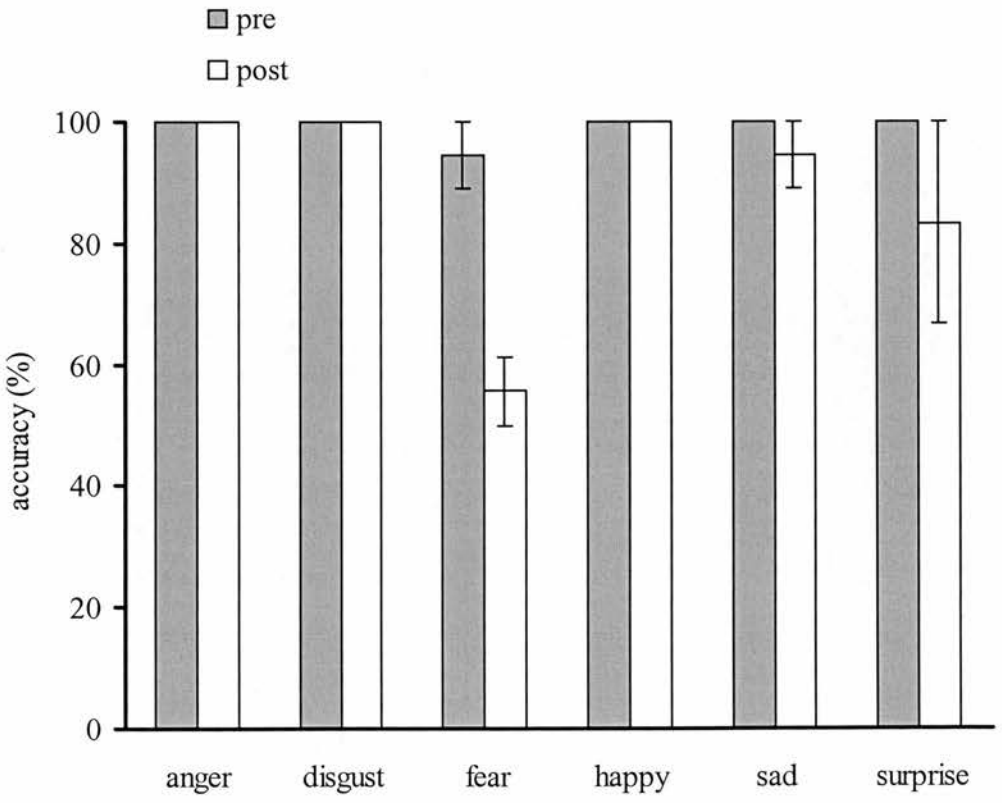


**Figure 6.2: Comparison of recognition accuracy scores across the 6 emotions for the 3 patients pre and post surgery.**

Type of error made is also of interest (see Table 6.4). Fear and surprise are usually commonly confused (see Ekman & Friesen, 1976); however, misidentification by the 3 surgical patients of fear as surprise only accounts for just over a third of the total errors made for fear, suggesting the deficit post surgery is not simply a straightforward confusion with surprise. Fear is also misidentified as the other 3 negative emotions. Particularly surprising perhaps is the incorrect recognition of fear as disgust, in that both the muscle pattern and the psychological connotations associated with these two emotions are quite distinct.

| | | MISINDENTIFIED AS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | anger | | disgust | | fear | | happy | | sad | | surprise | |
| ACTUAL EMOTION | ANGER | | | - | - | - | - | - | - | - | - | - | - |
| | DISGUST | - | - | | | - | - | - | - | - | - | - | - |
| | FEAR | - | **11.1** | - | **11.1** | | | | - | - | **5.6** | 5.6 | **16.7** |
| | HAPPY | - | - | - | - | - | - | | | - | - | - | - |
| | SAD | - | - | - | **5.6** | - | - | - | - | | | - | - |
| | SURPRISE | - | - | - | - | - | **5.6** | - | **11.1** | - | - | | |

Table 6.4: Error pattern for patients' pre (normal type) and post (bold) surgery.

### 6.3.4 Absolute sensitivity

Trials were again excluded from the sensitivity analysis if the expression had been misidentified (see sections 4.3.2.2 and 5.3.3). Frame numbers were converted into percentage values (between 0-150%).

A 2*6 within-subjects ANOVA was conducted with session and expression as within-subjects factors. There was no overall effect of session, $F_{(1,10)}=0.2$, $p=0.7$, but there was a main effect of expression, $F_{(5,10)}=6.8$, $p=0.005$. Once again, happiness required the lowest intensity of expression to be detected at 76.3%, then disgust 96.4%, fear 98.0%, anger 102.6%, surprise 104.0% and finally sadness 108.5%. (This pattern of sensitivity across emotions is similar to those found in studies 1 and 2, although notably the absolute sensitivity level required across all emotions is higher.) The interaction of expression and session was non-significant, $F_{(5,10)}=1.2$, $p=0.4$. The variance in sensitivity level in the first test session (pre surgery) across patients was shown to be greater than in the second session with a paired-samples t-test, $t(5)=6.5$, $p<0.001$ (see Figure 6.3).

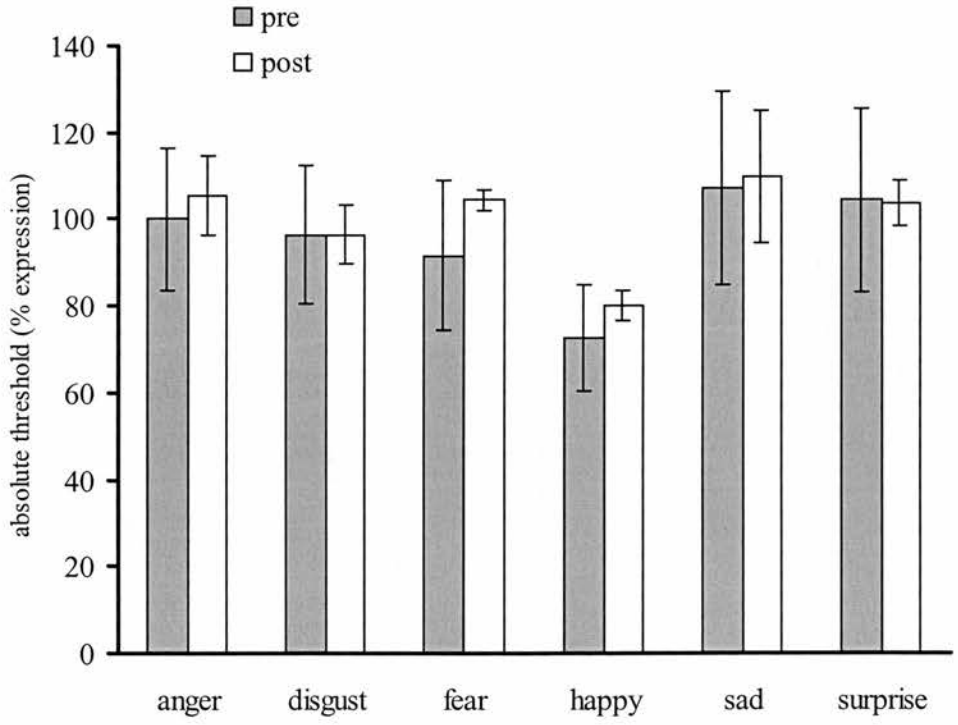


**Figure 6.3: A comparison of absolute sensitivity level (between 0-150%) for the 6 emotions between sessions (pre and post surgery) for the 3 patients.**

### 6.3.5 Relative sensitivity

The same method as that in studies 1 and 2 was used to estimate relative sensitivity.

Again a 2*6 within-subjects ANOVA was conducted. As the average of the relative sensitivity means for all the emotions for each session was zero, there could be no main effect of session (it was impossible to obtain an F-value). The main effect of expression was again significant, $F_{(5,10)}=10.6$, $p=0.001$, with happiness requiring the lowest relative threshold to be detected. However, relative sensitivity pattern across the emotions did not differ depending on test session as the interaction between expression and session was non-significant, $F_{(5,10)}=1.3$, $p=0.3$. Patients were therefore not differentially more sensitive to any particular emotion post surgical treatment than they had been pre surgery (see Figure 6.4).

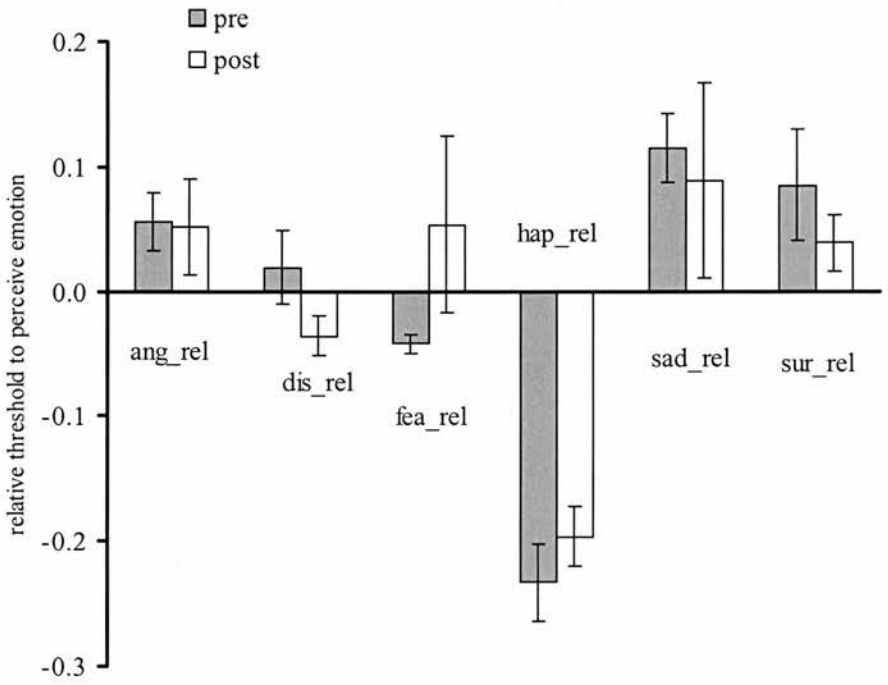


**Figure 6.4: A comparison of relative sensitivity pattern across the 6 emotions between session (pre and post surgery) for the 3 patients. A high threshold denotes decreased sensitivity.**

## 6.4 DISCUSSION

The most striking finding from the data of these anterior capsulotomy (AC) patients is the discrepancy in fear recognition across testing sessions.

Recognition ability was reduced from an average of around 95% to 55% correct after surgical intervention. Caution must be exercised in interpretation of these results due to the very small subject number. Having said this, it is notable that all three subjects showed the same decrease in ability to identify fear post surgery; therefore the result could not be due to idiosyncratic extreme results from a single patient.

Perseveration of response can occur following such lesions (as frontal lobe functioning can be affected; Hay et al., 1993), although this is unlikely to explain the deficit in fear recognition as fear was not always misidentified as the same emotion but as several different emotions (i.e., surprise, anger, disgust or sadness). Trials were also randomised (as in studies 1 and 2) to prevent the occurrence of a response set. In terms of perseverative responding, errors for fear did also not reflect the previous response (e.g., if the previous trial (and response) was anger, this did not result in the misidentification of fear as anger in the next trial). In addition, recognition of other emotions did not appear to be affected. Recognition of fear was not at chance however, so although these data hint at a selective deficit in fear identification following AC surgery it remains unclear both how severe it might be and whether it would be common to all patients.

It is interesting to note that the difficulty revealed post surgery in accuracy in identification of fear did not appear to be a trade-off with sensitivity for fear in that there was no significant difference in relative sensitivity to fear across sessions. Both absolute and relative sensitivity to fear did appear to shift in the direction of a

decrease in sensitivity post surgery, but there were no significant changes. It would be relevant to test such a group of patients (both pre and post surgery) on the original set of 100% veridical expressions to establish whether recognition accuracy would be significantly worse post surgery (i.e., near to chance) than with 150%-caricatured expressions. Using 150%-caricatured expressions might not be revealing the full extent of the difficulty as recognition is generally enhanced with these images (Calder et al., 1997) and could explain why recognition accuracy is still above chance in the current study.

Previous research has highlighted the possible role of the amygdala in perception of fear (Broks, 1998; Phillips et al., 1998; Calder et al., 1996a; Morris et al., 1996; Adolphs et al., 1995; Young et al., 1995; see sections 1.1.2 and 1.1.5). AC surgery primarily affects fronto-thalamic connections, particularly pathways between the orbitofrontal cortex and the mediodorsal nucleus of the thalamus and also involves disconnection of the septum and hippocampus from frontal cortex. Such lesions are likely to have indirect effects on limbic areas through the fronto-striatal-limbic loop, which might affect amygdala function. Magnetic Resonance Imaging (MRI) scans cannot be obtained from these patients until approximately one year after the surgery (once gliosis has occurred), so the exact site and extent of the lesion cannot be precisely determined at present.

Depression level as measured by the BDI did significantly decrease in all three patients and clinical reports also suggested a small improvement in mood. Reduction of anxiety appeared to be the most prominent clinical effect of the lesion though. It seems improbable that the difficulty in fear recognition seen post surgery is in any way connected to reduction in depressive state due to the results of studies 1

and 2 and is more likely due to the physiological change in brain activity following the surgical intervention.

Conclusions similar to those made by Hamann et al. (1996) following intact perception of fear in a patient with amygdala damage sustained late in life could be proposed with regard to these patients. Pathways have been destroyed in AC surgery, which might affect indirect connections with the amygdala, an area that is related to fear conditioning and perception (Killcross et al., 1997; Scott et al., 1997; Breiter et al., 1996; Calder et al., 1996a; Young et al., 1995; Adolphs et al., 1995, 1994). As such damage has occurred well after neural development with these patients, it is likely that some residual functioning will remain through other intact systems. Therefore although there might be some difficulties with recognition of fear, the deficit is unlikely to be as severe as one sustained early in life.

There were no apparent changes in sensitivity – either absolute or relative – across test sessions. This is perhaps surprising due to the findings in study 2, where the prediction might be that absolute sensitivity to all emotions might decrease alongside depression level post surgery, in addition to increased relative sensitivity to happiness. There were no obvious trends in the data, although the small number of subjects tested might have obscured possible differences. Again the importance of using a test that probes more than one facet of expression perception (i.e., recognition accuracy and sensitivity) is highlighted by the differential pattern of results here.

Future studies in this area would benefit from a larger subject number and use of an additional comparative control group of non-depressed patients undergoing surgical intervention in another brain region to attempt to eliminate any effects produced by the stress of surgery alone.

# CHAPTER 7: EATING DISORDERS (STUDY 4)

## 7.1 INTRODUCTION

This chapter incorporates a study examining facial expression perception in individuals with clinically diagnosed eating disorders. Prior to the actual study, a general background is provided on eating disorders: diagnostic features, putative aetiological bases and psychological aspects, to enable greater understanding and confer a knowledge basis on which to examine possible changes in emotion processing.

First the nature of each of the two main forms of eating disorder - anorexia nervosa (AN) and bulimia nervosa (BN) - will be briefly discussed. These subgroups are combined into a main eating disorder (ED) group in the current study as, in spite of different manifestations, AN and BN have many commonalties. A brief review of current theories regarding causal factors for the development of eating disorders will follow. Of particular relevance to the current study on facial expression perception is the section examining capacity for emotion processing in eating disorders and expectations regarding possible alterations in perception of facial expressions.

## 7.2 EATING DISORDERS: 2 MAIN TYPES

### 7.2.1 Anorexia nervosa: diagnosis, symptoms, treatment and outcome

Anorexia nervosa (literally translated as *'lack of appetite of nervous origin'*) is a severe psychiatric disorder, which manifests itself in self-imposed starvation and consequently a considerable reduction in body weight. Anorexia is therefore easily identifiable diagnostically, due to such weight loss, but a great deal of sufferers deny their behaviour is abnormal, do not present for treatment and indeed many refuse

help. Weight loss must be at least 15% of normal body weight to meet diagnostic criteria (DSM-IV; American Psychiatric Association, 1994). Three other crucial features of the condition are cessation of menstruation for at least 3 months, distorted perception of body image and a pathological fear of weight gain. About a half of all anorexics also suffer from bulimia (see section 7.2.2). The pure form of anorexia is classed as 'restrictor' subtype.

Prevalence is significantly greater in woman than in men (about a 9:1 ratio) and onset is most common around puberty and early to late adolescence, with approximately one in every hundred young females affected (about one in every two thousand of the general population). The high mortality rate of the condition, estimated between 5-8% of all cases over a period of 5-10 years[1] (Crow et al., 1999; Sullivan, 1995), in comparison with other psychiatric conditions emphasises the importance of continued research in this area. Another study investigating mortality rates in two anorectic patient groups in a 20-year follow-up confirmed the relatively high mortality rate and revealed that the most prevalent causes of death were illness complications and suicide (Crisp et al., 1992).

Intense preoccupation with weight and body image is a defining psychological symptom and comorbid expression of depressive, anxious or obsessional symptoms is also characteristic (Pollice et al., 1997). In addition to starvation, abuse of laxatives and diuretics is common and excessive amounts of exercise all contribute to weight loss in the anorexic's vehement pursuit of thinness. The determination exhibited to conceal such behaviour and weight loss becomes almost addict-like. In spite of self-starvation, anorexics initially have the same appetite as normal individuals but just refuse to eat and deny being hungry.

[1] Mortality is estimated in these studies as a ratio of observed to expected (matched for age and gender) deaths and is also adjusted for length of follow-up.

Enjoyment seems to be derived both vicariously from watching others eat and also in the act of cooking. Ritualistic eating behaviours often develop, such as chopping food up into very small pieces and excessive chewing.

The inherent malnourishment from starvation results in a large number of associated physiological complications, some of which can produce long lasting effects. Examples of such symptoms are: *amenorrhea* (loss of menarche) or lack of sexual interest and potency in males; if onset is prepubertal, cessation of normal developmental changes in puberty can occur, e.g., breasts do not develop; digestive problems; brittle hair and nails; a covering of fine, downy hair on the limbs and face (*lanugo*) to compensate for heat loss due to lack of bodily fat; insomnia; hyperactivity; endocrine changes and electrolyte disturbances. In severe cases, loss of calcium can result in osteoporosis or reduction in potassium intake may cause heart problems and could ultimately cause cardiac failure.

Although many of the affective symptoms experienced in anorexia overlap with depressive symptomatology, it is possible that any concomitant depression arises from physiological changes undergone due to both starvation and excessive exercising rather than acting itself as a causal factor in the onset of the eating problem. Pollice et al. (1997) examined levels of comorbid depression, anxiety and obsessive-compulsive symptoms in anorexic individuals at different stages of recovery. All 3 types of symptom were significantly more severe in the actively anorexic group compared to either partially or completely weight restored groups. Milder symptoms of depression, anxiety and obsessionality were still present in recovered states though, indicating that such psychopathological states might partially contribute to the development of anorexia. However, as Pollice et al.'s study involved a cross-sectional rather than a longitudinal design, it is possible that

the patients who had a good outcome and were less depressed etc. actually previously had a less severe form of anorexia (and associated negative affective symptoms) and recovered as a consequence. Whether depressive, anxious and obsessional states affect the pathogenesis of the condition therefore remains unclear.

Certain personality characteristics also appear to be typically associated with anorectic individuals: for example, perfectionism, fastidiousness, dependence, social anxiety and approval-seeking behaviour. In a recent paper, Fairburn et al. (1999) commented that traits of perfectionism and negative self-evaluation appeared to be elevated in eating disorder patients – particularly in anorexics – relative to both psychiatric and normal controls. An earlier study by Bastiani et al. (1995) reported elevated levels of perfectionism in not only malnourished but also weight-restored anorexics, compared to normal controls, suggesting that such perfectionistic attitudes are relatively persistent and could contribute to treatment resistance.

