UNIVERSITY OF SOUTHAMPTON

FACULTY OF MEDICINE, HEALTH & LIFE SCIENCES

School of Psychology

Appraisal biases and their association to the amygdala in social anxiety

Rebecca Lee

This thesis is submitted in partial fulfilment of the degree of

Doctor of Clinical Psychology

May 2008

Word Count: 14,818

Thesis Abstract

This thesis addresses biases in appraisal that are thought to contribute to the development and maintenance of social anxiety. The literature review outlines several theories of anxiety, before focussing on cognitive theories of social anxiety which predict that individuals with social anxiety have a bias in threat appraisal. Contemporary cognitive-motivational and neurocognitive theories of anxiety are then detailed and their relevance to social anxiety is described. Recent theories make efforts to integrate cognitive theory of appraisal with functional neuroanatomy, proposing that the amygdala is involved in threat appraisal. Research has provided support for the presence of appraisal biases in social anxiety and recent neuroimaging evidence suggests that threat appraisal of social cues is associated with potentiation of the amygdala in social anxiety. The utility of the startle response, in further investigating the predictions of the neurocognitive theory is described.

The empirical paper investigates the predictions of neurocognitive theory that sub-cortical appraisals of social cues are associated to potentiated amygdala response. The startle response, a behavioural index of sub-cortical appraisal, was used to investigate response to social cues (neutral and fear faces) and non-social fear cues (light and dark patches) in individuals high and low in social anxiety (HSA vs. LSA). It was found that both groups had a potentiated startle response to the dark condition compared to the light and face conditions and there were no group differences in the response to social cues contrary to theoretical predictions. The findings are discussed in relation to theory and previous research findings.

Acknowledgements

This research was carried out under the supervision of Dr. Matt Garner at the University of Southampton. I would like to thank him for his consistent support throughout the entire process of completing the dissertation. I would also like to thank Dr. David Baldwin for his guidance on the project. The experimental element of the research was conducted at the Department of Psychiatry, Royal South Hants Hospital, who kindly provided the facilities. Participant recruitment was undertaken with Caroline Gamble, a fellow Trainee Clinical Psychologist, I would like to thank her for her invaluable input. Finally, I would like to thank all the participants who took part in the study and made the research possible.

Contents Page

Literature Review Contents

Empirical Paper Contents

List of Tables

List of Figures

LITERATURE REVIEW

A neuro-cognitive approach to social anxiety: The association between appraisal biases and the amygdala.

Rebecca Lee

Prepared for submission to Clinical Psychology Review- see Appendix A for instructions to authors.

A neuro-cognitive approach to social phobia: The association between appraisalbiases and the amygdala.

Abstract

Social phobia is a prevalent anxiety disorder that can be debilitating for individuals who experience extreme distress within social situations. Neuro-cognitive models of social phobia draw upon two theoretical fields: cognitive psychology and neurobiology. This review will examine the origins and predictions of this contemporary approach to understanding social phobia and review evidence from cognitive and neurobiological experimental paradigms. In particular, the utility of fear-potentiated startle methodolgies in investigating the association between neural structures and emotion processing will be reviewed.

1. Introduction

Social phobia is a commonly diagnosed anxiety disorder, with lifetime prevalence lying between 7% and 13% (Fehm, Pelissolo, Furmark, & Wittchen, 2005). A large proportion of individuals diagnosed with social phobia experience academic underachievement, unemployment and relationship difficulties (Fehm et al., 2005; Judd, 1994). In addition, it is common for these individuals to experience co-morbid difficulties including depression and drug and alcohol abuse.

Cognitive theory suggests that concerns with social-evaluation are caused and maintained by a range of cognitive biases (biases in information processing) that promote perceptions of threat and danger within the social situation. Specific biases are proposed to exist in discrete aspects of cognition such as appraisal, interpretation of ambiguity, attention and memory. Of these biases, it has been argued that biases in threat appraisal result from maladaptive functioning in a neural structure implicated in fear acquisition and responding; the amygdala (Bishop, 2007).

The aim of this review is to provide a critical discussion of the predictions and evidence for a neuro-cognitive theory of social phobia, with particular reference to following questions:

(I) What is the evidence for appraisal biases in social phobia?

(II) Is there evidence for amygdala involvement in the appraisal of threat?