Interestingly, the anorexics in the Bastiani et al. (1995) study expressed that these high standards were *self-imposed* and were not a consequence of unrealistic parental or peer expectations. In contrast, Pliner and Haddock (1996) concluded that high levels of perfectionism in anorexics were due to a desire to be evaluated positively by others and to be socially acceptable, rather than an attempt to attain self-set unrealistic expectations. Such a desire for social approval and positive feedback could indicate that anorexics might be overly sensitive to facial expressions of others in terms of their emotional valence (e.g., whether others are happy or disgusted at them). Caution must be exercised in such an interpretation though, since the subject sample in Pliner and Haddock's study constituted high and low weight-concerned students rather than clinically diagnosed anorexics, which might have affected the results.

Most treatment plans involve general assessment and stabilisation of condition initially, followed by gradual weight restoration involving monitoring of eating habits and a reward system if certain goals are achieved, and then therapy for associated psychological problems. There is a great degree of variability in outcome for anorexic patients. Approximately 50% regain normal weight and restart menstruating but many residual psychological symptoms remain (such as obsessive thoughts about weight and eating). Around one in four remain chronically ill with anorexia. Crisp et al. (1992) emphasised the importance of treatment continuation as anorexics may often resort to self-starvation again once out of hospital. The first 5 years post recovery are particularly dangerous for susceptibility to relapse.

Such resistance to treatment could partly be a consequence of the perceived positive aspects of the disorder. Serpell et al. (1999) skilfully addressed this issue by asking patients to write two letters directed at their condition: one as if it was a friend and the other as if an enemy. Perceived benefits of anorexia, which recurred frequently, were feeling protected and safe, experiencing control over one's life and feeling 'special' and different from others. Prominent negative factors in contrast were recurrent thoughts about food, the feeling of wasting opportunities, the dislike of the disorder assuming control of one's life, health concerns and disrupted relationships with others. The apparent contradictory nature of the adaptive value of feeling in control compared to the negative aspect of feeling 'taken over' by the disorder epitomises the frequent 'black and white' thinking characteristic of the condition, which relates to rigid all-or-nothing beliefs (Zotter & Crowther, 1991). Treatment could perhaps become more efficacious if the patient is made more aware of the negative aspects of the condition (such as deterioration of health or damage

caused to relationships) and also if a greater understanding of the motivating factors, which might help maintain the condition, is achieved by clinicians.

### 7.2.2 Bulimia nervosa: diagnosis, symptoms and outcome

Bulimia nervosa (*'oxlike hunger of nervous origin'*) is another form of eating disorder, which is characterised by bouts of binge eating and subsequent purging, with sufferers experiencing psychopathological concern with weight control and obtaining and maintaining their desired body image.

Bulimia has only been recognised as a clinical condition fairly recently (Russell, 1979). As with anorexia, the incidence of bulimia is considerably greater in females than males and again development is more common in peripubertal years. Bulimia is about 2-3 times more common than anorexia. Diagnosis of bulimia is dependent on the sufferer engaging in cyclical binge-purge behaviour at least 2-3 times a week for a period of 3 months. As with anorexics, pathological concern over weight and shape is a central feature. Lower (although still substantial) rates of mortality have been noted for bulimics compared to anorexics (Crow et al., 1999) with a mortality rate of approximately 2-4% over the course of 10 years (Keel et al., 1997).

Psychological consequences of the secrecy intrinsic to binge-purge behaviour are excessive feelings of shame, guilt and self-loathing. Laxatives, diuretics and emetics are used in attempt to compensate after binge eating. In contrast to anorexics, bulimics tend to feel extreme loss of control over their eating, which can result in experience of helplessness comparable to that seen in depression (see section 2.3.2).

As with anorexia, many unpleasant physical consequences result from bulimic behaviour: for example, erosion of dental enamel and sore throats from vomiting; rupturing of the stomach and oesophageal lining; arrhythmia; fluid retention; swollen salivary glands and fatigue. Bulimia is more difficult to detect than anorexia in that marked weight loss is not usually apparent (Walsh & Devlin, 1998). However, bulimics tend to be more receptive to treatment and are fully aware their behaviour is abnormal and destructive.

In a review of over 80 follow-up assessments on bulimic patient groups, full recovery was noted in about 50% of cases, but at least 20% continued to suffer from the condition, with around 30% of all cases having relapsed at some point during the first 4 years (Keel et al., 1997). Similar estimates of bulimic individuals in complete remission and those experiencing relapse were observed in a further study by Keel et al. (1999) in which outcome after at least 10 years was investigated. A lower rate of recovery (around 30% of cases) was reported by Johnson-Sabine et al. (1992); however, this could partly be a consequence of the shorter follow-up duration of 5 years, with relapse rates tending to decline as the duration of follow-up increases (Keel et al., 1999). Symptom severity was also shown to decrease over time in most cases.

Johnson-Sabine et al. (1992) also noted that social difficulties (particularly with intimates) were usually present in individuals experiencing poorer outcome. Likewise Rorty et al. (1999) found that bulimic individuals reported fewer individuals in their social network they could turn to for *emotional* support than controls. They were particularly dissatisfied with the availability and quality of emotional support from family members (as opposed to friends and peers) and also reported poor social functioning. Bulimics did not, however, view themselves as

lacking in more practical forms of support compared to controls, such as receiving advice or help. Deficits in both the quantity and quality of social support received by bulimic individuals were also observed by Tiller et al. (1997). Anorexic patients in the latter study, in contrast, did not perceive their degree of emotional support as unsatisfactory, although they reported a smaller social network than controls.

As bulimia is a very distressing condition, it is vital that affected individuals have people they can lean on who can help them to cope with and manage their condition and recovery. However, the secrecy inherent in the initial stages of the condition before treatment and the associated embarrassment and shame hinder the bulimic individual from achieving and fostering such close relationships. The remitted bulimic group in Rorty et al.'s (1999) study did appear to have more access to emotional support than the actively bulimic group, although they still expressed some dissatisfaction with the calibre of emotional support from kin members. Relative to control subjects they also experienced some residual deficits in social adjustment. It remains unclear whether these deficits are a consequence of the illness or whether they were in fact a predisposing factor to its onset.

## 7.3 AETIOLOGY

Researchers have put forward a number of putative risk factors for the development of eating disorders, resulting in various biological, social and psychological theories, which will be described in the following sections.

### 7.3.1 Serotonergic dysfunction

Dysregulation of serotonergic function is a recognised feature of eating disorders (Brewerton, 1995). Although not fully understood, similar neurotransmitter

dysfunction seems to be present in bulimia as in depression (see section 2.2), with decreased CNS serotonergic responsiveness apparent in bulimic individuals (Jimerson et al., 1997). Serotonin (5-HT) is associated with control of feeding behaviour; in animals, reduced levels have been shown to result in lack of experience of satiety and consequent weight gain (Tecott et al., 1995).

The etiological significance of serotonergic dysfunction in bulimia nervosa has not yet been established. Smith et al. (1999) reduced brain serotonin levels in remitted bulimic individuals in an attempt to assess its relative contribution to the pathogenesis of the condition. Subjects ingested either a tryptophan[2]-free solution or a solution that contained tryptophan on one of two separate test sessions. (Subjects fasted for over 8 hours overnight before testing and maintained a low-protein diet for the 24 hours prior to the test session). Following consumption of the tryptophan-free mixture, subjects with a history of bulimia nervosa reported increased worry about body image, feelings of loss of control over eating and lowered mood state, which did not result after ingestion of the balanced solution (containing tryptophan). This pattern of aversive symptomatology was not apparent in either condition in normal controls without a history of eating disorder. These findings suggest that reduced levels of serotonin can trigger clinical symptoms in recovered bulimics, which is perhaps particularly alarming in light of the fact that dieting (from which bulimia often evolves) can produce a decrease in brain serotonin (Anderson et al., 1990). Therefore dieting behaviours in individuals predisposed to bulimia could result in the onset or recurrence of the condition.

Pharmacological treatment with SSRI's (selective serotonin re-uptake inhibitors), for example, *fluoxetine* (Prozac), is relatively effective for bulimia

[2] Tryptophan is a precursor for serotonin

resulting in a significant reduction in the frequency of binge-purge episodes. A recent study by Goldstein et al. (1999) examined the efficacy of fluoxetine treatment on bulimics experiencing varying degrees of comorbid depressive symptomatology. As depression is frequently a concomitant of bulimia (Willcox & Sattler, 1996), Goldstein et al. wanted to determine whether or not fluoxetine's remedial effects were simply a consequence of its antidepressant properties, which was indicated by Fava et al. (1997). The number of binge-purge episodes was shown to reduce in all patients following treatment with 60mg daily dose of fluoxetine *regardless of level (or presence) of depressive symptomatology*. Although the exact mechanism through which the SSRI works is uncertain, this study would suggest that reduction in aversive symptoms in bulimia is not merely due to reduction in level of comorbid depression.

Anorexics do not appear to respond so well to SSRI therapy though (Attia et al., 1998), with little benefit being derived before weight is restored to a normal level (Kaye et al., 1998). An *increase* in serotonin responsiveness has been suggested as a possible contributing factor for anorexia (Treasure & Tiller, 1993), which would account for some of the observed pathophysiology as pharmacological elevation of brain serotonin level (using a 5-HT receptor agonist like *d-fenfluramine*) is associated with increased satiety and a reduction in appetite (Hill & Blundell, 1990). More evidence for the possible causal role of serotonergic dysfunction in anorexia is suggested by the finding of increased concentration of the primary 5-HT metabolite in weight-restored anorexics (Kaye et al., 1991), i.e., altered serotonin levels are not merely a transient consequence of malnutrition.

### 7.3.2 Social pressure

There is a higher incidence of eating disorders in Western cultures, which could be due in part to social pressures, although there have been rare reports of such conditions in other (more primitive) cultures. Thinness is aspired to and has become the blueprint for the so-called 'ideal' figure for women in modern Western societies (McCarthy, 1990), even though the improvement in nutrition over recent decades has resulted in an increase in the natural weight of females. In spite of being exposed to such pressures, the majority of women do not develop clinical eating conditions so although such social factors could be a contributing factor for onset of anorexia or bulimia, they are unlikely to be a cause.

### 7.3.3 Psychological factors

Lack of perceived control over one's life is believed to be a motivating factor for the onset of an eating disorder (Slade, 1982), particularly anorexia. The desire to obtain control over an aspect of one's life then becomes manifest in rigid control of body size and eating behaviour.

The next section describes another core psychological feature of eating disorders in more detail – body image dissatisfaction.

### 7.3.4 Body image disturbance

Body image disturbance is a central feature of both anorexia and bulimia. Researchers believe that it is not just a consequence of the illness but acts as a predisposing factor which can be accentuated by depressed mood and low self-esteem in women who are overly concerned with their figure (Cohen-Tovée, 1993; Cooper & Taylor, 1988). Induction of depressed mood in normal women who set

high importance on their body shape has been shown to result in increased body image concern with overestimation of body size resulting (Cohen-Tovée, 1993).

Of course, if perceptual distortion arises for the central concern of body image in eating disorders, it is also plausible that similar misinterpretation of other salient visual cues could arise, such as facial expressions (see section 7.4.1).

#### *7.3.4.1 Body size estimation and body image dissatisfaction*

Cash and Deagle (1997) conducted a meta-analysis on studies investigating body image disturbance. They highlighted the importance of not viewing body image disturbance as a unitary concept but instead one encapsulating at least two different aspects: perceptual body image distortion and attitudinal body dissatisfaction. The former concerns actual perceptual distortion, with eating disorder patients overestimating their own body size compared to its veridical size and the latter relates to their self-concerns and degree of repulsion with their own body image. Probst et al. (1998) also confirmed the multidimensional nature of body image distortion and the importance of examining perceptual, cognitive and affective aspects independently when examining this concept.

Cash and Deagle (1997) concluded that both perceptual and attitudinal types of body image distortion were present in individuals suffering from eating disorders compared to controls but the effect was greater for body dissatisfaction than for perceptual distortion. Within the perceptual distortion effects, overestimation of whole body size was found to be greater than the average of the distortion effect for specific body parts. Perceptual distortion of own body image does *not* appear to be a consequence of a more general sensory deficit as size estimation of neutral stimuli is accurate (Gardner & Moncrieff, 1988). Self-deprecating attitudes were again much

greater for eating disorder patients than controls, but effects were relatively larger for bulimics than anorexics. This discrepancy could be due to the emaciated anorexic being closer to her ideal weight and size and the normal weight bulimic not having achieved a marked reduction in body size.

Perception of attributes associated with varying body shapes was also shown to differ between anorexic and control women in a study by Baluch et al. (1997). Anorexic women rated pictures of fatter women as appearing significantly less attractive, less confident and more unpopular and unhealthy than either age-matched or teenage females did. Positive characteristics were instead associated with a thinner female body shape by anorexics. A preference for positive attributes to be associated with thinner figures was also exhibited by teenage, but not age-matched, controls, although larger body figures were not rated so negatively by either control group as with the anorexic group. Interestingly, anorexics did not rate larger male figures negatively and indeed rated all male shapes more positively than did their control counterparts. However, differences in body rating preferences according to sex of the stimulus figure do not seem to be apparent in normal subjects. Baluch et al. surmised that the presence of such body image distortions in anorexics might be used as a justification for their behaviour.

Lautenbacher et al. (1997) demonstrated that perceptual overestimation of body image does not normalise in weight-restored anorexics, implying that malnourishment is not the sole cause of body image disturbance. Overestimation of body size also appears to be a stable characteristic of bulimia and body size estimation has been shown to be additionally (unfavourably) influenced by low mood state and exposure to high-risk food (with regards to acts of binging) in a study by Carter et al. (1996).

Similar to the idea of depressive-realism mentioned in section 2.3.3, Doll and Fairburn (1998) reported that bulimics tended to be highly accurate at reporting their actual weight and height, whereas control subjects were inclined to underestimate their weight and overestimate their height (i.e., were optimistic with regard to their figure). Such an accurate estimation of weight seems perhaps surprising in view of bulimic individuals obvious dissatisfaction with their body image and perceptual distortion of their own figure mentioned above; however, their intense preoccupation with their body image is also likely to result in them being aware of how much they weigh.

#### *7.3.4.2 Selective attention to shape and weight related stimuli*

Modifications of the Stroop (1938) colour-naming test have been used by several groups of investigators to determine the type of stimuli that elicit processing biases in eating disorder patients (Sackville et al., 1998; Cooper & Todd, 1997). Sackville et al. reported that anorexic patients were slower (i.e., experienced interference) to colour-name words related to body shape (both extremes of thinness and fatness) and were also slightly delayed in naming words connoting high calorie food compared to controls. This bias for body shape related words was not present when stimulus visibility was reduced by background masking though, suggesting selective attention to these stimuli is not subconscious. In addition, delayed colour-naming latency was not present for emotionally valenced words (e.g., *happy*) indicating the effect is specific to stimuli of direct concern to anorexics. Anorexics and bulimics were also shown to experience interference for weight-related words in a study by Cooper and Todd (1997) also using a variation of the Stroop paradigm.

In order to reduce possible semantic and linguistic biases, Walker et al. (1995) designed a pictorial version of the Stroop task to assess body size concerns in anorexic, bulimic and control subject groups. Subjects had to colour-name coloured pictures of female body shapes differing in size from thin to obese (as well as control pictures, e.g., different coloured balls used for sport). As predicted, eating disorder subjects took more time to colour-name all the figures (i.e., from thin through to obese shapes) than control subjects did, suggesting a general preoccupation with body shape and weight and no differential effects depending on actual body size (under- or overweight). There were no differences between anorexic and bulimic groups.

Criticism has been levelled at the Stroop test as delayed response latency could indicate attention directed away from the stimulus instead of necessarily implying attention towards it. Rieger et al. (1998) countered this difficulty by assessing selective attention in eating disorder patients using an adapted version of the visual dot-probe paradigm (MacLeod et al., 1986). The visual probe detection procedure involves brief presentation of two stimuli at different spatial locations (e.g., one above the other) followed by the appearance of a dot in the same location as one of the two stimuli, with participants required to signal as soon as they detect the probe. Response latency should be shorter if subjects' attention was drawn to the stimulus in the same location as the probe.

Individuals suffering from eating disorders exhibited *delayed* latencies to detect probes that occurred in the same location as words connoting *thinness* (Rieger et al., 1998). In contrast, a trend revealed that attention appeared to be selectively directed towards words suggestive of a large physique. Eating disorder patients might therefore tend to selectively ignore positively valenced (undersize) body shape

related stimuli with preference directed instead towards negative (oversize) body image information, which could indicate why weight concerns remain prominent in spite of the availability of conflicting information.

#### *7.3.4.3 Selective attention to threatening stimuli*

The possibility that binge-eating behaviour might be induced following exposure to threat-related cues was explored in a study by Meyer and Waller (1999) using a non-clinical female sample. They found that individuals with high scores on the Eating Disorders Inventory (EDI; Garner et al., 1983) ate more food following exposure to subliminally presented threat-related verbal stimuli than to either positive ('*happy*'), neutral ('*gallery*') or appetitive ('*hungry*') cues. Indeed all subjects consumed more food if they had been exposed to the abandonment cue ('*lonely*') rather than the other cues. The negative emotion cue ('*hostile*') only resulted in increased eating in the high EDI group though. Although replication is obviously required with a clinical group, this study does suggest a preattentive bias towards threatening stimuli in individuals with eating disorder symptomatology. The possibility that overeating might partly function as an avoidance mechanism in bulimics after exposure to threat-related stimuli requires further investigation.

#### *7.3.4.4 Perception of others' body size*

The possibility that such focus on body image is not restricted to how the patient views herself but might also affect how she perceives others is raised in a paper by Beebe et al. (1996). Subjects with elevated scores on an eating disorders inventory tended to focus on the body image of others more than low scoring

subjects did and also expected others to place a fairly high value on their own body size. The results must be viewed with caution as a subclinical rather than clinical subject group was used.

A similar finding of more critical and negative estimation of body size of others in addition to oneself has been reported by Smeets (1999) in anorexic individuals. She used a morphing technique similar to that applied in this thesis, with a movie shown of a female figure changing from a thin to obese shape. Subjects were required to choose points along the continuum that corresponded with what they would consider was a thin, normal, fat and obese body shape for themselves and also for another person. Anorexic subjects were shown to mark all body weights closer to the thin end of the continuum than control subjects for the 'self' category. For example, the image started to look obese to anorexics approximately one third of the way along the continuum (starting at the thin end), whereas controls first observed the image as obese over half-way along. Similar results were revealed when anorexics were instructed to imagine the image was someone else, i.e., underestimating what appeared thin, normal and fat compared to controls (although the obese category was selected at approximately the same point). Again these results would seem to suggest that image distortions in anorexia are not necessary specific to the 'self' concept but might also affect both how others are viewed and expectations of how others should perceive themselves.

#### *7.3.4.5 Self-disparagement*

Ben-Tovim and Walker (1992) examined body attitudes over a range of different subject groups (anorexic, bulimic, physically ill, psychiatric controls and normal controls) and discovered, as predicted, that eating disorder patients exhibited

greater concern over their weight and shape than other subject groups. However, the most interesting aspect of this study was the finding that the eating disorder group was best distinguished from other groups according to questionnaire subscales relating to 'body disparagement' and 'attractiveness' (i.e., more so than 'salience of weight and shape' or 'feeling fat' subscales).

Such findings imply that more emphasis should be directed towards identifying and correcting maladaptive feelings of extreme body loathing and hatred in treatment. It is also perhaps notable that anorexic and bulimic groups did not differ at all in their attitudes, in spite of the different behavioural manifestations of each disorder (i.e., starvation versus binge-purge behaviour) and also varying body sizes associated with each condition (i.e., emaciation versus approximately normal body weight). Such intense feelings of self-disgust are likely to affect how eating disorder patients process the emotion of disgust and might indicate that they will be overly sensitive to the expression of disgust in others (see section 7.4.1).

#### *7.3.4.6 Specificity?*

Joiner et al. (1997) have commented on the specificity of body dissatisfaction to eating disorders as depressives in their study (with varying degrees of comorbid bulimic symptomatology) were shown to have similarly high levels of low self-esteem regarding body satisfaction as the bulimic subjects did. It is possible that feelings of body dissatisfaction in depressed individuals merely relate to a more global negative self-esteem concept about themselves rather than being purely contingent on body image. More research is required with other non-bulimic psychiatric patient groups to determine whether the strong relation of global self-esteem to body satisfaction is only central and unique to eating disorders.

### *7.3.4.7 Development of negative body image*

The possibility that lack of touch in childhood might contribute to body image dissatisfaction in anorexics and bulimics was explored in a study by Gupta and Schork (1995). In a non-clinical sample, females were found to be more likely to currently desire more physical contact and nurturing (e.g., hugging) and have the belief that they were relatively deprived of touch in childhood if they also reported an elevated desire for thinness. Increased body dissatisfaction was also associated with lack of tactile nurturing when young. Correlations between body satisfaction or desire for thinness and touch deprivation were not significant in males though. Previous research has indicated the importance of hugging from the primary caregiver during childhood in the development of an accurate and positive body image (Kreuger, 1989; Huang et al., 1976). Although replication of such a study is necessary before generalisations are made to individuals with body image pathology, these findings suggest that tactile stimulation, particularly in childhood and also at later stages, might be an important defence against the development of body image dissatisfaction.

## 7.4 EXPERIENCE OF EMOTION

The construct of *alexithymia* was first described by Nemiah and Sifneos (1970) and embodied the difficulty observed in some psychiatric patients to identify, experience and express emotion and to distinguish feelings from physical sensations (for example, the sensation of tingling skin being interpreted as feeling afraid). Inability to identify feelings appears to be a prominent psychological feature of eating disorders with several studies (Troop et al., 1995; Schmidt et al., 1993) demonstrating that most anorexic and bulimic individuals score significantly higher

than controls on the Toronto Alexithymia Scale (TAS; Taylor et al., 1985). In both of these studies a relatively greater preponderance for alexithymic traits was exhibited by anorexics than bulimics. Although level of depression has been shown to influence this ability to differentiate between emotional states and bodily sensations in eating disorder patients (Sexton et al., 1998), deficits in expressing feelings to others were still found to be present in anorexics with restrictor subtype, although not bulimics, after comorbid depression had been factored out. Bourke et al. (1992) also demonstrated a high level of alexithymic traits in a group of anorexics regardless of concomitant depressive state.

Difficulty in self-expression might account for clinical observations of the problems anorexic patients have forming close relationships and their feelings of alienation from others. Diminished communication and expression of feelings and lack of fantasy (i.e., daydreaming) have also been reported in anorexics by Troop et al. (1995), again distinguishing them from both bulimics and controls who did not experience such difficulties. Although alexithymic traits might not be so severe in bulimia, Schmidt et al. (1993) demonstrated that alexithymia is nonetheless a stable attribute in bulimics, with high scores on the TAS persisting in spite of substantial symptomatic improvement. Both groups of researchers (Troop et al., 1995; Schmidt et al., 1993) have commented that the identification and conveyance of feelings in treatment might be a vital (and necessary) component for long-term recovery in eating disorders.

Smith et al. (1997) also examined empathic ability in eating disorder patients by asking subjects to describe their impression of a face (with an ambiguous expression) which was repeatedly flashed on a screen at a brief exposure rate. To help manipulate emotional response, a subliminal prime of an emotionally charged

phrase – 'I ILL', 'I WELL' or the control 'I' - was presented prior to the face. Although patients tended to use more emotional words to summarise their impressions of how the person displayed looked and felt than control subjects, the manner in which they utilised emotional descriptions seemed to be rather maladaptive in the respect that the phrases used suggested misunderstanding of emotion, diminished self-comprehension and avoidance of empathy. (An example given for the description after the 'I WELL' prompt is: *"There is something more happy about the face... I don't know what she is feeling."*) The authors concluded by stating that individuals suffering from eating disorders showed a "*...pronounced incapacity for emotional understanding*".

### 7.4.1 Differential processing of facial expressions?

Difficulty in expression of feelings and understanding of one's own emotional state might extend to difficulties in interpretation of other individuals' emotions. Anorexics and bulimics might therefore experience more problems comprehending others facial expressions and might require a greater intensity of expression to perceive the underlying emotion. Interpersonal problems are prevalent in eating disorders. Whether social difficulties are a consequence or contributing factor to the onset of the disorder remains unclarified, but it is probable that they could be exacerbated by incorrect interpretation of others' facial expressions which might result in inappropriate responding by the eating disorder sufferer in social situations. If anorexic or bulimic individuals are unable to detect more subtle nuances of emotion expressed by others, due to their problems in awareness of their own emotional state, their social development is liable to be hindered. If they do not

respond to others sufficiently (or in a suitable manner), people might tend to withdraw from their company (cf. depression, see Chapter 5).

An additional factor, which might result in altered perception of specific facial expressions in eating disorder patients, is the well-documented self-loathing and disgust of their body image (Ben-Tovim & Walker, 1992; see section 7.3.4.5). Such heightened experience of disgust, both with their physical being and presumably with their behaviour (i.e., shame and guilt following binge-purge acts in bulimics) could alter sensitivity to other individuals' expressions of disgust. If individuals with eating disorders are overly sensitive to others' opinions of them, as some studies suggest (Pliner & Haddock, 1996; see section 7.2.1.), and strive to attain approval, enhanced sensitivity to facial expressions of disgust might result. Individuals suffering from eating disorders might therefore respond overly to others' mild expressions of disgust, which could in turn heighten their own feelings of low self-esteem and lack of self-worth. Indeed, high levels of maternal criticism have been shown to be predictive of poorer outcome in patients with eating disorders (van Furth et al., 1996).

Sensitivity to disgust-related stimuli was shown to be enhanced in anorexic patients (particularly in relation to bodily products) by Davey et al. (1998). In addition, this study also revealed that eating disorder symptomatology was associated with increased sensitivity to disgust in female students. However, Troop et al. (in press) failed to replicate this finding in a clinical group, with only non-significant trends being apparent between sensitivity to disgust-related stimuli and presence of eating disorder symptoms. (It is perhaps notable though that enhanced sensitivity to disgust did *not* correlate with general psychopathology in this study.)

The frequent vomiting behaviour representative of bulimia might also influence sensitivity to disgust in bulimics. Normally retching occurs in response to noxious stimuli in order to remove poisonous substances quickly and effectively from the body. This stimulus-response pattern becomes uncoupled in bulimics with vomiting being induced voluntarily in an attempt to compensate following binge eating episodes due to extreme feelings of self-disgust, as opposed to purely in response to a disgusting stimulus. Heightened experience of the emotion of disgust and disgust-related behaviour such as vomiting could cause the bulimic to be overly sensitive to either disgusting stimuli or stimuli signalling a disgust response, such as others' facial expressions of disgust.

It is perhaps important to note that the opposite finding was observed in obsessive-compulsive patients (Sprengelmeyer, 1997b), who associate disgust with stimuli that other individuals do not find aversive, i.e., one would assume that individuals with obsessive-compulsive disorder (OCD) would be highly responsive to disgust-related stimuli. OCD subjects were, however, found to be selectively impaired in their recognition of disgust in others compared to controls. The authors proposed a deficit in identification of disgust might be due to a loss of association between others' expressions of disgust and experience of disgust in OCD individuals, as OCD individuals are frequently disgusted by stimuli that other people would not express disgust towards. Although it is possible a similar pattern might arise here in perception of disgust in individuals with eating disorders, it seems much more intuitive that sensitivity to disgust will be *enhanced* in these individuals due to greater experience of the emotion. The fact that *recognition of*, rather than *sensitivity to*, disgusted expressions was examined in the Sprengelmeyer et al. study should also be considered.

Comorbid experience of depression in eating disorders could also result in some of the changes in facial expression perception documented in Chapter 5. For example, patients might also be relatively less sensitive to happy expressions compared to controls in that their depressive state is liable to result in feelings of anhedonia and loss of positive affect. However, it is also possible that the effects demonstrated in depressives might not be apparent in eating disorder patients as depressive state is believed by many merely to be a secondary consequence of malnutrition.

## 7.5 STUDY PREDICTIONS

In summary then, the predictions for the study of facial expression perception in eating disorders were as follows. It was anticipated that individuals suffering from eating disorders would be less sensitive overall to all facial expressions and require a greater intensity of each emotion to first perceive it, due to reported difficulties in identification, experience and expression of emotion (see section 7.4). In addition, selective alterations in relative pattern of sensitivity across the emotions were anticipated, with eating disordered individuals expected to be relatively more sensitive to expressions of disgust than control subjects as a consequence of intense self-loathing and desire for approval from others (see sections 7.3.4.5 and 7.2.1). A secondary hypothesis was that bulimic individuals' might be differentially more sensitive to disgust than anorexics due to the repeated vomiting inherent in the condition. Another possibility was that the ED group might exhibit decreased relative sensitivity to happy expressions and perhaps increased sensitivity to sadness compared to controls due to comorbid depressive symptomatology, in line with the findings in Chapter 5.

## 7.6 METHODS

Anorexic and bulimic subjects were combined to form a general eating disorder group to increase the subject number as no major differences in response pattern were expected between the different conditions. (A comparison of results of the two groups is however conducted as a subsidiary analysis, see section 7.9.) The following section analyses and compares the results of the eating disorder and control groups.

The materials, interactive test and procedure used were identical to those in Chapter 5, with the addition of the Eating Disorders Inventory. The EDI is a widely used questionnaire, which assesses tendency for eating disorder symptomatology (see Appendix 7.1). Questions are divided into the following eight subscales to evaluate the preponderance of different eating disorder traits: '*drive for thinness*', '*body dissatisfaction*', '*bulimia*', '*perfectionism*', '*interpersonal distrust*', '*ineffectiveness*', '*interoceptive awareness*' and '*maturity fears*'.

### 7.6.1 Participants

There was a total of 22 participants in the eating disorder group. 14 of these patients had a primary diagnosis of anorexia nervosa (restrictor subtype, ICD-10 of F50.0) and ranged in age from 16-42. The remaining 8 individuals had a primary diagnosis of bulimia nervosa (ICD-10 of F50.2) with an age range of 15-34. All patients were female.

Anorexia nervosa patients were recruited from an inpatient specialist clinic for eating disorders at the Bethlem Royal Hospital, London. Participants with bulimia nervosa were recruited through an outpatient clinic attached to the Maudsley Hospital, Institute of Psychiatry, London. The Beck Depression Inventory (BDI) was

used to assess depressive state in eating disorder patients. Pulos (1996) demonstrated the BDI was a valid instrument to use with such a population (in spite of the symptom overlap between eating disorders and depression). Participation in the study was entirely voluntary and did not affect the patient's treatment in any way. Pharmacological treatment was not controlled for.

14 females with an age range of 18-36 were selected for the control group. Subjects satisfied criterion for inclusion in the control group if they scored less than pathological cut-off values on each scale of the EDI and if they reported not having had an eating problem at any point[3]. In addition, subjects had to score less than 10 on the BDI. (As a consequence of the strict cut-off scores for both the EDI and the BDI, there were fewer participants in the control group than in the eating disorder group.)

All subjects gave informed consent to participate in the study (see Appendix 7.2 for the patient consent form).

Ethical approval for this study was obtained from both the Ethics Committee for the School of Psychology, University of St Andrews and also from the Bethlem and Maudsley NHS Trust for recruitment and testing of controls and individuals with eating disorders respectively.

[3] Two criteria for presence of an eating disorder were useful as subjects might not *currently* be experiencing problems but might have experienced eating disorder symptomatology in the past, or conversely might not report having an eating disorder but might actually score over healthy cut-off values for eating disorder pathology.

## 7.7 RESULTS OF EATING DISORDERS GROUP VERSUS CONTROLS

### 7.7.1 Comparison of age and BDI score for control and eating disorder groups

The average age of the control group was 24.7 years (SE 1.6) compared to 26.0 years (SE 1.5) for the eating disorder group. A between-subjects t-test (equal variance) revealed no significant age difference between the two groups, $t(34)=-0.6$, $p=0.6$.

The average BDI score for the control group was 3.2 (SE 0.9) in contrast to an average of 25.8 (SE 2.2) in the eating disorder group. (BDI data from 3 patients, 1 anorexic and 2 bulimics, were not available.) This difference was shown to be highly significant with a between-subjects (unequal variance) t-test, $t(22.7)=-9.7$, $p=0.001$.

### 7.7.2 Recognition accuracy

A 2*6 mixed-design ANOVA, with subject group (control versus eating disorder) as the between-subjects factor and expression as the within-subjects factor (6 levels), was conducted on the recognition accuracy data.

As expected there was a within-subjects main effect of expression, $F_{(3.0,102.8)}$[4]$=13.5$, $p=0.001$. Accuracy across all subjects was greatest for happiness at 99.1%, then anger at 97.2%, fear at 95.8%, disgust at 92.6%, sadness at 91.7% and surprise at 78.2%. The relatively high misidentification of surprise by both groups seemed to be a function of incorrect recognition of one particular 'surprise' face as fear (Ekman identity 'wf'). Overall ability to recognise the emotions at 150% did not significantly differ between the two subject groups, $F_{(1,34)}=0.1$, $p=0.7$, nor was there

[4] The Greenhouse-Geisser correction is used throughout as the within-subjects factor has more than 2 levels.

a significant interaction between expression and group, $F_{(3.0,102.8)}=0.2$, p=0.9, (see Figure 7.1 and Table 7.1).

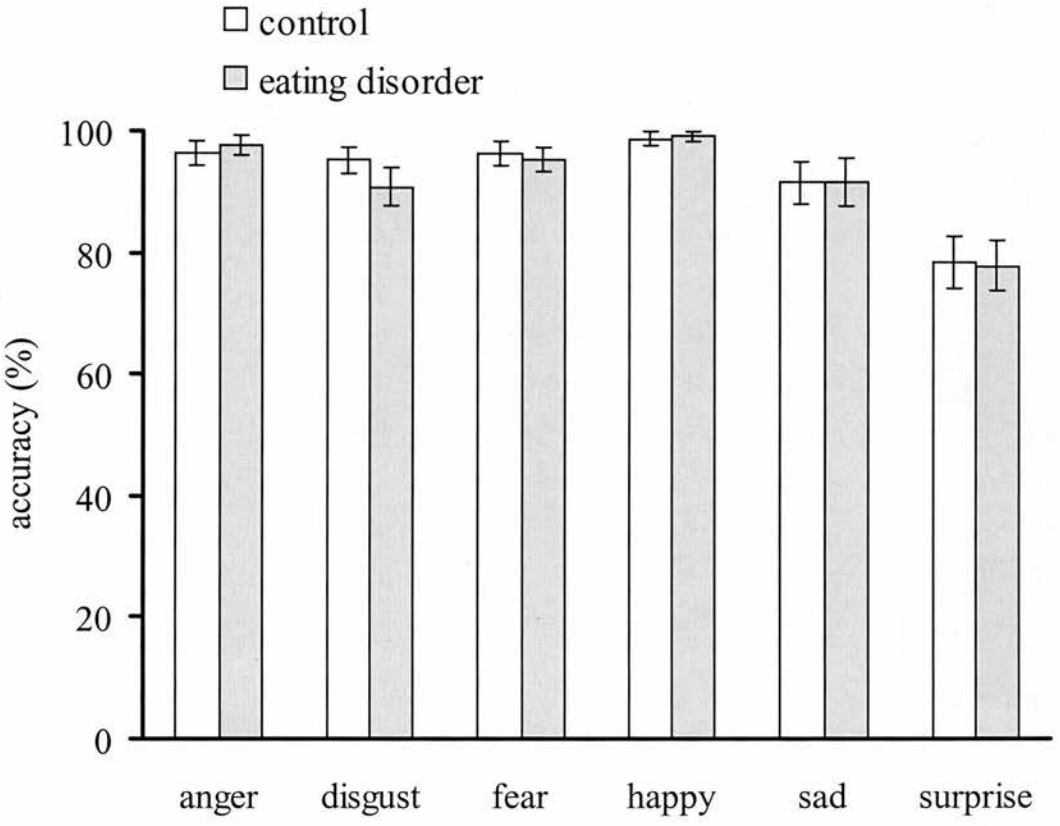


**Figure 7.1: A comparison of recognition accuracy (mean and SE) for the 6 emotions between control (n=14) and eating disorder (n=22) groups.**

| ACTUAL EMOTION | MISINDENTIFIED AS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | anger | | disgust | | fear | | happy | | sad | | surprise | |
| | ANGER | | | **1.5** | 1.2 | **0.8** | 2.4 | - | - | - | - | - | - |
| | DISGUST | **8.3** | 4.8 | | | - | - | - | - | **0.8** | - | - | - |
| | FEAR | - | - | - | - | | | - | - | - | - | **4.5** | 3.6 |
| | HAPPY | - | - | - | - | - | - | | | **0.8** | 1.2 | - | - |
| | SAD | **0.8** | - | - | 2.4 | **5.3** | 6.0 | - | - | | | **2.3** | - |
| | SURPRISE | **0.8** | - | - | - | **20.5** | 21.4 | **0.8** | - | - | - | | |

Table 7.1 Error pattern for eating disorder (bold) and control (normal type) groups. The value given is the average percentage of errors made across the group of subjects.

### 7.7.3 Absolute sensitivity

Again a 2*6 mixed-design ANOVA was conducted with subject group (control versus eating disorder) as the between-subjects factor and expression as the within-subjects factor.

There was a main effect of expression, $F_{(4.0,137.0)}=21.9$, $p=0.001$, with all subjects finding happiness easiest to detect at 59.3%, then surprise at 69.4%, disgust at 70.8%, fear at 71.0%, anger at 74.7% and finally sadness at 84.8%. (These averages and the order across the emotions are comparable to the averages across both groups in section 5.3.3.) There was a main effect of group, $F_{(1,34)}=7.1$, $p=0.012$. As Figure 7.2 reveals, individuals suffering from an eating disorder required more intensity to perceive *all* the emotions than control subjects did. The interaction

between expression and group was also significant, $F_{(4.0,137.0)}=2.6$, $p=0.040$, which can be more easily examined in the relative sensitivity analysis in the next section.

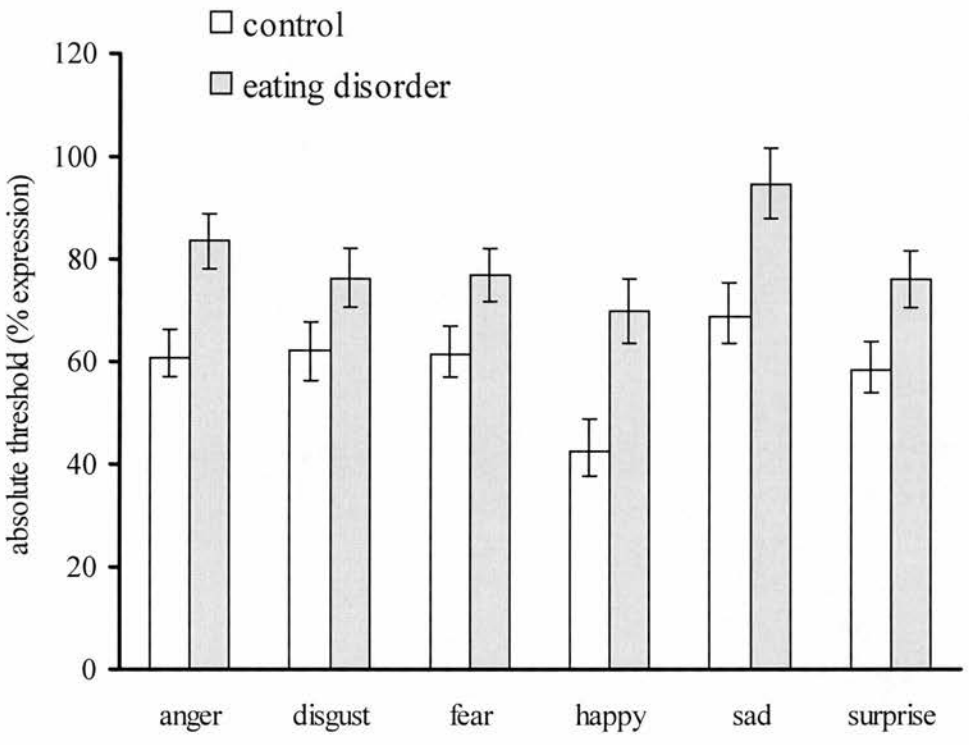


**Figure 7.2: A comparison of absolute sensitivity level (between 0-150%) for the 6 emotions between control (n=14) and eating disorder (n=22) groups.**

### 7.7.4 Relative sensitivity

As in the previous experimental chapters, the question of interest was whether differences in sensitivity would be present *across* the six different emotions according to subject group, so a contrast measure (see section 5.3.4) was used to address this issue.