(III) Is there evidence of an association between appraisal bias and amygdala activity in social phobia?

A literature search was undertaken using *Web of Science,* searching for articles published between 1997 and 2008. The rationale for the selected time period was to ensure a review of the current literature, key historical papers were also selected and were identified through frequent citations in current papers.

Key terms: anxiety, social phobia, fear, attention bias, interpretation bias, cognitive bias, self-focused attention, startle, neurobiology, neural pathway.

2. Social phobia: nature and prevalence

2.1. Anxiety

Anxiety is an evolutionary and normal reaction in response to danger (Greenberger & Padesky, 1995). The experience of anxiety allows a person to detect danger and respond both promptly and effectively in fearful situations (Mogg & Bradley, 1998). The autonomic arousal that is associated with anxiety, such as a racing heart or muscle innervation, are adaptive in the face of danger allowing the individual to either fight, flee, freeze or faint (Beck, Emery &

Greenberg, 1985). As well as the physiological symptoms described, anxiety is marked by behavioural symptoms such as escape or avoidance of the feared object to minimise risk taking in the individual and ensure safety. Affective symptoms include feeling frightened and apprehensive, whilst cognitive symptoms involve feelings of unreality, hyper-vigilance to threat, self-consciousness, poor concentration and difficulty reasoning (Beck et al., 1985). Despite its adaptive evolutionary basis, anxiety can become a problem for individuals when it occurs in the absence of actual danger (Greenberger & Padesky, 1995).

2.2 Social Phobia

Social phobia was first officially recognised as an anxiety disorder in 1980 in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association, 1980). The DSM-IV describes social phobia as a: "marked and persistent fear of social or performance situations in which embarrassment may occur" (p.411). It is marked by a fear of social situations in which the individual fears they are under scrutiny from others, which could be as extreme as walking into a waiting room where nobody notices the individual (Wells, 1997). Other common situations that people with social phobia fear include public speaking, social gatherings, eating in public, meeting new people and disagreeing with others (Leahy & Holland, 2000). Characteristics of an individual with social phobia are a fear of negative evaluation and negative self-evaluation (Wells, 1997). To meet the diagnostic criteria, an individual must fear one or more situations where they are exposed to scrutiny or strangers for fear of acting in a way that is humiliating or embarrassing. Exposure to the feared situation must provoke symptoms of anxiety in the individual and feared situations are either avoided or the individual suffers through them. The person must also recognise that the fear they experience in response to social situations is excessive and/or unreasonable. The fear, distress or avoidance that the individual experiences must also interfere with functioning to meet the diagnostic criteria. This can include disruption of relationships, social activities, occupational functioning and/or daily routine (APA, 1994). The DSM-IV also distinguishes between generalised social phobia and discrete social phobia, the former refers to an individual that fears most social or performance situations, whereas the latter is a category for those that only fear a small number of situations.

Fehm et al. (2005) undertook a review of European epidemiological studies and found a mean lifetime prevalence of 6.65% and a mean 12-month prevalence of 2%. In a review of forty-three studies, Furmark (2002) found a lifetime prevalence of 7-13%, suggesting that the variability was due to methodological variables. However, reports seem to clearly suggest that social phobia is highly prevalent, persistent, marked by profound disability and often has a high degree of co-morbid conditions (Fehm et al., 2005). Judd (1994) reported findings from California which found that 50% of people known to have social phobia fail to complete school and 70% lie within the lowest two quartiles of socio-economic status. A large proportion of individuals with social phobia have been found to have co-morbid conditions including: 59% with specific phobia, 45% with agoraphobia, 19% with concurrent alcohol abuse, 17% with major depression, 13% with concurrent drug abuse, 12% with dysthymia, 11% with obsessive-compulsive disorder and 5% with panic disorder. In 77% of cases, social phobia preceded the co-morbid condition (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992). The modal age of onset is 11-15 years of age, but social phobia has also been reported in children under the age of ten. Despite the early age of onset, the mean age of presentation for treatment is thirty. However, the majority of individuals are thought not to seek treatment (Rapee, 1995). The female to male ratio has been reported as two to one, however more males present for treatment than females. It has been suggested that this may be because it is less acceptable in society for men to be unable to assert themselves. These findings suggest that social phobia is a significant problem for individuals, society and services. It is therefore important to understand the aetiology and maintenance of the disorder in order to establish effective treatments.

3. Theoretical models of social phobia

3.1 Early cognitive models of anxiety

Cognitive theorists propose that emotional disorders, including anxiety disorders such as social phobia, result from biases in the way that an individual processes emotional information (Wells, 1997). Early cognitive models refer to anxiety in general, rather than specific diagnoses, however they provide relevant predictions regarding the aetiology of social phobia and provide background information to more recent models. Beck (1976) was the first cognitive theorist to produce a model to explain emotional disorders. His schema theory assumed that emotional disorders are maintained by distortions in thinking. He proposed that cognition is driven by schemas, which in the case of anxiety and social phobia, are sensitive to threat. Two types of schemata were argued to exist: beliefs such as 'I am vulnerable' and assumptions such as 'if I can't control anxiety I am a failure'. Beck believed that these beliefs and assumptions biased thinking, causing cognitive distortions or cognitive biases. Anxious individuals' schemata were thought to reflect a greater perceived sense of danger and decreased perceived ability to cope. Schemas were argued to bias an individual's cognitive processes including attention to threat, appraisal and memory (Beck et al., 1985). Social phobia is understood to be extreme anxiety specifically in response to social situations therefore, according to Beck's cognitive model, maladaptive cognitive biases would appear in response to social cues.

Although Beck's theoretical understanding of emotional disorders has been decisive in the development of cognitive theory, its shortcoming was that it did not distinguish between processing biases that differentiate emotional disorders e.g. depression and anxiety. Beck proposed that the biases in attention, appraisal and memory were a feature of both depression and anxiety. However, research revealed that it was only anxiety that was characterised by an attention bias towards threat (MacLeod, Mathews & Tata, 1986; Mogg, Mathews & Weinman, 1987), whereas depression was associated with a memory bias towards negative information (Clark & Teasdale, 1982).

Williams, Watts, MacLeod & Mathews (1988) were the first to develop a model that noted specific differences in cognitive function in anxiety and depression. Moving away from Beck's idea of a schema driven information processing model that influences attention, appraisal and memory, Williams et al. presented a model that focused upon pre-attentive processing of threat. The model involves the stimulus first entering an 'affective decision mechanism' which makes an initial appraisal of the stimulus to determine the threat value. The affective decision mechanism is influenced by state anxiety, such that high state anxiety mimics the effects of high threat input. Therefore as state anxiety increases more stimuli will be awarded a high threat value. An individual with social phobia is likely to experience high state anxiety in a social situation and therefore make more threatening appraisals of social cues. If the threat value is high enough, information is then passed to the 'resource allocation mechanism' which is influenced by trait anxiety. Those high in trait anxiety will direct attention towards threat, whereas those low anxious individuals will not attend to the threat. Thus, whereas Beck (1976,1985) argued that schemas biased cognitive processing, including attention, interpretation and memory of threat, Williams et al. proposed that pre-attentive threat appraisal biases influence cognitive processing.

However, a problem with the Williams et al. model is its prediction that low trait anxious individuals will direct attention away from threat, regardless of the severity of threat. This does not fit with the evolutionary models of fear, that instead propose that significant threat should be attended to by all individuals irrespective of level of trait anxiety (Mathews & Mackintosh, 1998; Mogg & Bradley, 1998).

Beck and Clark (1997) presented a three-stage schema based model. They proposed that the first stage of information processing involved the initial registration/recognition of threat information, which is likely to involve appraisal of threat. It was thought that this stage of processing is rapid, involuntary, outside conscious awareness and requires minimal processing resources. This is similar to the concept of pre-attentive processing described by Williams et al. (1988). This mode acts as an early warning detection system, which allows allocation of resources specifically to threat stimuli, which in social phobia are likely to be social cues. It is thought that at this stage anxious individuals hold an attentional bias towards threat. The next stage is the activation of a primal threat mode that allows immediate preparation. Cognitive, behavioural and affective patterns occur to minimise danger and maximise safety. These patterns include autonomic arousal, behavioural mobilisation (i.e. escape) or inhibition (i.e. avoidance), automatic repetitive thoughts/images, fear and hypervigilance. This stage of processing is also thought to be rigid and inflexible as it constricts processing to eliminate the capacity for reflexive thinking. At this stage an initial threat impression is made, however it is incomplete due to limited processing and thought to be biased towards an overestimation of threat in anxiety. Finally, the last stage of processing is 'secondary elaboration', which is the activation of more reflective modes of thinking. This is argued to be slow, effortful and schema driven. Both schemas relating to the context and self are activated and consideration of coping resources is undertaken by the individual. It is at this stage of processing that there is scope to thoughtfully re-appraise a situation to give it a different meaning (ie. less threatening) and hence bring about a change in the patterns described. Beck and Clark suggest that during this stage of processing, anxious individuals are likely to experience one of three outcomes. They may experience heightened anxiety as they block the re-appraisal of a situation or alternatively they may find that their anxiety decreases due to successful re-appraisal of the situation. Finally, the individual may have a reduction in anxiety due to the employment of the defensive behaviour described within the primal mode (eg. avoidance). This model combines aspects of both the Beck and Williams models, drawing together pre-attentive processing with more conscious, effortful and schema driven processing. Again, with reference to social anxiety, this model describes an initial appraisal of social stimuli as threatening, which precedes other cognitive biases such as attention to threat.