Again a 2*6 mixed-design ANOVA was conducted. A significant main effect of expression was once again present, $F_{(3.9,133.5)}=23.1$, $p=0.001$. There was no main

effect of group as expected due to the contrast measure, $F_{(1,34)}=0.0$, p=1.0. The interaction between expression and group was significant, $F_{(3.9,133.5)}=2.6$, p=0.038.

Post-hoc Tukey HSD tests carried out on the interaction revealed that control and eating disorder subjects differed significantly on relative sensitivity to happiness, p=0.006, and to disgust, p=0.034. As illustrated by Figure 7.3, the eating disorder group are relatively *less* sensitive to happiness than the control group, but relatively *more* sensitive to disgust than controls.

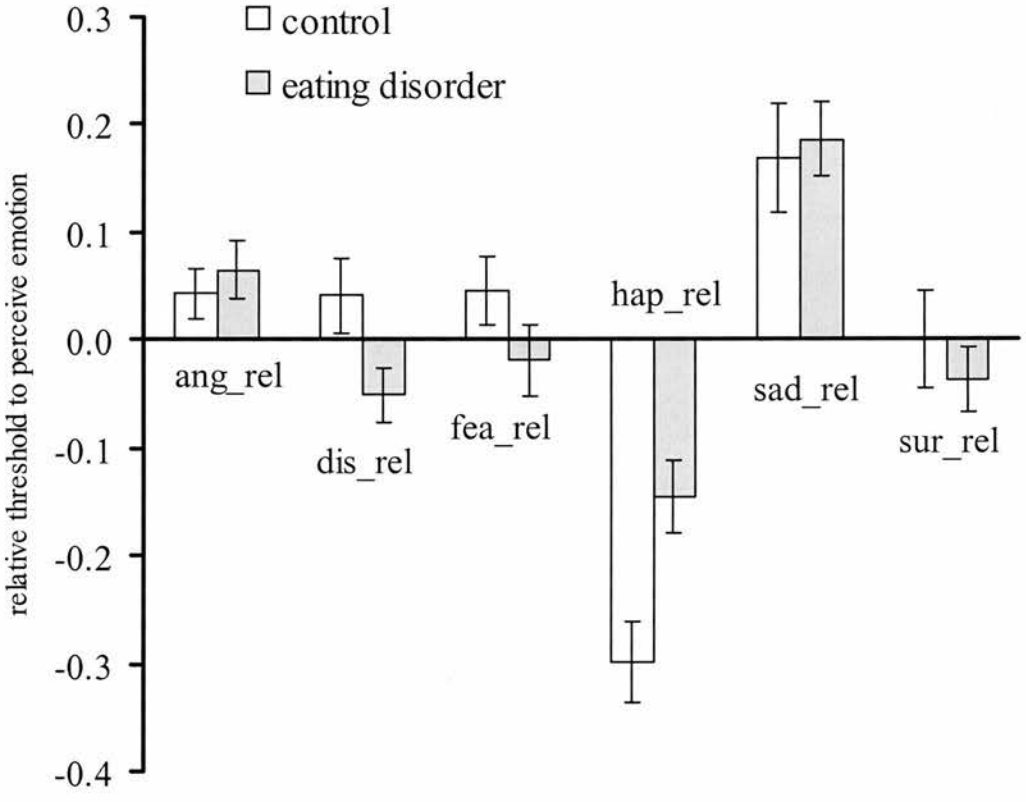


**Figure 7.3: A comparison of pattern of relative sensitivity across the 6 emotions between control (n=14) and eating disorder (n=22) groups. A high threshold denotes decreased sensitivity.**

## 7.8 COMPARISON WITH RESULTS OF DEPRESSED PATIENTS

As both similarities and differences seem apparent between results of the eating disorders group and those of the clinically depressed group in Chapter 5, an additional analysis has been conducted contrasting the 3 groups – control, eating disorder and clinically depressed. This subsidiary analysis is also an attempt to establish whether the alterations in facial expression processing in eating disorders are merely a secondary function of comorbid depressive symptomatology.

Again there were 14 control subjects, who scored below cut-offs for both the EDI and the BDI, 22 participants in the eating disorder group and 15 participants in the clinically depressed group (according to the number of female depressed subjects; see Chapter 5). (None of the clinically depressed participants had a secondary diagnosis of an eating disorder.) All participants were female. (See Table 7.2 for group details.)

| | **CONTROL (n=14)** | **EATING DISORDER (n=22)** | **CLINICALLY DEPRESSED (n=15)** |
|---|---|---|---|
| **AGE** | **24.7** (1.6) | **26.0** (1.5) | **40.7** (2.7) |
| **BDI SCORE** | **3.2** (0.9) | **25.8** (2.2) | **29.3** (1.9) |

Table 7.2 Average age and BDI score for the 3 groups. Standard error values are given in brackets

### 7.8.1 Recognition accuracy

A 3*6 mixed-design ANOVA with group as the between-subjects factor and expression as the within-subjects factor was conducted on the data.

There was a main effect of expression, $F_{(3.5,166.5)}=16.0$, $p=0.001$, but neither a main effect of group, $F_{(2,48)}=0.7$, $p=0.5$, nor an interaction between group and expression, $F_{(3.5,166.5)}=0.8$, $p=0.5$. Once again there appears to be no difference between groups on ability to identify 150%-caricatured emotions (see Figure 7.4).

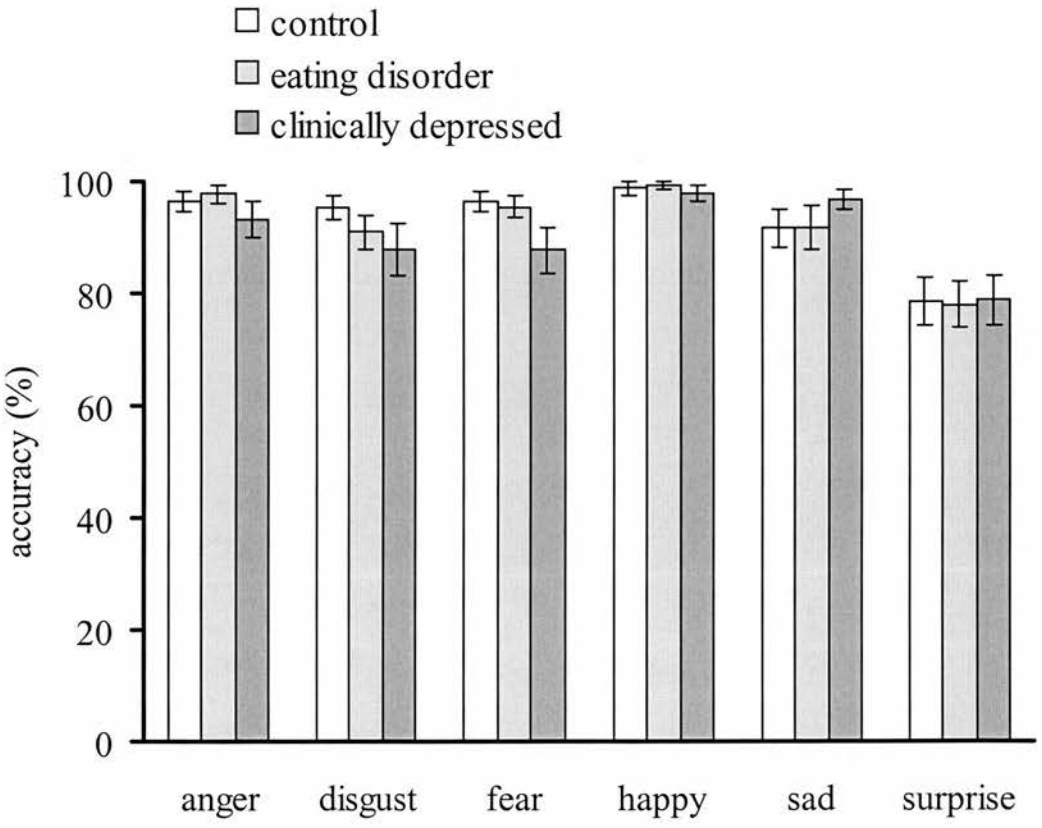


**Figure 7.4: A comparison of recognition accuracy (mean and SE) for the 6 emotions between control (n=14), eating disorder (n=22) and clinically depressed (n=15) groups.**

### 7.8.2 Absolute sensitivity

A 3*6 mixed-design ANOVA revealed a main effect of expression, $F_{(4.2,199.5)}=19.7$, $p=0.001$, a main effect of group, $F_{(2,48)}=4.2$, $p=0.021$, and a significant interaction between group and expression, $F_{(4.2,199.5)}=3.0$, $p=0.003$. Post-hoc Tukey HSD tests revealed that the control group and the eating disorders group differed in absolute sensitivity across all the emotions, $p=0.015$, with the eating disorders group requiring a greater intensity of any emotion to first perceive it (see Figure 7.5). Neither the difference in absolute sensitivity between the control and clinically depressed groups ($p=0.2$) nor that between the eating disorders and clinically depressed groups ($p=0.5$) was significant. The interaction between group and expression will be examined in the relative sensitivity analysis in the subsequent section.

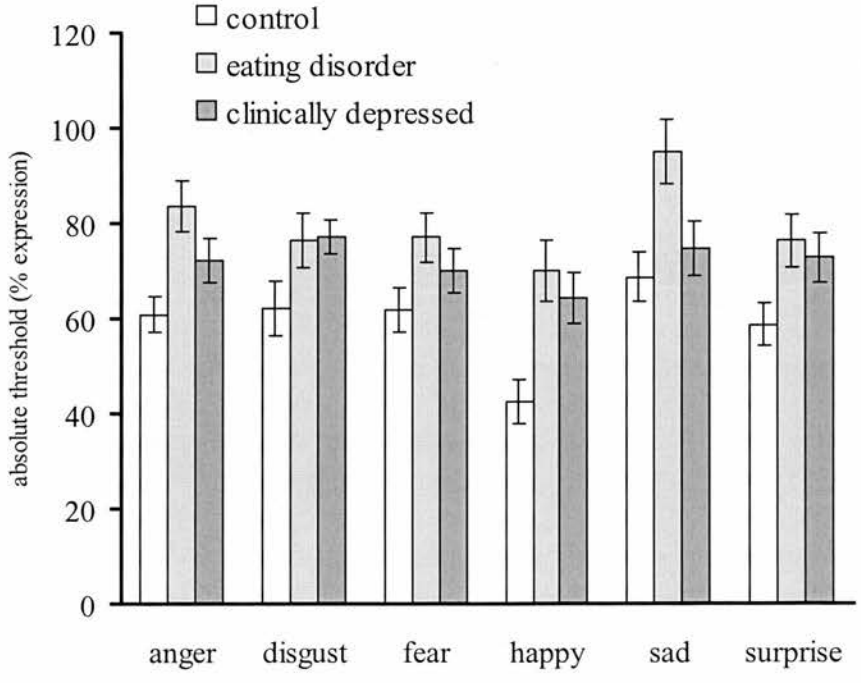


**Figure 7.5: A comparison of absolute sensitivity level (between 0-150%) for the 6 emotions between control (n=14), eating disorder (n=22) and clinically depressed (n=15) groups.**

### 7.8.3 Relative sensitivity

A 3*6 mixed-design ANOVA revealed a main effect of expression, $F_{(4.0,194.3)}=20.7$, $p=0.001$, and an interaction between group and expression, $F_{(4.0,194.3)}=3.3$, $p=0.001$. As expected due to the contrast measure there was no main effect of group, $F_{(2,48)}=0.0$, $p=0.95$. Post-hoc Tukey HSD tests revealed differences between groups for relative sensitivity to disgust, happiness and sadness (see Figure 7.6). The eating disorders and clinically depressed groups differed in relative sensitivity to disgust with the eating disorder group being relatively *more* sensitive than the clinically depressed group, $p=0.012$. For happiness, both the eating disorder and the clinically depressed groups differed from the control group ($p=0.021$ and $p=0.014$ respectively) - both groups were relatively *less* sensitive to happy expressions than controls. In addition, the clinically depressed group were revealed to be relatively *more* sensitive to sad expressions than the eating disorder group ($p=0.017$).

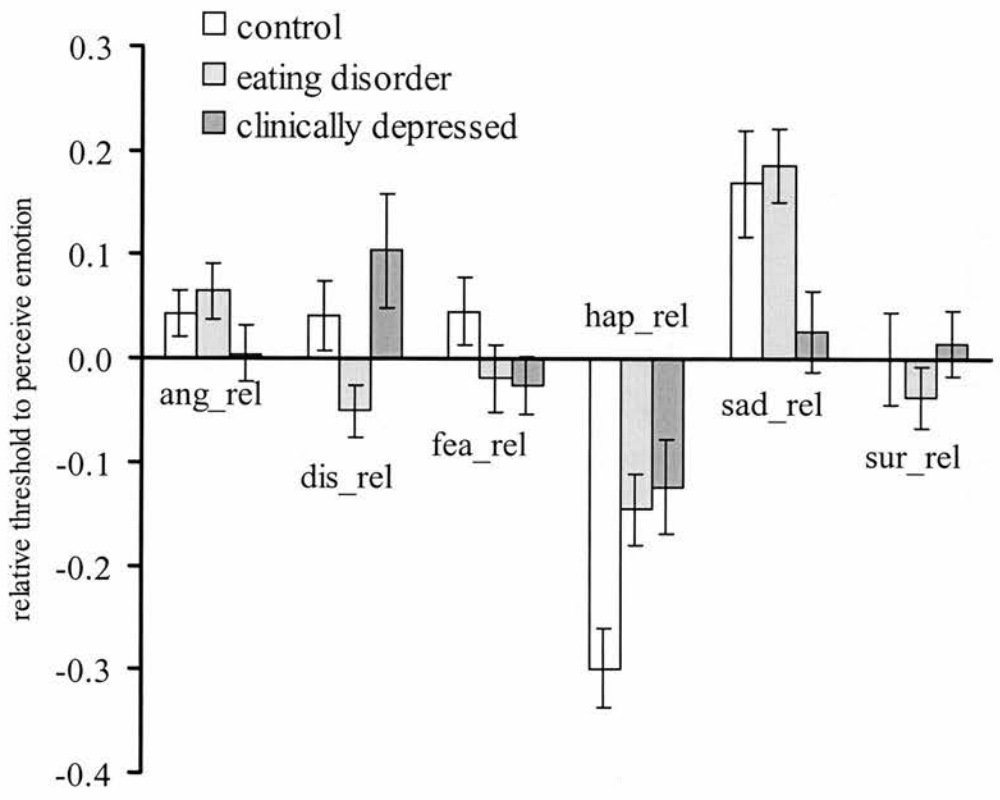


**Figure 7.6: A comparison of pattern of relative sensitivity across the 6 emotions between control (n=14), eating disorder (n=22) and clinically depressed (n=15) groups. A high threshold denotes decreased sensitivity.**

## 7.9 COMPARISON OF ANOREXIC AND BULIMIC SUBJECT GROUPS

A subsidiary analysis comparing data from anorexic and bulimic groups was conducted to examine whether differences were present in facial expression perception according to differential diagnosis of type of eating disorder and variation in symptom expression.

### 7.9.1 Comparison of age and BDI score for the two eating disorder groups

The average age of the AN group was 26.5 years (SE 2.1) compared to 25.3 years (SE 2.1) for the BN group. A between-subjects t-test (equal variance) revealed no significant age difference between the two groups, $t(20)=0.4$, $p=0.7$.

The average BDI score for the AN group was 27.3 (SE 2.8) compared to 22.5 (SE 3.3) for the BN group. No significant difference between these scores was shown in a between-subjects t-test (equal variance), $t(17)=1.0$, $p=0.3$.

### 7.9.2 Recognition accuracy

A 2*6 repeated measures ANOVA was conducted on the data with subject group as the between-subjects factor and expression as the within-subjects factor. There was a main effect of expression, $F_{(3.0,59.9)} =8.0$, $p=0.001$, but neither the main effect of group, $F_{(1,20)}=0.2$, $p=0.6$, nor the interaction between group and expression, $F_{(3.0,59.9)}=1.3$, $p=0.3$, was significant. There therefore appears to be no difference in recognition ability between anorexic and bulimic groups when using 150%-caricatured images (see Figure 7.7). There is also no major difference in the pattern of errors made between the two groups (see Table 7.3).

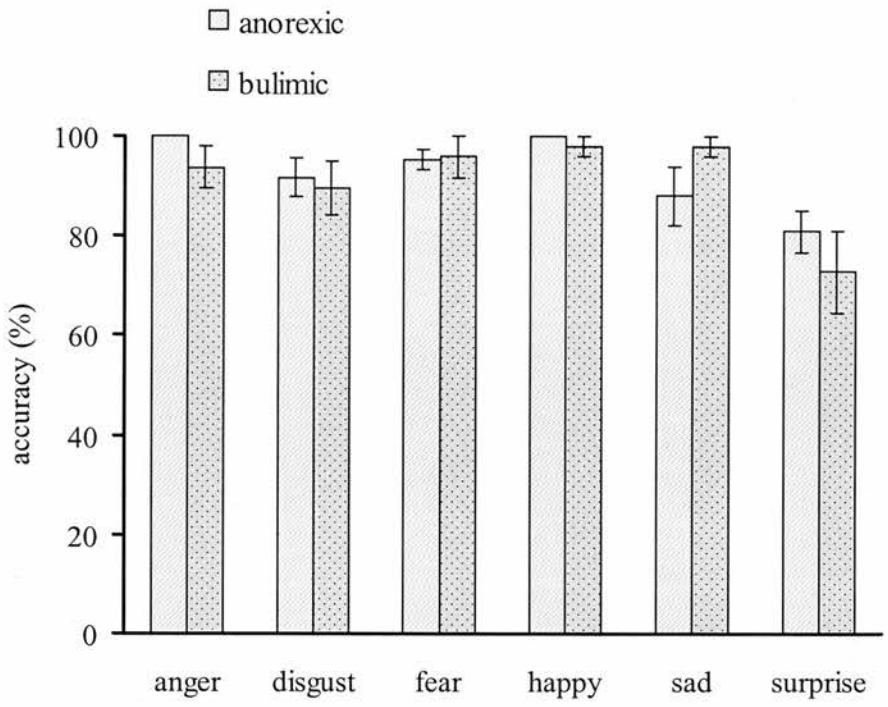


**Figure 7.7: A comparison of recognition accuracy (mean and SE) for the 6 emotions between anorexic (n=14) and bulimic (n=8) groups.**

| | | MISINDENTIFIED AS | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | anger | | disgust | | fear | | happy | | sad | | surprise | |
| ACTUAL EMOTION | ANGER | | | - | 4.2 | - | 2.1 | - | - | - | - | - | - |
| | DISGUST | **8.3** | 8.3 | | | - | - | - | - | - | 2.1 | - | - |
| | FEAR | - | - | - | - | | | - | - | - | - | **4.8** | 4.2 |
| | HAPPY | - | - | - | - | - | - | | | - | 2.1 | - | - |
| | SAD | **1.2** | - | - | | **7.1** | 2.1 | - | - | | | **3.6** | - |
| | SURPRISE | **1.2** | - | - | - | **17.9** | 25.0 | - | 2.1 | - | - | | |

Table 7.3 Error pattern for anorexic (bold) and bulimic (normal type) groups. The value given is the average percentage of errors made across the group of subjects.

### 7.9.3 Absolute sensitivity

A 2*6 mixed-design ANOVA was conducted on the absolute sensitivity scores. Again there was a significant main effect of expression, $F_{(3.8,75.1)}=12.7$, $p=0.001$, but no main effect of either group, $F_{(1,20)}=0.2$, $p=0.6$, or an interaction between group and expression, $F_{(3.8,75.1)}=0.5$, $p=0.7$ (see Figure 7.8). These results indicate no major differences in absolute sensitivity between the two eating disorder groups.

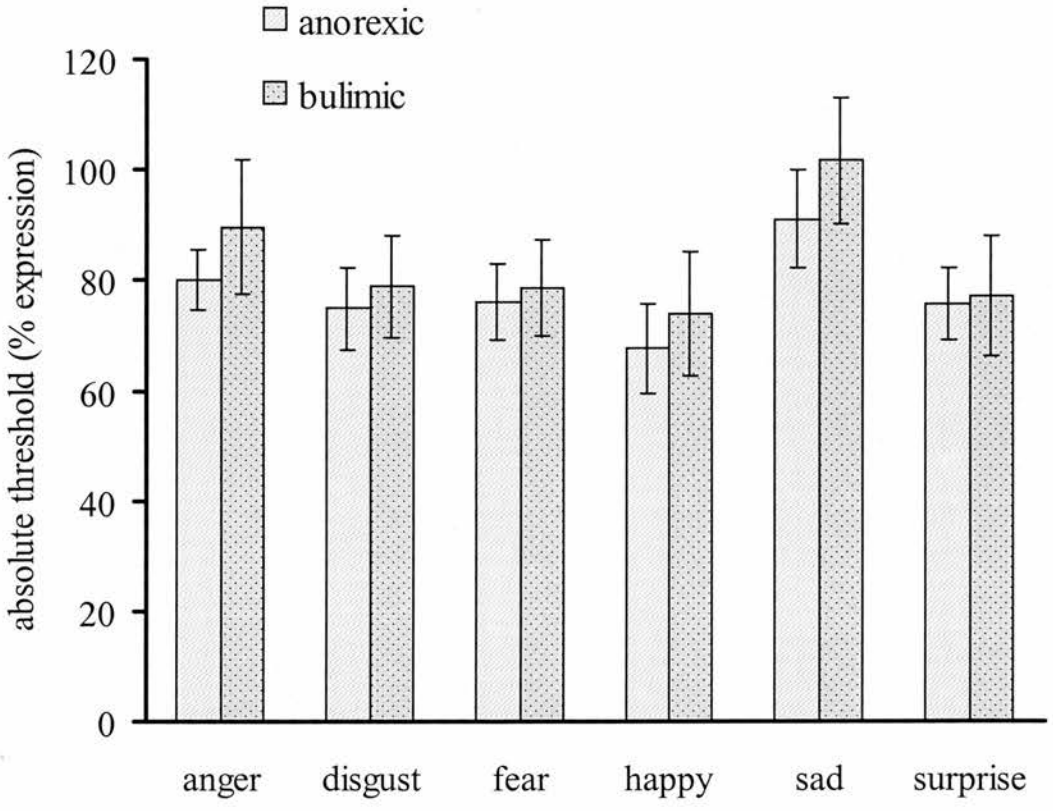


**Figure 7.8: A comparison of absolute sensitivity level (between 0-150%) for the 6 emotions between anorexic (n=14) and bulimic (n=8) groups.**

### 7.9.4 Relative sensitivity

A 2*6 mixed-design ANOVA was carried out on the relative sensitivity data. The main effect of expression was significant, $F_{(3.5,70.3)}=10.6$, $p=0.001$. There was no main effect of group, $F_{(1,20)}=0.0$, $p=1.0$, as anticipated due to the contrast measure used (see section 4.3.2.3). The interaction between group and expression was not significant, $F_{(3.5,70.3)}=0.4$, $p=0.8$, implying no differences between the two groups in pattern of sensitivity across the different emotions (see Figure 7.9).

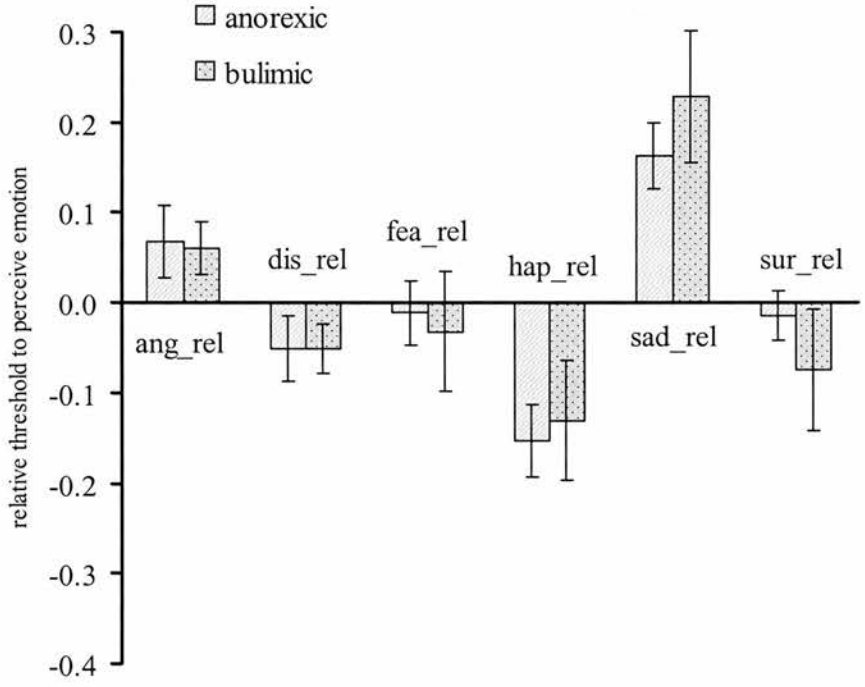


**Figure 7.9: A comparison of pattern of relative sensitivity across the 6 emotions between anorexic (n=14) and bulimic (n=8) groups. A high threshold denotes decreased sensitivity.**

Lack of any differences in results on any of the expression perception measures between the anorexia nervosa and bulimia nervosa groups provides justification for the combination of the two groups into a larger eating disorder group, which also allowed the analyses greater power due to the larger subject number.

## 7.10 DISCUSSION

These results reveal that individuals suffering from eating disorders, like depressives, have differential sensitivity to facial expressions compared to normal individuals.

### 7.10.1 Overall sensitivity level

Although the eating disorders group was just as capable of accurately identifying all emotions at the 150%-caricatured endpoint as control and clinically depressed subjects, the point at which they could first recognise the emotion (from neutral) required significantly more intensity (i.e., greater percentage emotion) than controls. Clinically depressed patients showed a similar pattern of decreased sensitivity to all emotions (see section 5.3.3), although, as the 3-way comparison reveals here, this effect seems to be slightly stronger in the eating disorder group.

Diminished overall sensitivity to facial expressions of emotion in others could relate to the difficulties eating disorder patients have in understanding their own emotional state and identifying and differentiating between emotions experienced, i.e., alexithymia (Smith et al., 1997; Troop et al., 1995; Schmidt et al., 1993; Bourke et al., 1992; see section 7.4). Problems expressing feelings to others could also feedback to difficulties detecting emotional displays in others. Alexithymic traits are also common concomitants of clinical depression (Loas et al. 1998; Parker et al., 1991), which might explain why depressives also require greater intensity of emotion to first perceive expressions.

However, decreased sensitivity to emotions could instead merely reflect general psychopathology, with all psychiatric patient groups tending to be less responsive to emotional cues. Diminished sensitivity could also be a consequence of

medication effects (blunting of response), although this is unlikely as anorexic, bulimic and clinically depressed patients were undergoing different pharmacological treatment (and indeed several patients were not taking any medication). Hospitalisation is not likely to have had an effect as only anorexic patients were in-patients, with both bulimics and depressives receiving treatment on an out-patient basis. Another explanation could be that reduced responsiveness is just a secondary consequence of depressive symptomatology, as depressives exhibit similar responses, although again it is notable that the effect is stronger for eating disorder patients than depressives, suggesting this is not the case.

Examination of overall sensitivity in other psychiatric patient groups (both in- and out-patients), for example, schizophrenics or social phobics, could help establish whether the cause of this insensitivity effect relates to general psychopathy, hospitalisation or treatment or whether instead it is due to more specific emotion processing difficulties in eating disorder and depressed individuals.

### 7.10.2 Sensitivity to specific emotions

Alterations in sensitivity to *specific* emotions were also revealed in the relative sensitivity analyses. As predicted in section 7.4.1, eating disorder individuals were relatively *more* sensitive to expressions of disgust than both controls (section 7.7.4) and the female clinically depressed group (section 7.8.3).

The possibility that sensitivity to disgust might be further enhanced in bulimics, as a consequence of their frequent vomiting and purging behaviour (an evolutionary response to repel noxious stimuli), did not appear to be the case as anorexic and bulimic subjects were *not* shown to differ in their pattern of sensitivity across the emotions (see section 7.9.4). Obviously larger sample sizes would be

necessary to conclusively establish no differences are present between these two forms of eating disorder. Enhanced sensitivity to disgust relative to controls might therefore be a consequence of the extreme maladaptive self-loathing both anorexics and bulimics feel. Such self-disparagement is a core concept of eating disorders with patients frequently experiencing disgust towards themselves, their behaviour, food etc. Such heightened processing of the emotion of disgust is liable to result in increased awareness of disgust-related stimuli, for example facial expressions of disgust in others.

Desire for social acceptance and positive evaluation in the eyes of others (Pliner & Haddock, 1996; see section 7.2.1) are also likely to make such individuals overly sensitive to criticism from others and therefore perception of expressions of contempt or disgust might become magnified. High levels of criticism directed at eating disorder sufferers by their mothers have been shown to predict a worse prognosis (van Furth et al., 1996).

As suggested by Ben-Tovim and Walker (1992), examination and identification of feelings or disgust and self-hatred should take priority in psychological treatment of eating disorders. Realisation that such intense self-depreciating thoughts are abnormal and destructive in addition to therapy restructuring attitudes towards one's body image should result in dampened feelings of self-disparagement, increased self-esteem and in turn should reduce sensitivity to disgust-related stimuli (including others' facial expressions of disgust) in eating disorder sufferers.

Using a different experimental paradigm, Sprengelmeyer and colleagues (1997b) found a selective *deficit* in recognition of facial expressions of disgust in obsessive-compulsive disorder patients (OCD). This contrasting finding is surprising

given the overlap between obsessive-compulsive disorder and eating disorders, particularly anorexia (Bastiani et al., 1996; Kasvikis et al 1986), e.g., abnormal preoccupation with food and weight. It is possible that this contrast may arise from the different methodologies used.

However, there is no apparent deficit in disgust recognition, or indeed in recognition of any emotion, by the eating disorder group here. Although 150%-caricatured images, rather than veridical 100% expression stimuli were used here, it seems unlikely that these patients would exhibit any difficulty in recognition of disgust at 100% as it was the not the most difficult emotion for them to identify (see Table 7.1) and less than 1 in 10 errors were made in identification of disgust. In addition, individuals with eating disorders have not been shown to have any impairment in disgust recognition with 100% expression images (Murphy, personal communication) using the emotional hexagon paradigm (Calder et al., 1996b). (This method involves morphing of images in steps between different emotions, for example, anger to disgust, with subjects required to identify which emotion is being presented using a 6-way forced-choice paradigm. Results, with regard to recognition accuracy, are compared with control data defining each morph as most like one emotion.) Instead the eating disorder patients tested here exhibited a selective *increase* in relative sensitivity to disgust. The extreme self-loathing characteristic of anorexia and bulimia, but not OCD, could explain this discrepancy in the findings. As a consequence of self-hatred, eating disorder sufferers might be more perceptive of disapproval or contempt expressed by others towards them in that it is concordant with their own self-evaluation. (Bastiani et al., 1996 also comments on the symptomatic differences between OCD and anorexia.) An obvious extension of the unexpected findings for OCD relative to these for individuals with eating disorders

would be to examine *sensitivity* to different expressions (using the current interactive test) in individuals with OCD.

Abnormalities in insular cortex function of anorexics have been noted in PET studies, both following food intake (Nozoe et al., 1993) and also in response to images of high calorie drinks (Ellison et al., in submission). This could perhaps relate to alterations in processing of disgust, as insular cortex is known to be associated with responding to offensive tastes and smells (Zald et al., 1998; Zatorre et al., 1992) in addition to more general feeding-related responses (Augustine, 1996). Indeed Phillips et al. (1998, 1997) discovered that selective activation of the right *anterior* insular cortex occurred in response to facial expressions of disgust (but not fearful or neutral expressions) in normal subjects. Disgusting stimuli were also shown to activate the left insular region in OCD and control subjects (Phillips et al., in press). The insular cortex therefore appears to be involved in processing different types of disgust-related stimuli: both those signalling disgust (such as facial expressions of disgust) and those actually eliciting a disgust reaction (e.g., bodily products). It is feasible then that the specific enhancement in relative sensitivity to disgust revealed in eating disorder individuals in this study could relate to discrete brain abnormalities in this region.

#### *7.10.2.1 Comparison with depressives*

Of particular interest in the current study is the finding that the opposite pattern of relative sensitivity (compared to controls) appears to be present for eating disorder and clinically depressed groups for perception of disgust. As stated in Chapter 5, the reason for relatively decreased sensitivity to disgust in depressed individuals is unclear. However the presence of such differences (in addition to

similarities) between eating disorder and depressed patients suggests that the relative sensitivity effects are *neither* due to general psychopathology *nor* are they a secondary consequence of depressive symptomatology in eating disorder individuals. Differential patterns of facial expression perception in clinically depressed, eating disorder and control groups also points to the sensitivity of the interactive test method used and further promotes its use in testing of other patient groups.

It is also notable that eating disorder patients exhibit the *same* pattern of decreased relative sensitivity to happy expressions as clinically depressed individuals. Presumably this is a result of both comorbid depressive state and the reduction in feelings of happiness and pleasure due to malnourishment and intense feelings of guilt. Reduced ability to detect positively valenced external cues will prevent individuals with eating disorders experiencing subtle gestures of happiness or pleasure expressed by others. Diminished experience of positive interactions and feedback is liable to have a negative effect on any individual's general well-being, especially on those already experiencing depressive affect and low self-esteem, and might serve to exacerbate the negative cognitions associated with the syndrome.

The increased relative sensitivity to sadness exhibited by clinical depressives is not apparent in the eating disorder group. Again the ensuing implication is that depressive symptomatology experienced by individuals with eating disorders is of a different nature to actual clinical depression and might often just be a subsidiary consequence of starvation (Pollice et al., 1997).

## 7.11 SUMMARY

Individuals suffering from eating disorders exhibit altered sensitivity to facial expressions compared to controls. In addition to a general insensitivity to all facial

emotion cues, individuals with eating disorders also show a selective *increase* in relative sensitivity to disgust (with the opposite pattern manifested by depressives) and a relative *decrease* in ability to detect happy expressions (analogous to depressives) compared to controls. In addition, eating disorder sufferers do not exhibit enhanced relative sensitivity to expressions of sadness demonstrated by female depressives.

It is also interesting that anorexics and bulimics do not appear to differ from each other in facial expression perception, in spite of the different manifestations of each condition. However, future studies would benefit from larger sample sizes to determine whether lack of differences seen here between anorexic and bulimic groups was instead just a function of inability to achieve significance due to the small subject number.

In concordance with the continuing research on facial expression perception in depressives, further investigation is planned with regard to the persistence of these alterations in sensitivity in eating disorders. Responses of both recovered anorexics and bulimics will be examined using the same interactive test in an attempt to determine whether differential processing of facial expressions is a state or trait effect in eating disorders. Such a study could also help elucidate whether or not any consequent social difficulty acts as a contributing factor in the development of anorexia or bulimia nervosa, as opposed to being an outcome of the condition.

# CHAPTER 8: CONCLUSIONS

## 8.1 GENERAL

*"A fundamental tenet of psychosomatic medicine is that interference with the experience and expression of emotions can have an adverse affect on health"* (Lane et al., 1996).

The studies in this thesis have revealed that, in addition to altered affective experience and interpersonal expression of emotion in psychiatric conditions, perception of facial expressions of emotion is also affected in individuals suffering from eating and depressive disorders. Although it remains unclear whether these perceptual difficulties are merely a consequence of altered emotional state or whether they play some role in the pathogenesis of such conditions, it does seem likely such alterations in sensitivity to non-verbal cues contribute to the interpersonal problems experienced by such individuals.

Indeed, a study of interpersonal behaviour in college roommates has indicated a relationship between good decoding ability of non-verbal signals and positive social interactions (Hodgins & Zuckerman, 1990). Interactions between two individuals who exhibited high levels of non-verbal sensitivity were rated more positively by the interactants than those between poor decoders of non-verbal cues. In particular the amount of emotional support (given and received) was greater in individuals with good non-verbal decoding ability. Although there are several types of non-verbal cues, probably the most important is the information conveyed by other individuals' facial expressions. If individuals with depression or eating disorders are therefore not so adept at decoding expressions of facial affect in others, as the current studies suggest, interactions will not be so successful. Instead of receiving beneficial aspects of contact with others (such as emotional sharing and

support), negative feeling might be generated (in both participants) and future contact avoided, ostracising the sufferer from social situations.

Coyne (1976a) stressed the importance of interpersonal factors in the maintenance of affective disorders, producing a repetitive cycle of problematic interactions, negative affect, decreased self-esteem and further social difficulties. Lack of social support is known to have a detrimental effect on psychological well-being (Berkman, 1995; Berkman & Syme, 1979; Cobb, 1976), indicating the significance of investigation into putative causal factors of interpersonal difficulties. The ultimate objective of such research of course would be the development of an intervention to help alleviate social problems in psychiatric conditions, which in turn might increase chance of recovery. The increasing prevalence of psychiatric problems in the community, particularly affective conditions such as depression and anxiety, emphasises the expediency of such inquiry.

## 8.2 SYNOPSIS OF FINDINGS

It would seem apparent from the results of the two studies of depression reported earlier (Chapters 4 and 5) that sensitivity to different facial expressions is altered in individuals experiencing some degree of depressive state. Dysfunction of internal regulation of emotions occurs in affective conditions so in many respects it seems logical that perception of emotional expression would also differ.

In addition, altered sensitivity to facial expressions of affect was revealed in individuals suffering from different forms of eating disorder (Chapter 7).

### 8.2.1 General insensitivity to emotional cues

In general, emotional responsivity appears to become blunted in more severe clinical cases of depression to other individuals' displays of facial affect, with increased intensity of *all expressions* required before depressed individuals first report perceiving the emotion in question. Decreased responsiveness to emotional cues has been noted by other researchers, for example, Baker et al. (1997) comment that *"... lack of emotional reactivity is pathognomic of depression"*.

Similar insensitivity to all expressions was exhibited by patients with eating disorders. It is possible then that diminished response to emotionally valenced stimuli is a general symptom of psychopathy, arising in different psychiatric populations, or alternatively it could be a reflection of some level of depressive symptomatology (as comorbid depressive state is present in the majority of eating disorder patients too, see section 7.7.1).

It is unlikely that the decreased responsiveness to expressions of emotion shown in depressives and eating disorder patients reflects a blunting effect of medication, in the respect that different pharmacological treatments were administered both within and between subject groups and indeed some patients were not receiving any medication. It was also unlikely that the non-clinical 'mildly depressed' group were taking any pharmacological treatment, yet they appeared to exhibit a trend in the same direction as the clinically depressed group, i.e., were less sensitive overall to all the expressions.

Examination of sensitivity to facial expression cues in other psychiatric patient groups, for example, schizophrenics or social phobics, could help establish the underlying cause of such insensitivity, i.e., the relative effect of psychopathy, comorbid depressive state or treatment. The fact that individuals with subclinical

levels of anxiety were shown to exhibit *increased* sensitivity to all expressions (see Chapter 4) would suggest however that diminished responsiveness relates to a more specific emotion processing difficulty in eating disorder and depressed individuals.

Difficulties in both comprehension and awareness of one's own emotional experience are commonly associated with eating disorders and depressive state (Sexton et al., 1998; Smith et al., 1997; Troop et al., 1995; Cochrane et al., 1993; Schmidt et al., 1993; Bourke et al., 1992; Loas et al., 1998; Parker et al., 1991). These alexithymic traits (for example, inability to distinguish between a physical sensation, such as increased heart rate – perhaps due to exercise - and feelings of fear) are also likely to influence perception of emotions. Several studies have investigated emotion recognition ability in alexithymic individuals with varying results. Although McDonald and Prkachin (1990) did not observe any differences, both Parker et al. (1993) and Lane et al. (1996) reported that alexithymic individuals (both students and individuals with a psychiatric history of alexithymia respectively) were significantly worse at identifying facial expressions of emotion than non-alexithymic subjects. In addition, Lane et al. (1996) demonstrated that this impairment was not purely a consequence of difficulty assigning verbal labels to emotions, as individuals with high scores for alexithymia also experienced difficulty on a non-verbal task, which involved matching facial expressions with scenes depicting the corresponding emotion. The authors concluded instead that alexithymic individuals had a more general problem in processing of emotional stimuli.

It is notable however that neither depressives nor individuals with eating disorders in the current studies exhibited any problem in the assignment of emotion labels to facial expressions, at least with high intensity 150%-caricatured expression stimuli. Instead, difficulties detecting emotions at low intensity were observed.

Similar findings were reported in alexithymic individuals by Berenbaum and Prince (1994), although the task involved interpretation of emotional content in stories, rather than recognition of facial expressions. Identification of emotional content was unimpaired in alexithymic subjects, yet they were shown to underestimate the intensity of the emotion described, suggesting that they require greater stimulation to become aware of all emotional cues. Alexithymic individuals were also shown to rate affectively valenced pictures as appearing less intense than controls in a study by Vanman et al. (1998). Although levels of alexithymic traits were not actually assessed in either the clinically depressed or eating disorder population studied here, it seems possible that such difficulties in emotional understanding could result in the general insensitivity to others' facial expressions apparent in these groups. Evaluation of the presence (or absence) of alexithymic traits would be a valuable addition in future studies.

It is of course possible that the insensitivity seen here relates instead to a general conservative response bias in these patient groups. Clinically depressed patients have been shown to exhibit lack of confidence in their own judgement, resulting in abnormally conservative responding, in a recognition memory task (Corwin et al., 1990). As there was no additional control task in this thesis, it is impossible to say whether or not such insensitivity was merely the result of increased caution in response, rather than a consequence of selective hyposensitivity to emotional expressions. Future studies would benefit from the use of a comparative control test, for example, morphing between a male and female face and assessing when the face was first perceived as female (or vice versa). If patients were shown to exhibit a similarly overcautious pattern compared to controls, e.g., requiring a higher intensity to classify the face as appearing female, then it would seem likely that the

general hyposensitivity revealed for perception of facial expressions is a consequence of a more general conservative bias in response.

Regardless of the cause of diminished sensitivity to emotions, further research into this area is a priority as such insensitivity is likely to contribute to the maintenance of any interpersonal problems in these individuals. Good communication and understanding between all parties is necessary for an interaction to proceed smoothly. If an individual is unable to detect more subtle nuances in an interaction, due to insensitivity to emotional cues, then he/she is unlikely to respond to the other individual, which could result in a breakdown in communication.

### 8.2.2 Alterations in sensitivity to specific emotions

#### *8.2.2.1 Anger*

When examining differences in pattern of sensitivity change across the six expressions, i.e., relative sensitivity, individuals experiencing depressed affect were found to be relatively *more* sensitive to expressions of anger than control subjects. Heightened sensitivity to anger could reflect a depressive's increased concern over whether others are responding in a hostile manner towards him/her and could relate to a desire to avoid conflict. In addition, enhanced perception of anger could reflect a depressed individual's fears that his/her behaviour might inadvertently be annoying others or causing them to disapprove in some way, due to the characteristic low self-esteem experienced in depressive conditions. Of course, if a depressive believes a friend or stranger is irritated with him/her (due to increased sensitivity to angry expressions), when this is not the case, then feelings of self-dislike, low confidence and failure will be enhanced and could cause the depressive to evade future interactions with this person.

In addition, depressives frequently feel extremely resentful towards others (Wolfersdorf & Kiefer, 1998) and can become quite aggressive in their behaviour (Bjork et al., 1997; Yesavage, 1983; Hokanson et al., 1980). Presumably intensified emotional experience of anger in depressives could relate to increased sensitivity to this emotion in other individuals. Indeed, Berenbaum (1992) observed enhanced *expression* of anger in depressed patients (in addition to reduced expression of happiness) and commented that such a pattern of emotional response in depressives was merely likely to *"...reflect their underlying emotional state"*.

Such enhanced sensitivity to expressions of anger could perhaps be moderated by administration of the anxiolytic, benzodiazepam. Blair et al. (in press) have revealed that impaired recognition of facial expressions of anger results from the pharmacological effect of this drug in normal subjects, so presumably a similar outcome might arise if depressives were treated with benzodiazepam. If reduced perception of anger results from administration of benzodiazepam, which is prescribed to decrease anxious symptomatology, one could perhaps conversely predict that enhanced sensitivity to anger might also be present in non-medicated clinically anxious patients, which could be investigated in future studies.

Interestingly, in terms of the putative relationship between depression and alexithymia (see section 8.2.1), an association between alexithymia and increased sensitivity to anger has also been reported. In the study mentioned earlier by Berenbaum and Prince (1994), alexithymic individuals tended to favour interpretations of emotion portrayed in fictional situations that related to anger and dominance. Another study by Berenbaum and Irwin (1996) revealed that individuals with high levels of alexithymia were more likely to express anger facially in response to an anger-provoking situation than subjects low in alexithymic traits,

suggesting they were experiencing anger[1]. In spite of this, subjects in the high alexithymia group did not *subjectively* report experiencing anger or displeasure during the interaction, although their responses afterwards indicated increased interpersonal avoidance of future contact. Lack of subjective report of feeling angry could suggest alexithymic individuals wanted to avoid confrontation, even though they were aware of feeling hostile towards the other individual. Alternatively, it seems more probable that they were merely unable to interpret their own internal state or have insight into their experience of anger.

Individuals suffering from eating disorders, who (like depressives) are believed to possess alexithymic characteristics (Smith et al., 1997; Troop et al., 1995; Schmidt et al., 1993; Bourke et al., 1992), did *not* exhibit enhanced sensitivity to angry expressions relative to controls. Such a selective alteration in sensitivity to anger in clinically depressed patients might therefore indicate that a mechanism other than alexithymia is accounting for this effect.

#### *8.2.2.2 Disgust*

Relative sensitivity to disgust was differentially affected in clinically depressed and eating disorder patients. Empirical findings substantiated the hypothesis that individuals with eating disorders would be relatively more sensitive to expressions of disgust than control subjects were. Extreme feelings of self-loathing and disgust of both body image and illness-related behaviour are likely to account for the relatively enhanced detection of disgusted expressions by eating disorder sufferers, as general experience and processing of disgust is heightened in these individuals.

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[1] Participants were unaware they were being video-taped during the interaction.

Both clinically and subclinically depressed individuals were conversely shown to be relatively *less* sensitive to disgusted expressions than controls, although the reason for this is not explicit. More severe impairments in recognition of expressions of disgust have been observed in both Huntington's Disease and obsessive-compulsive disorder sufferers (see sections 1.1.5 and 4.4.1). Fronto-striatal abnormalities are common to all three conditions and could possibility account for difficulties in perception of disgust.

Alternatively, impaired perception of disgust could perhaps be accounted for by learning theory, with understanding of others' facial expressions of disgust emanating from coincident experience of disgust in oneself (see Sprengelmeyer et al., 1997b and Rozin et al., 1993). The validity of a learning theory explanation is perhaps evident for individuals suffering from Huntington's Disease and OCD but is less obvious for depressives. In addition, it cannot account for the differences found between disgust perception in OCD sufferers and individuals with eating disorders, as the premise of loss of correlation between experience of disgust in oneself and expression of disgust in others should apply equivalently to both conditions. Until a group of patients with OCD are tested using the current interactive test and eating disorder sufferers are assessed with the methods used by Sprengelmeyer et al. (i.e., recognition of static 100% expression images and morphed images in the emotion hexagon), it cannot be determined whether this contrast is contingent on the different technologies used.

#### *8.2.2.3 Happiness*

Patients with both depression and eating disorders were *less* able to detect mild expressions of happiness than control subjects were, which might reflect

concomitant feelings of anhedonia. Loss of pleasurable affect is a crucial diagnostic symptom in depression and can be a consequence of malnourishment and feelings of guilt in eating disorder sufferers. Reduced experience of happiness is likely to result in diminished sensitivity to other people expressing happiness. Again, one could posit a learning theory explanation, with expression of happiness in others not equating to experience of happiness in the perceiver, resulting in disconnection between experiential, expressive and perceptive aspects of happiness.

Decreased perception of happiness in others and therefore diminished positive feedback is liable to have a negative impact on an individual's general well-being, which could maintain, if not exacerbate, negative psychological symptoms associated with these conditions. Interestingly, individuals with high levels of alexithymia also appear to have a reduced ability to experience pleasure in social situations, even though they are able to experience physical pleasure normally (Prince & Berenbaum, 1993). It is possible then that these alexithymic traits are contributing to insensitivity to happy expressions in individuals with depression or eating disorders. Future studies in this area with depressed or eating disordered populations should additionally evaluate levels of alexithymia in these individuals. Investigation of facial expression perception in a non-psychiatric group of alexithymic individuals also seems crucial, in order to examine the relative influence of this condition on sensitivity in patients with eating disorders or depression.

The fact that differences, in addition to similarities, have been found between individuals with eating disorders and depression indicate that these results cannot be incidentally explained by either general psychopathology, or secondary depressive symptomatology in eating disorder sufferers. In addition, the sensitivity of the

interactive method is manifest from such divergent findings across subject groups and supports the use of this test in other patient groups.

### 8.2.3 Recognition deficits in depressed patients undergoing psychosurgery

Examination of change in facial expression perception in patients undergoing psychosurgery for treatment of refractory depression revealed an substantive impairment in recognition of 150%-caricatured expressions of fear. Although recognition was not reduced to chance, the fact that the images used were 150%-caricatured expressions (which should have facilitated identification, see Calder et al., 1997), rather than veridical 100% images, indicates a fairly severe deficit in perception of fear. Future research with these patients should include standard test batteries for facial expression perception, i.e., recognition of fear in static 100% expression images.

Impaired recognition of fear has been reported in individuals with amygdala damage (see section 1.1.5). As AC surgery itself involves disconnection of the frontal lobe from thalamic regions, it is possible that connections to the amygdala have been affected and damage has occurred. The acquisition of MRI scans one-year post-surgery, once gliosis has occurred and scar tissue has appeared, will help elucidate both the exact location and the extent of the lesion and consequently whether damage to the amygdala or its projections has actually arisen.

If reduction in depressed affect occurs after surgical treatment, one might assume that sensitivity changes in closer accord with findings for non-depressed individuals might arise. Sensitivity to different expressions did not appear to change, however, following the surgical procedure. Lack of alteration in sensitivity might be

accounted for by clinical indications of reduced anxiety and obsessive symptoms post-surgery, rather than a major decrease in negative affect.

The need for extension and replication of this study is considerable due to the small subject number. In addition, inclusion of a non-psychiatric control group undergoing another type of intracranial surgery, e.g., patients undergoing surgery for removal of tumours, would enable determination whether any effects seen are due to craniotomy, neuro-anaesthesia or the stress inherent in the surgical procedure.

## 8.3 METHODOLOGICAL ASPECTS

### 8.3.1 Benefits

The usefulness of the current methodology is evident in the respect that assessment of more than one aspect of perception in different subject groups is necessary to obtain a better understanding of the nature of any deficit. It is clear from the studies presented in this thesis that impairment in recognition of emotion can occur, as with surgical patients, in the absence of any apparent changes in sensitivity. (N.B. Sensitivity was only assessed for emotions that were recognised correctly.) Conversely, alterations in the intensity required to first perceive an emotion can be present even though the ability to assign emotion labels to expressions remains intact (as seen in patients with eating disorders and both subclinically and clinically depressed individuals). Evaluation solely of recognition ability (in terms of accuracy) in certain patient groups might overlook potential differences in interpretation of expressions. Additional examination of sensitivity seems crucial as any alterations could have an important impact on interpersonal behaviour. Assessment of both recognition accuracy and sensitivity to different emotions should

therefore be viewed as complementary testing methods. It is recommended that further studies on other patient groups employ such a test.

Previously, most tests have just measured one aspect of perception – usually recognition, using static images of 100% (or 150%) emotional intensity. The dynamic and interactive nature of the current method is more 'user-friendly' and appears to enhance motivation for test completion. The graded change in intensity of emotion has also been shown to be sufficiently sensitive to detect subtle effects in a functional subclinical population experiencing mild forms of depressed mood state.

The possible diagnostic value of such a test should perhaps also be considered. In addition to being straightforward to use, the fact that it is sensitive to slight changes in perception (for example, alterations in sensitivity in non-clinical depressed groups here) suggests that it could perhaps be used to assist detection of a psychiatric condition at an early stage.

### 8.3.2 Possible refinements

The test method could be improved by inclusion of a greater number of different identities expressing the emotions, as only 3 different exemplars (from the Ekman & Friesen (1976) series of 10) were used for each expression in the current test. In addition, to ensure against possible stimulus effects, it would be useful to replicate these studies using a completely different set of faces, rather than being limited to the extensively used set of Ekman faces. Such a criticism has been addressed by the development of another test, using new images of posed facial expressions. Individuals were photographed while posing different expressions. The resulting stimuli were rated by another group of subjects (n=36) with regard to how well each conveyed the appropriate emotion. Stimuli shown to be well-recognisable

were delineated (see Figure 4.2, section 4.2.2) and transformed into morphed images for the interactive test format. This new test is currently undergoing piloting.

Another useful extension of the current test method would be inclusion of gaze direction as an additional variable. Ability to comprehend direction of attention is another crucial aspect of social communication, enabling inference to be made regarding another individual's intentions. Although the ability to decode and interpret facial expressions irrespective of other transient information (such as changes in lighting and orientation) is important, it is obviously of additional use to be able to establish to whom an expression is directed. Perrett and Emery (1994) stress the importance of decoding direction of attention in making attributions about the motives and intentions of others. In this respect, a method that enables analysis of sensitivity to different facial expressions in combination with changes in attention direction would be very useful. One would predict that sensitivity might be more enhanced for detection of expressions directed at the perceiver than those directed elsewhere. Interactions with specific emotions might be present in certain patient groups, for example, anxious individuals might be differentially more sensitive to expressions of anger directed *at* them, rather than away from them.

Such a test is currently being developed using the new expression images mentioned above. Individuals were photographed posing expressions whilst both facing and directly looking at the camera and also in the same head position (i.e., front) but with eyes directed to an image 45 degrees to their left. (Examples of morphed expressions with eye gaze both at the perceiver and averted are presented in Figure 8.1.) Once this test has been fully piloted, it would be beneficial to use it both on the patient groups already examined and also in individuals suffering from anxiety, who might be particularly sensitive to threat cues directed at them.

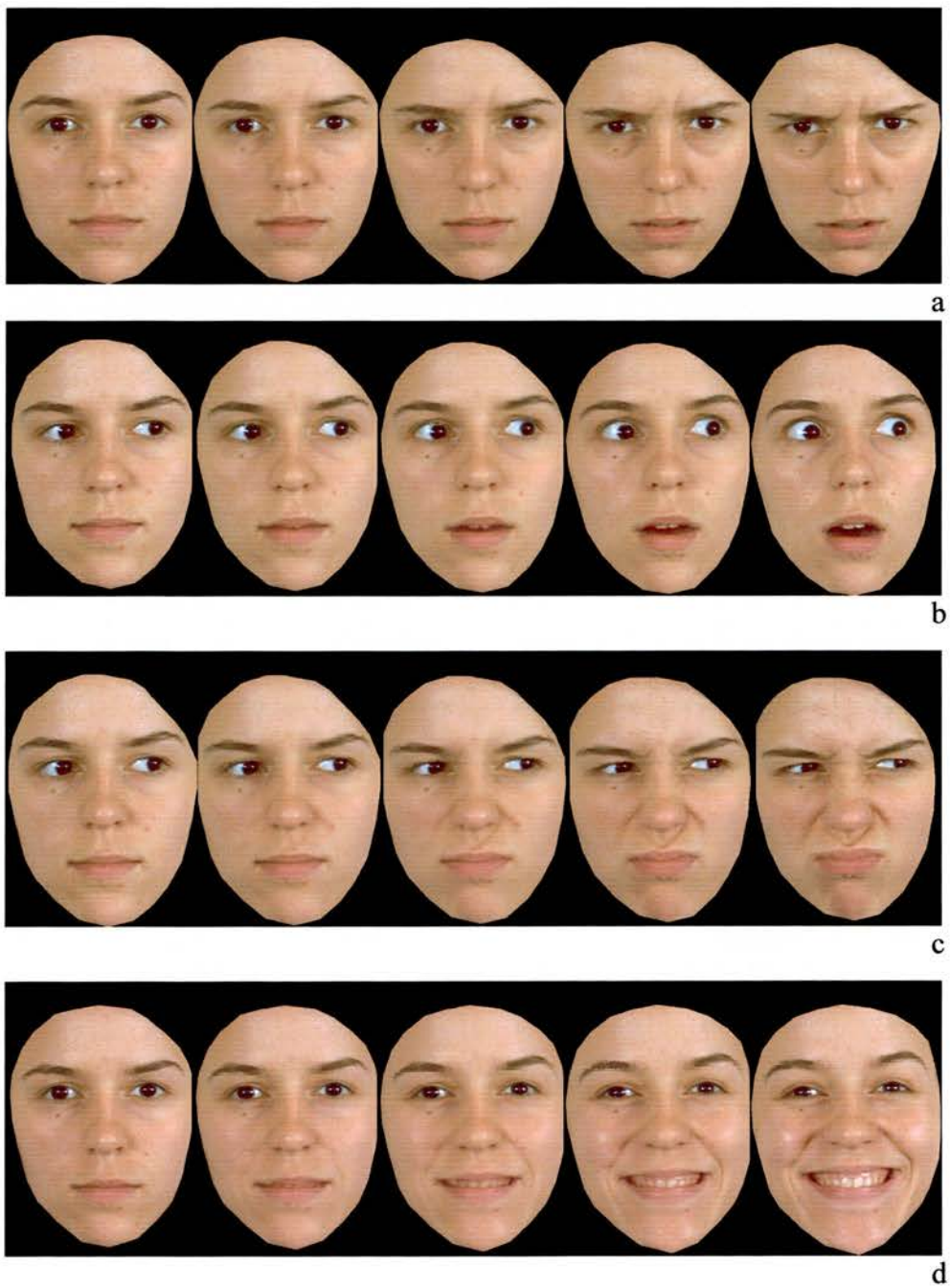


**Figure 8.1: An example of the new expression images with gaze direction as an additional variable. The expressions represented are *anger* (image a), *fear* (image b), *disgust* (image c), and *happiness* (image d), with gaze direction *front* in images a and d and *away* in images b and c. The change in morph intensity is in 25% steps from 0%-100%.**

## 8.4 DIRECTIONS FOR FUTURE RESEARCH

### 8.4.1 State or trait effect?

An important continuation of the studies conducted in this thesis is to examine whether differences in sensitivity to facial expressions seen in the psychiatric groups tested here normalise with clinical recovery. Verification of whether alterations in sensitivity are state-dependent or not, e.g., whether the effect of general hyposensitivity to facial expressions remains in a remitted state or whether sensitivity similar to non-depressed individuals is instead apparent, should enable determination of the relative influence of such factors in the pathogenesis of these conditions.

Such a study is currently being conducted on clinically depressed patients in Ninewells Hospital and Medical School. Both a within- and between-subjects design is being implemented, with as many clinically depressed patients as possible from the initial study (Chapter 5), in addition to a new group of recovered depressives, being (re)tested on the same interactive test and clinically assessed for recovery. Resolution of this issue has important implications for understanding predisposing factors for depression and progression of depressive symptomatology. If altered sensitivity to facial expressions is revealed to be trait-like in depressives, i.e., stable pre, during and post depressive episode, then one might assume that this pattern of sensitivity seen in depressives might be contributing to their interpersonal difficulties and might act as a predisposing factor for depression. Conversely, if sensitivity changes are demonstrated to be dependent on experience of depressive state and shown to normalise with recovery, one can assume that alterations in sensitivity to different facial expressions seen here in depressives are merely a manifestation of depressed state and do not play a causal role in the development of depression.

Application of prospective, longitudinal study designs would be particularly useful in assessment of the possible contributory role of misinterpretation of facial expressions to depression onset.

A study by George et al. (1998) suggests that changes in facial expression processing might be state-dependent in depression, as opposed to a persistent trait. The authors tested a single patient with ultra-rapid-cycling bipolar affective disorder on a number of occasions during which he experienced cyclical changes in his condition. He was tested on a total of 11 occasions: 5 when depressed, 5 when euthymic and once when hypomanic (i.e., he was not experiencing depressed affect on 6 occasions). Facial expression perception was examined by asking the patient to rate faces along one dimension as to whether they looked happy or sad (using a 7-point Likert rating scale from *'very happy'* to *'very sad'*). A control test of age estimation (from *'teens'* to *'70s'*) was also conducted.

Performance on the facial emotion task differed significantly according to whether the patient was in a depressed or non-depressed phase. When depressed, the patient tended to rate sad faces as appearing extremely sad and neutral faces as appearing sad, whereas this pattern did not arise when he was not feeling depressed (i.e., sad faces were rated as sad – not as *very sad* – and neutral faces as appearing neutral). (It should perhaps be noted that there was no apparent change in the rating of intensity of happy faces across phases for this patient.) Ability on the age discrimination task did not differ, however, according to depressive state, i.e., there was no response shift in perception of age. These results suggest that alterations seen in facial expression perception in depressed patients are dependent on state and time-locked to the depressive episode, rather than being long-term enduring deficits.

This study by George et al. (1998) is important in that most other studies have only attempted to examine whether facial expression processing deficits are *present* in depression and have not investigated whether any effects still occur outwith the depressive episode (e.g., Gur et al., 1992; Rubinow & Post, 1992; Mandal & Bhattachayra, 1985). A replication of such a study using a larger number of bipolar patients cycling between depressed and euthymic phases is really required before any conclusive statement can be made as to the nature of the facial affect deficit, as these results might be idiosyncratic (although unlikely) to the patient in question. Alternatively, as proposed above, unipolar patients could be tested using a longitudinal within-subjects design (i.e., both while depressed and during recovery).

### 8.4.2 Different patient groups

In the light of findings of distinct deficits in facial expression perception in the subject groups investigated in this thesis, examination of potential deficits in other psychiatric patient groups seems imperative.

Alexithymia seems a relevant candidate for assessment, due to difficulties in emotional awareness and inability to distinguish between emotions. Alexithymic characteristics are commonly found in individuals with eating disorders and depression. As the presence of alexithymic traits was not assessed in either patient group here, it seems important to examine ability to process facial expressions in non-psychiatric alexithymic individuals to establish whether these traits could have affected perception in depressives or eating disorder sufferers.

Another prevalent affective condition worth investigation is anxiety. Students with high anxiety scores were shown to be more sensitive to all facial expressions than low scoring students in a study in this thesis, although highly anxious subjects

did not appear to be differentially sensitive to any particular emotion(s). Replication of this study using a clinical population is necessary. In addition, differentiation between different types of anxiety is perhaps important. Investigation of facial expression perception would probably be most pertinent in individuals with social phobia. As the name suggests, these individuals are extremely anxious in social situations and are afraid of criticism from others. Heightened sensitivity to expressions of anger or disgust could therefore result, in the fear that another individual is either annoyed with them or expressing disapproval. Perpetuation and aggravation of anxiety symptoms is likely to result from such enhanced sensitivity and again could result in the sufferer unnecessarily withdrawing from an interaction.

A supplementary benefit of such research would be the resolution of the contention that deficits observed in psychiatric groups here were merely a consequence of general psychopathy, when in fact it seems more probable that they relate to more specific emotion processing mechanisms associated with each condition.

## 8.5 FINAL NOTE

The studies in this thesis indicate that affective state influences sensitivity to different facial expressions. As such changes are likely to affect behaviour towards others, it seems crucial to continue investigation in this area, particularly in psychiatric patient groups who are known to experience social difficulties. The importance of interpersonal factors in maintenance of some psychiatric conditions should not be underestimated. Although the relative impact of such alterations in sensitivity to expressions on illness course has not yet been established, it does seem

clear that interpersonal difficulties will be aggravated in certain patient groups due to inaccurate interpretation of other individuals' expressions.

Therapeutic interventions in the future might gain from increased focus on interpersonal aspects of a condition. In particular, training individuals to reappraise facial expression cues might enhance prospects of recovery and help guard against relapse.

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# APPENDIX 4.1: THE BECK DEPRESSION INVENTORY (BDI)

BDI®

Date: _____

Name: _____ Marital Status: _____ Age: _____ Sex: _____

Occupation: _____ Education: _____

This questionnaire consists of 21 groups of statements. After reading each group of statements carefully, circle the number (0, 1, 2 or 3) next to the one statement in each group which **best** describes the way you have been feeling the **past week, including today.** If several statements within a group seem to apply equally well, circle each one. **Be sure to read all the statements in each group before making your choice.**

**1**
0 I do not feel sad.
1 I feel sad.
2 I am sad all the time and I can't snap out of it.
3 I am so sad or unhappy that I can't stand it.

**2**
0 I am not particularly discouraged about the future.
1 I feel discouraged about the future.
2 I feel I have nothing to look forward to.
3 I feel that the future is hopeless and that things cannot improve.

**3**
0 I do not feel like a failure.
1 I feel I have failed more than the average person.
2 As I look back on my life, all I can see is a lot of failures.
3 I feel I am a complete failure as a person.

**4**
0 I get as much satisfaction out of things as I used to.
1 I don't enjoy things the way I used to.
2 I don't get real satisfaction out of anything anymore.
3 I am dissatisfied or bored with everything.

**5**
0 I don't feel particularly guilty.
1 I feel guilty a good part of the time.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

**6**
0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

**7**
0 I don't feel disappointed in myself.
1 I am disappointed in myself.
2 I am disgusted with myself.
3 I hate myself.

**8**
0 I don't feel I am any worse than anybody else.
1 I am critical of myself for my weaknesses or mistakes.
2 I blame myself all the time for my faults.
3 I blame myself for everything bad that happens.

**9**
0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

**10**
0 I don't cry any more than usual.
1 I cry more now than I used to.
2 I cry all the time now.
3 I used to be able to cry, but now I can't cry even though I want to.

**11**
0 I am no more irritated now than I ever am.
1 I get annoyed or irritated more easily than I used to.
2 I feel irritated all the time now.
3 I don't get irritated at all by the things that used to irritate me.

**12**
0 I have not lost interest in other people.
1 I am less interested in other people than I used to be.
2 I have lost most of my interest in other people.
3 I have lost all of my interest in other people.

**13**
0 I make decisions about as well as I ever could.
1 I put off making decisions more than I used to.
2 I have greater difficulty in making decisions than before.
3 I can't make decisions at all anymore.

Subtotal Page 1

CONTINUED ON BACK

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| | | | | | |
|---|---|---|---|---|---|
| 14 | 0 | I don't feel I look any worse than I used to. | 19 | 0 | I haven't lost much weight, if any, lately. |
| | 1 | I am worried that I am looking old or unattractive. | | 1 | I have lost more than 5 pounds. |
| | 2 | I feel that there are permanent changes in my appearance that make me look unattractive. | | 2 | I have lost more than 10 pounds. |
| | 3 | I believe that I look ugly. | | 3 | I have lost more than 15 pounds. |
| | | | | | I am purposely trying to lose weight by eating less. Yes _____ No _____ |
| 15 | 0 | I can work about as well as before. | 20 | 0 | I am no more worried about my health than usual. |
| | 1 | It takes an extra effort to get started at doing something. | | 1 | I am worried about physical problems such as aches and pains; or upset stomach; or constipation. |
| | 2 | I have to push myself very hard to do anything. | | 2 | I am very worried about physical problems and it's hard to think of much else. |
| | 3 | I can't do any work at all. | | 3 | I am so worried about my physical problems that I cannot think about anything else. |
| 16 | 0 | I can sleep as well as usual. | 21 | 0 | I have not noticed any recent change in my interest in sex. |
| | 1 | I don't sleep as well as I used to. | | 1 | I am less interested in sex than I used to be. |
| | 2 | I wake up 1-2 hours earlier than usual and find it hard to get back to sleep. | | 2 | I am much less interested in sex now. |
| | 3 | I wake up several hours earlier than I used to and cannot get back to sleep. | | 3 | I have lost interest in sex completely. |
| 17 | 0 | I don't get more tired than usual. | | | |
| | 1 | I get tired more easily than I used to. | | | |
| | 2 | I get tired from doing almost anything. | | | |
| | 3 | I am too tired to do anything. | | | |
| 18 | 0 | My appetite is no worse than usual. | | | |
| | 1 | My appetite is not as good as it used to be. | | | |
| | 2 | My appetite is much worse now. | | | |
| | 3 | I have no appetite at all anymore. | | | |

_____ Subtotal Page 2

_____ Subtotal Page 1

_____ Total Score

TPC 0528-003 28 29 30 B C D E

The BDI is scored by summing up the question numbers (0-3) circled as responses for each item. If more than one statement has been circled for any one item then the highest number is taken. Scoring can therefore range between 0 and 64.

The personal information section at the top of each BDI questionnaire was concealed in the studies conducted and not recorded.

## APPENDIX 4.2: THE STATE-TRAIT ANXIETY INVENTORY (STAI)

# SELF-EVALUATION QUESTIONNAIRE

Developed by Charles D. Spielberger
in collaboration with
R.L. Gorsuch, R. Lushene, P.R. Vagg, and G.A. Jacobs

**STAI Form Y-1**

Name: _____ Date: _____ S ____

Age: _____ Sex: M _____ F _____ T ____

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel *right* now, that is, *at this moment.* There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

| | | NOT AT ALL | SOMEWHAT | MODERATELY SO | VERY MUCH SO |
|---|---|---|---|---|---|
| 1. | I feel calm | ① | ② | ③ | ④ |
| 2. | I feel secure | ① | ② | ③ | ④ |
| 3. | I am tense | ① | ② | ③ | ④ |
| 4. | I feel strained | ① | ② | ③ | ④ |
| 5. | I feel at ease | ① | ② | ③ | ④ |
| 6. | I feel upset | ① | ② | ③ | ④ |
| 7. | I am presently worrying over possible misfortunes | ① | ② | ③ | ④ |
| 8. | I feel satisfied | ① | ② | ③ | ④ |
| 9. | I feel frightened | ① | ② | ③ | ④ |
| 10. | I feel comfortable | ① | ② | ③ | ④ |
| 11. | I feel self-confident | ① | ② | ③ | ④ |
| 12. | I feel nervous | ① | ② | ③ | ④ |
| 13. | I am jittery | ① | ② | ③ | ④ |
| 14. | I feel indecisive | ① | ② | ③ | ④ |
| 15. | I am relaxed | ① | ② | ③ | ④ |
| 16. | I feel content | ① | ② | ③ | ④ |
| 17. | I am worried | ① | ② | ③ | ④ |
| 18. | I feel confused | ① | ② | ③ | ④ |
| 19. | I feel steady | ① | ② | ③ | ④ |
| 20. | I feel pleasant | ① | ② | ③ | ④ |

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Copy of the STAI-state scale to assess transient experience of anxious symptomatology.

# SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-2

Name: _____ Date: _____

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

| | | ALMOST NEVER | SOMETIMES | OFTEN | ALMOST ALWAYS |
|---|---|---|---|---|---|
| 21. | I feel pleasant | ① | ② | ③ | ④ |
| 22. | I feel nervous and restless | ① | ② | ③ | ④ |
| 23. | I feel satisfied with myself | ① | ② | ③ | ④ |
| 24. | I wish I could be as happy as others seem to be | ① | ② | ③ | ④ |
| 25. | I feel like a failure | ① | ② | ③ | ④ |
| 26. | I feel rested | ① | ② | ③ | ④ |
| 27. | I am "calm, cool, and collected" | ① | ② | ③ | ④ |
| 28. | I feel that difficulties are piling up so that I cannot overcome them | ① | ② | ③ | ④ |
| 29. | I worry too much over something that really doesn't matter | ① | ② | ③ | ④ |
| 30. | I am happy | ① | ② | ③ | ④ |
| 31. | I have disturbing thoughts | ① | ② | ③ | ④ |
| 32. | I lack self-confidence | ① | ② | ③ | ④ |
| 33. | I feel secure | ① | ② | ③ | ④ |
| 34. | I make decisions easily | ① | ② | ③ | ④ |
| 35. | I feel inadequate | ① | ② | ③ | ④ |
| 36. | I am content | ① | ② | ③ | ④ |
| 37. | Some unimportant thought runs through my mind and bothers me | ① | ② | ③ | ④ |
| 38. | I take disappointments so keenly that I can't put them out of my mind | ① | ② | ③ | ④ |
| 39. | I am a steady person | ① | ② | ③ | ④ |
| 40. | I get in a state of tension or turmoil as I think over my recent concerns and interests | ① | ② | ③ | ④ |



Copy of the STAI-trait scale to measure general proneness for anxiety.

The personal information section at the top of each scale was concealed and not recorded.

## APPENDIX 4.3

### Results of BDI analysis with STAI as a covariate

Recognition accuracy:

main effect of group, $F_{(1,70)}=0.7$, p=0.4 (n.s.)

main effect of expression, $F_{(3.6,252.5)}=6.7$, p=0.001 (sig.)

interaction of expression and group, $F_{(3.6,252.5)}=0.5$, p=0.7 (n.s.)

Absolute sensitivity:

main effect of group, $F_{(1,70)}=0.1$, p=0.8 (n.s.)

main effect of expression, $F_{(3.6,254.3)}=13.7$, p=0.001 (sig.)

interaction of expression and group, $F_{(3.6,254.3)}=1.8$, p=0.1 (n.s.)

Relative sensitivity:

main effect of group, $F_{(1,70)}=0.0$, p=0.9 (n.s.)

main effect of expression, $F_{(3.6,252.3)}=12.5$, p=0.001 (sig.)

interaction of expression and group, $F_{(3.6,252.3)}=2.4$, p=0.05 (sig.)

**APPENDIX 4.4**

**Results of STAI analysis with BDI as a covariate**

Recognition accuracy:

main effect of group, $F_{(1,70)}=0.6$, p=0.5 (n.s.)

main effect of expression, $F_{(3.6,252.5)}=5.9$, p=0.001 (sig.)

interaction of expression and group, $F_{(3.6,252.5)}=3.3$, p=0.014 (sig.)

Absolute sensitivity:

main effect of group, $F_{(1,70)}=2.7$, p=0.1 (n.s.)

main effect of expression, $F_{(3.6,254.3)}=8.1$, p=0.001 (sig.)

interaction of expression and group, $F_{(3.6,254.3)}=1.0$, p=0.4 (n.s.)

Relative sensitivity:

main effect of group, $F_{(1,70)}=0.1$, p=0.8 (n.s.)

main effect of expression, $F_{(3.6,252.3)}=8.4$, p=0.001 (sig.)

interaction of expression and group, $F_{(3.6,252.3)}=0.8$, p=0.5 (n.s.)

## APPENDIX 5.1: DIAGNOSTIC AND GENERAL PATIENT INFORMATION

| Subject No. | Sex | Age | Date tested | BDI score | ICD-10 | Diagnosis |
|---|---|---|---|---|---|---|
| 1 | FEMALE | 23 | 23.06.98 | 22 | F32.00 | Mild depressive episode without somatic syndrome |
| 2 | FEMALE | 49 | 24.06.98 | 27 | F32.00 | Mild depressive episode without somatic syndrome |
| 3 | MALE | 38 | 30.06.98 | 22 | F32.11 | Moderate depressive episode with somatic syndrome |
| 4 | MALE | 55 | 07.07.98 | 28 | F32.11 | Moderate depressive episode with somatic syndrome |
| 5 | FEMALE | 34 | 08.07.98 | 35 | F32.10 | Moderate depressive episode without somatic syndrome |
| 6 | MALE | 45 | 24.07.98 | 38 | F32.10 | Moderate depressive episode without somatic syndrome |
| 7 | MALE | 59 | 29.07.98 | 29 | F32.20 | Severe depressive episode without psychosis |
| 8 | FEMALE | 25 | 04.08.98 | 22 | F32.11 | Moderate depressive episode with somatic syndrome |
| 9 | FEMALE | 35 | 04.08.98 | 26 | F32.11 | Moderate depressive episode with somatic syndrome |
| 10 | MALE | 18 | 07.08.98 | 27 | F32.10 | Moderate depressive episode without somatic syndrome |

| Subject No. | Sex | Age | Date tested | BDI score | ICD-10 | Diagnosis |
|---|---|---|---|---|---|---|
| 11 | FEMALE | 43 | 07.08.98 | 22 | F32.10 | Moderate depressive episode without somatic syndrome |
| 12 | MALE | 45 | 11.08.98 | 22 | F43.20 | Adjustment reaction, depressive |
| 13 | FEMALE | 56 | 28.08.98 | 46 | F33.10 | Recurrent depressive episode (moderate severity) w/out somatic syndrome |
| 14 | MALE | 32 | 01.09.98 | 21 | F32.01 | Mild depressive episode with somatic syndrome |
| 15 | FEMALE | 40 | 28.09.98 | 36 | F32.11 | Moderate depressive episode with somatic syndrome |
| 16 | FEMALE | 47 | 28.09.98 | 28 | F32.10 | Moderate depressive episode without somatic syndrome |
| 17 | FEMALE | 46 | 28.09.98 | 32 | F32.11 | Moderate depressive episode with somatic syndrome |
| 18 | MALE | 51 | 28.09.98 | 37 | F32.11 | Moderate depressive episode with somatic syndrome |
| 19 | FEMALE | 37 | 13.10.98 | 16 | F32.11 | Moderate depressive episode with somatic syndrome |
| 20 | MALE | 42 | 28.10.98 | 35 | F33.11 | Recurrent depressive episode (moderate severity) with somatic syndrome |
| 21 | MALE | 47 | 28.10.98 | 29 | F33.11 | Recurrent depressive episode (moderate severity) with somatic syndrome |
| 22 | MALE | 32 | 30.10.98 | 24 | F32.10 | Moderate depressive episode without somatic syndrome |

| Subject No. | Sex | Age | Date tested | BDI score | ICD-10 | Diagnosis |
|---|---|---|---|---|---|---|
| 23 | FEMALE | 27 | 30.10.98 | 30 | F32.10 | Moderate depressive episode without somatic syndrome |
| 24 | FEMALE | 54 | 20.11.98 | 32 | F32.10 | Moderate depressive episode without somatic syndrome |
| 25 | MALE | 55 | 04.12.98 | 41 | F32.20 | Severe depressive episode without psychosis |
| 26 | FEMALE | 50 | 04.12.98 | 33 | F32.11 | Moderate depressive episode with somatic syndrome |
| 27 | MALE | 35 | 12.01.99 | 13 | F32.20 | Severe depressive episode without psychosis (recovering) |
| 28 | MALE | 57 | 12.01.99 | 33 | F32.11 | Moderate depressive episode with somatic syndrome |
| 29 | MALE | 73 | 29.01.99 | 22 | F33.30 | Recurrent depressive episode (severe) with psychosis |
| 30 (sp) | MALE | 55 | 01.12.98 | 40 | F31.4 | Bipolar affective disorder, current episode severe depression w/o psychotic sypmtoms |
| 31 (sp) | MALE | 49 | 08.12.98 | 49 | F31.4 | Bipolar affective disorder, current episode severe depression w/o psychotic sypmtoms |
| 32 (sp) | FEMALE | 44 | 19.01.99 | 32 | F42.2 | Obsessive compulsive disorder – mixed obsessional thoughts and acts<br>In addition suffering from bipolar affective disorder |

ICD-10 is the Classification of Mental Health Disorders (World Health Organisation). BDI scores can range between 0-60. sp = patient about to undergo anterior capsulotomy surgery for depression (tested pre surgery)

## APPENDIX 5.2: MEDICATION INFORMATION FOR INDIVIDUAL PATIENTS

| Subject No. | SSRI | Tricyclic | SNRI | Antipsychotic | NaSSA | Steroid | Sedative | Lithium | Herbal | NIL |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 40mg fluoxetine | | | | | | | | | |
| 2 | 40mg fluoxetine | | | | | | | | | |
| 3 | 100mg sertraline | | | | | | | | | |
| 4 | 150mg sertraline | | | | | | | | | |
| 5 | 40-50mg paroxetine | | | | | | | | | |
| 6 | 20mg citalopram | | | | | | 5-10mg nitrazipam | | | |
| 7 | | 250mg clomipramine | | | | | | 600mg lithium | | |
| 8 | 20mg paroxetine | | | | | | | | | |
| 9 | | 150mg dothiepin | | | | | | | | |
| 10 | | 210mg lofepramine | | | | | | | | |
| 11 | 40mg paroxetine | | | | | | | | | |

| Subject No. | SSRI | Tricyclic | SNRI | Antipsychotic | NaSSA | Steroid | Sedative | Lithium | Herbal | NIL |
|---|---|---|---|---|---|---|---|---|---|---|
| 12 | | | | | | | | | | ✓ |
| 13 | | | 75mg venlafaxine | | | | | | | |
| 14 | | | | | | | | | | ✓ |
| 15 | 20mg paroxetine | | | | | | | | | |
| 16 | | 50mg amitriptyline | | | | | | | | |
| 17 | 300mg nefazodone | | | | | | | | | |
| 18 | 20mg paroxetine | | | | | | | | | |
| 19 | | | | | | | | | St John's Wort | |
| 20 | 20-30mg paroxetine | | | | | | | | | |
| 21 | | | | | | | | | | |
| 22 | 20mg paroxetine | | | | | | | | | |
| 23 | 50mg citalopram | | | | | | | | | |

| Subject No. | SSRI | Tricyclic | SNRI | Antipsychotic | NaSSA | Steroid | Sedative | Lithium | Herbal | NIL |
|---|---|---|---|---|---|---|---|---|---|---|
| 24 | | | | | | | | | | ✓ |
| 25 | | | | | mirtazepine | | | 800mg lithium | | |
| 26 | 50mg paroxetine | | | | | | | | | |
| 27 | 30mg paroxetine | | | | | | | | | |
| 28 | | | 37.5mg venlafaxine | | | | | | | |
| 29 | | 200mg amitriptyline | | 200mg quetiapine | | 7.5mg prednisolone | | 200mg lithium | | |
| 30 (sp)* | 200mg nefazodone 300mg imipramine | | | 10mg olanzapine | | | 5mg diazepam | | | |
| 31 (sp)* | | | | | | | 5mg diazepam | 400mg lithium | | |
| 32 (sp)* | | | | | | | 1mg lorazepam | 1g lithium | | |

sp = patient about to undergo anterior capsulotomy surgery for intractable depression (tested pre surgery)

* Complete details of all medication (pre and post surgery) can be found in Appendix 6.1

## APPENDIX 5.3: GENERAL INFORMATION FOR CONTROL SUBJECTS

| Subject No. | Sex | Age | Date tested | BDI score |
|---|---|---|---|---|
| 1 | FEMALE | 30 | 16.12.98 | 0 |
| 2 | FEMALE | 28 | 17.12.98 | 8 |
| 3 | FEMALE | 28 | 18.12.98 | 1 |
| 4 | FEMALE | 44 | 21.12.98 | 2 |
| 5 | FEMALE | 48 | 21.12.98 | 7 |
| 6 | FEMALE | 36 | 22.12.98 | 0 |
| 7 | FEMALE | 31 | 22.12.98 | 4 |
| 8 | FEMALE | 46 | 22.12.98 | 1 |
| 9 | FEMALE | 31 | 29.01.99 | 3 |
| 10 | FEMALE | 30 | 02.02.99 | 0 |

| Subject No. | Sex | Age | Date tested | BDI score |
|---|---|---|---|---|
| 11 | FEMALE | 39 | 03.02.99 | 9 |
| 12 | FEMALE | 40 | 03.02.99 | 4 |
| 13 | FEMALE | 34 | 04.02.99 | 5 |
| 14 | FEMALE | 35 | 08.02.99 | 0 |
| 15 | FEMALE | 54 | 14.02.99 | 5 |
| 16 | MALE | 29 | 13.08.99 | 2 |
| 17 | MALE | 32 | 14.08.99 | 2 |
| 18 | MALE | 26 | 28.08.98 | 7 |
| 19 | MALE | 21 | 09.09.98 | 3 |
| 20 | MALE | 35 | 09.09.98 | 3 |

| Subject No. | Sex | Age | Date tested | BDI score |
|---|---|---|---|---|
| 21 | MALE | 39 | 05.11.98 | 5 |
| 22 | MALE | 37 | 10.11.98 | 3 |
| 23 | MALE | 50 | 17.11.98 | 9 |
| 24 | MALE | 47 | 22.11.98 | 1 |
| 25 | MALE | 59 | 15.12.98 | 7 |
| 26 | MALE | 40 | 28.01.99 | 2 |

| Subject No. | Sex | Age | Date tested | BDI score |
|---|---|---|---|---|
| 27 | MALE | 46 | 28.01.99 | 3 |
| 28 | MALE | 32 | 28.01.99 | 6 |
| 29 | MALE | 39 | 02.02.99 | 1 |
| 30 | MALE | 30 | 02.02.99 | 0 |
| 31 | MALE | 31 | 04.02.99 | 1 |
| 32 | MALE | 42 | 05.02.99 | 6 |

BDI scores can range between 0-60.

## APPENDIX 5.4: INFORMATION AND CONSENT FORMS FOR PATIENT AND CONTROL PARTICIPANTS

### Patient Information Form

FACIAL EXPRESSION SENSITIVITY IN DEPRESSION.

We would like to ask you to take part in a research project. To help you understand what the research is about, please read the following information. Be sure to ask any questions you have and we will do our best to explain and to provide any further information you require.

*Why are you asking me to participate?*

We are interested in examining whether perception of facial expressions is affected by people's mood. We hope results from this study will help us understand more about depression.

*What will the research involve?*

This study involves you completing a short questionnaire about your mood, reading out a short list of irregular words and looking at some faces showing different expressions. This will probably last about 30 minutes. Faces with changing expressions will be shown on a computer screen. You will be asked to decide which expression is being shown and to choose the point where you can first recognize the emotion. There are several practice trials so that you become accustomed to the testing procedure, which is very straightforward. A researcher will be with you throughout should you have any questions at any point.

*Who will have access to the tests that I do?*

We will treat all of your results as highly confidential. Only three researchers will have access to your data and it will be stored using a code rather than your real name. The results will not be entered into your medical records. You will be entitled to see the results of any test that you have done.

# Facial Expression Sensitivity in Depression

STATEMENT OF CONSENT

I agree to take part in the above named study. I have read and understood the information given to me. I understand that I am taking part voluntarily and am under no obligation. I can withdraw from the study at any time. I give consent for appropriate personnel to review my medical notes on a strictly confidential basis, and for my GP to be told of my participation in this study.

Name (Print): ..... Date: .....

Signature: .....

I confirm that I have fully explained the purpose and nature of this study to the above named patient, that I have supplied the appropriate written information, and given the opportunity to ask questions.

Doctor's Name: ..... Date: .....

Researcher's Name: .....

Signature: .....

# Participant Consent Form

## INDIVIDUAL DIFFERENCES IN FACIAL EXPRESSION SENSITIVITY

I am interested in examining whether perception of facial expressions is affected by people's mood and will be comparing your data as a control participant with data from patients with clinically diagnosed affect disorders. For example, clinically depressed individuals could be more sensitive to expressions of sadness compared to non-depressed individuals, as this emotion is consistent with their own affective state.

This study involves you completing a short questionnaire about your mood, reading out a short list of irregular words and then looking at some faces showing different expressions on a computer. You will be asked to decide which expression is being shown and to choose the point where you can first recognize the emotion. There are several practice trials so that you become accustomed to the testing procedure, which is very straightforward. The study will probably take you about 30-40 minutes in total. A researcher will be with you throughout should you have any questions at any point.

Your data will be kept totally confidential and only my supervisor, Dr. David Perrett and I, will have access to it.

The purpose of this form is to ensure that you are willing to take part in this study and to let you understand what it entails. Signing this form does not commit you to anything you do not wish to do.

Have you read and understood the purpose of the experiment, described above? **YES / NO**

Have you had the opportunity to ask questions and discuss the study? **YES / NO**

Have you received satisfactory answers to your questions? **YES / NO**

Do you understand that you are free to withdraw from the study:

- at any time
- without having to give a reason for withdrawing

**YES / NO**

Do you agree to take part in the study? **YES / NO**

Signed _____ Date _____

Name in block letters _____ Date of Birth _____

## APPENDIX 5.5: GENERAL INFORMATION FOR MILDLY DEPRESSED SUBJECTS

| Subject No. | Sex | Age | Date tested | BDI score |
|---|---|---|---|---|
| 1 | FEMALE | 55 | 19.08.98 | 11 |
| 2 | FEMALE | 33 | 26.08.98 | 11 |
| 3 | FEMALE | 68 | 21.12.98 | 10 |
| 4 | FEMALE | 31 | 21.12.98 | 10 |
| 5 | FEMALE | 27 | 28.01.99 | 10 |
| 6 | FEMALE | 47 | 01.02.99 | 12 |
| 7 | FEMALE | 42 | 09.02.99 | 13 |
| 8 | FEMALE | 36 | 11.02.99 | 13 |
| 9 | FEMALE | 40 | 15.02.99 | 10 |

| Subject No. | Sex | Age | Date tested | BDI score |
|---|---|---|---|---|
| 10 | MALE | 54 | 19.08.98 | 11 |
| 11 | MALE | 38 | 05.11.98 | 12 |
| 12 | MALE | 38 | 09.11.98 | 19 |
| 13 | MALE | 62 | 02.02.99 | 10 |

BDI scores can range between 0-60.

## APPENDIX 6.1: COMPLETE MEDICATION INFORMATION (PRE AND POST SURGERY) FOR ALL 3 PATIENTS

| PATIENT 1 ON ADMISSION (NOVEMBER 1998) | | |
|---|---|---|
| **DRUG** | **THERAPEUTIC CLASS** | **DOSE** |
| NEFAZODONE | ANTIDEPRESSANT | 200mg, 2 times daily |
| IMIPRAMINE | ANTIDEPRESSANT | 300mg, daily |
| OLAZAPINE | ANTIPSYCHOTIC | 10mg, at night |
| BUSPIRONE | ANXIOLYTIC | 10mg, 3 times daily |
| DIAZEPAM | ANXIOLYIC | 5mg, 2 times daily<br>10mg, at night |
| TEMAZEPAM | SEDATIVE | 20mg, at night |
| ZOPICLONE | SEDATIVE | 15mg, at night |
| DIHYDROCODEINE | ANALGESIC | 4-6 hourly |
| LIOTHYRONINE | for HYPOTHYROIDISM | 40μg, daily |
| METHYLPHENIDATE | ANTI-NARCOLEPSY | 20mg, daily |

| PATIENT 1 ON DISCHARGE (08.01.99) | | |
|---|---|---|
| **DRUG** | **THERAPEUTIC CLASS** | **DOSE** |
| NEFAZODONE | ANTIDEPRESSANT | 200mg, 2 times daily |
| IMIPRAMINE | ANTIDEPRESSANT | 300mg, daily |
| OLAZAPINE | ANTIPSYCHOTIC | 10mg, at night |
| BUSPIRONE | ANXIOLYTIC | 10mg, 3 times daily |
| DIAZEPAM | ANXIOLYIC | 5mg, 2 times daily<br>10mg, at night |
| TEMAZEPAM | SEDATIVE | 20mg, at night |
| ZOPICLONE | SEDATIVE | 15mg, at night |
| LIOTHYRONINE | for HYPOTHYROIDISM | 40μg, daily |
| METHYLPHENIDATE | ANTI-NARCOLEPSY | 20mg, 3 times daily |

| PATIENT 2 ON ADMISSION (NOVEMBER 1998) | | |
|---|---|---|
| **DRUG** | **THERAPEUTIC CLASS** | **DOSE** |
| LITHIUM CARBONATE | ANTI-MANIA | 400mg, 2 times daily |
| CLONAZEPAM | ANTICONVULSANT | 2mg, 3 times daily |
| DROPERIDOL | ANTIPSYCHOTIC | 10mg, 2 times daily |
| PROCYCLIDINE | ANTICHOLINERGIC | 5mg, 2 times daily |
| DIAZEPAM | ANXIOLYTIC | 5mg, max. 3 in 24hrs (as required) |
| LACTULOSE | AMMONIA INHIBITOR | 10mls, 2 times daily |
| GAVISCON | ANTACID | 10mls, max. 4 in 24hrs (as required) |
| HYOSCINE | for HYPERSALIVATION | 300µg, max. 3 in 24hrs (as required) |
| CO-CODAMOL | ANALGESIC | 2 tablets, every 4-6hrs (as required) |
| CIPRIMIL | - | 60mg, daily |

| PATIENT 2 ON DISCHARGE (08.01.99) | | |
|---|---|---|
| **DRUG** | **THERAPEUTIC CLASS** | **DOSE** |
| LITHIUM CARBONATE | ANTI-MANIA | 400mg, 2 times daily |
| CLONAZEPAM | ANTICONVULSANT | 2mg, 3 times daily |
| DROPERIDOL | ANTIPSYCHOTIC | 10mg, 2 times daily |
| PROCYCLIDINE | ANTICHOLINERGIC | 5mg, 2 times daily |
| GAVISCON | ANTACID | 10mls, max. 4 in 24hrs (as required) |
| LACTULOSE | AMMONIA INHIBITOR | 10mls (as required) |
| HYOSCINE | for HYPERSALIVATION | 300µg, max. 3 in 24hrs (as required) |
| CITALOPRAM | ANTIDEPRESSANT | 60mg, daily |

| PATIENT 3 ON ADMISSION (18.01.99) | | |
|---|---|---|
| **DRUG** | **THERAPEUTIC CLASS** | **DOSE** |
| LITHIUM CARBONATE | ANTI-MANIA | 1g, at night |
| LORAZEPAM | ANXIOLYTIC | 1mg, in the morning |
| DISULFIRAM | ANTI-ALCHOLIC / METABOLIC BALANCE | 200mg, in the morning |
| THYROXINE | REGULATION OF THYROID FUNCTION | 150µg, daily |
| CO-DYDRAMOL | ANALGESIC | 2 tablets, max. 8 in 24hrs (as required) |

| PATIENT 3 ON DISCHARGE (12.02.99) | | |
|---|---|---|
| **DRUG** | **THERAPEUTIC CLASS** | **DOSE** |
| LITHIUM CARBONATE | ANTI-MANIA | 1g, at night |

## APPENDIX 7.1: THE EATING DISORDERS INVENTORY (EDI)

| ALWAYS | USUALLY | OFTEN | SOMETIMES | RARELY | NEVER | | |
|---|---|---|---|---|---|---|---|
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 1. | I eat sweets and carbohydrates without feeling nervous |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 2. | I think that my stomach is too big |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 3. | I wish that I could return to the security of my childhood |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 4. | I eat when I am upset |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 5. | I stuff myself with food |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 6. | I wish that I could be younger |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 7. | I think about dieting |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 8. | I get frightened when my feelings are too strong |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 9. | I think that my thighs are too large |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 10. | I feel ineffective as a person |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 11. | I feel extremely guilty after overeating |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 12. | I think my stomach is just the right size |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 13. | Only outstanding performance is good enough in my family |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 14. | The happiest time in life is when you are a child |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 15. | I am open about my feelings |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 16. | I am terrified of gaining weight |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 17. | I trust others |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 18. | I feel alone in the world |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 19. | I feel satisfied with the shape of my body |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 20. | I feel generally in control of things in my life |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 21. | I get confused about what emotion I am feeling |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 22. | I would rather be an adult than a child |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 23. | I can communicate with others easily |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 24. | I wish I were someone else |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 25. | I exaggerate or magnify the importance of weight |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 26. | I can clearly identify what emotion I am feeling |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 27. | I feel inadequate |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 28. | I have gone on eating binges where I have felt that I could not stop |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 29. | As a child, I tried very hard to avoid disappointing my parents and teachers |

| ALWAYS | USUALLY | OFTEN | SOMETIMES | RARELY | NEVER | | |
|---|---|---|---|---|---|---|---|
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 30. | I have close relationships |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 31. | I like the shape of my buttocks |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 32. | I am preoccupied with the desire to be thinner |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 33. | I don't know what is going on inside me |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 34. | I have trouble expressing my emotions to others |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 35. | The demands of adulthood are too great |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 36. | I hate being less than best at things |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 37. | I feel secure about myself |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 38. | I think about bingeing (over-eating) |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 39. | I feel happy that I am not a child anymore |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 40. | I get confused as to whether or not I am hungry |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 41. | I have a low opinion of myself |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 42. | I feel that I can achieve my standards |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 43. | My parents have expected excellence of me |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 44. | I worry that my feelings will get out of control |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 45. | I think that my hips are too big |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 46. | I eat moderately in front of others and stuff myself when they are gone |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 47. | I feel bloated after eating a normal meal |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 48. | I feel that people are happiest when they are children |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 49. | If I gain a pound, I worry that I will keep gaining |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 50. | I feel that I am a worthwhile person |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 51. | When I am upset, I don't know if I am sad, frightened or angry |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 52. | I feel that I must do things perfectly, or not do them at all |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 53. | I have the thought of trying to vomit in order to lose weight |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 54. | I need to keep people at a certain distance (feel uncomfortable if someone tries to get too close) |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 55. | I think that my thighs are just the right size |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 56. | I feel empty inside (emotionally) |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 57. | I can talk about personal thoughts or feelings |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 58. | The best years of your life are when you become an adult |

| ALWAYS | USUALLY | OFTEN | SOMETIMES | RARELY | NEVER | | |
|---|---|---|---|---|---|---|---|
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 59. | I think that my buttocks are too large |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 60. | I have feelings I can't quite identify |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 61. | I eat or drink in secrecy |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 62. | I think that my hips are just the right size |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 63. | I have extremely high goals |
| ( ) | ( ) | ( ) | ( ) | ( ) | ( ) | 64. | When I am upset, I worry that I will start eating |

## APPENDIX 7.2: PATIENT INFORMATION AND CONSENT FORM

THE MAUDSLEY
*Advancing Mental Health Care*

### Facial Expression Study Consent Form

**Contact: Fay Murphy at the Eating Disorders Unit, Bethlem Royal Hospital or Institute of Psychiatry (ext.: 3134)**

We would like to invite you to take part in a study looking at peoples' ability to recognise different emotions. The study will take approximately 60 minutes in all. There are two procedures which include.

1. You will firstly be asked to complete a questionnaire in which you will be required to tick boxes in an attempt to assess your mood.
2. Secondly, you will be asked to rate emotions on a computer generated programme displaying different levels of emotion. This part of the project involves the use of a computer. It is a simple task and will be carefully explained to you.

Participation in this study is entirely voluntary. If you consent to take part, all information will be strictly confidential.

If you decide not to take part this in no way affects any treatment you may be receiving at the Maudsley and Bethlem Royal Hospitals.

If you have any questions before taking part or the instructions are not very clear, please ask the investigator.

This study is being carried out by Dr. Janet Treasure, Lindsey Murray and Fay Murphy.

---

I have received sufficient information regarding this study and I consent to take part.

Name
(please print)
Signature

Witness Name

Signature