One of the most contemporary cognitive models of anxiety is the cognitive-motivational model proposed by Mogg & Bradley (1998). The model draws upon the concept of pre-attentive processing first described by Williams et al. (1998), which refers to rapid involuntary processing outside of conscious awareness. Mogg and Bradley predict that information processing is reliant on the combined functioning of two systems, a 'valence evaluation system' and 'goal engagement system'. Environmental stimuli are initially processed by the valence evaluation system, which generates threat appraisals. It is suggested that stimulus, context, state anxiety, biological preparedness and prior learning from previous contact with the stimulus all influence the functioning of this system. Therefore, it is predicted that anxious individuals will make more threat appraisals than those with low levels of anxiety. An individual with social phobia who is presented with a feared social situation, will probably experience high state anxiety and remember previous negative experiences, which would increase the likelihood of a threat appraisal being made. Threat appraisals to stimuli (i.e. social cues) that reach a high enough threshold activate the goal engagement system which interrupts goals and orientates attention to the threat stimulus. Overcoming some of the evolutionary criticisms of earlier models, such as Williams et al. (1988), Mogg and Bradley explain that in circumstances of high threat even low trait anxious individuals will direct attention to threat. The prediction is that high anxious individuals are characterised by an appraisal bias that is more likely to evaluate mild threat cues as threatening, and that this preattentive maladaptive bias in appraisal distorts subsequent aspects of higher-level cogntion (i.e. cognitive biases in attention, interpretation and memory). As such the model proposes that a pre-attentive threat appraisal bias is the principle vulnerability factor for anxiety disorders such as social anxiety.

Taken together, these models suggest social anxiety is chartacterised by a pre-attentive appraisal bias that readily evaluates concern relevant social cues as threatening. However, while general cognitive models of normal fear and pathological anxiety have implications for understanding the aetiology of social phobia, it is important that they are evaluated alongside predictions from disorder specific cognitive models.

3.2. Cognitive models of social phobia

Cognitive theorists suggest that information processing biases detailed in general anxiety models are also of relevance to understanding social phobia (Clark & McManus, 2002). However, social phobia differs somewhat from other conditions such as generalised anxiety disorder, as anxiety is experienced specifically in social scenarios rather than across several situations/areas of functioning.

Clark and Wells (1995) argued that a social situation activates negative assumptions based upon early social experience (e.g. 'I must always sound intelligent'), similar to the assumptions that Beck (1976) describes in his earlier model. The individual, based upon earlier experiences and the assumptions they hold, will appraise the social situation as dangerous. They will then become highly self-focused, using internal feelings to inform a perceived self-image of themselves in the situation. For example, if they were feeling hot or flushed, they may appraise this with a negative bias and think that they appear red and sweaty. At this stage the individual will engage in safety strategies (i.e. escaping) to prevent their feared catastrophe occurring. The result of engaging in safety strategies is that dysfunctional assumptions are not disproved and the subsequent reduction in anxiety is attributed to the safety behaviour, thus increasing the chances it will be used again. The experience of anxiety and the appraisals that are made by a social phobic are thought to create a vicious cycle that is repeated frequently. As with previous cognitive theories of anxiety, this model describes an initial appraisal bias towards threat in social phobia, highlighted by the relationship between "social situation" and "activates assumptions" in figure 1.

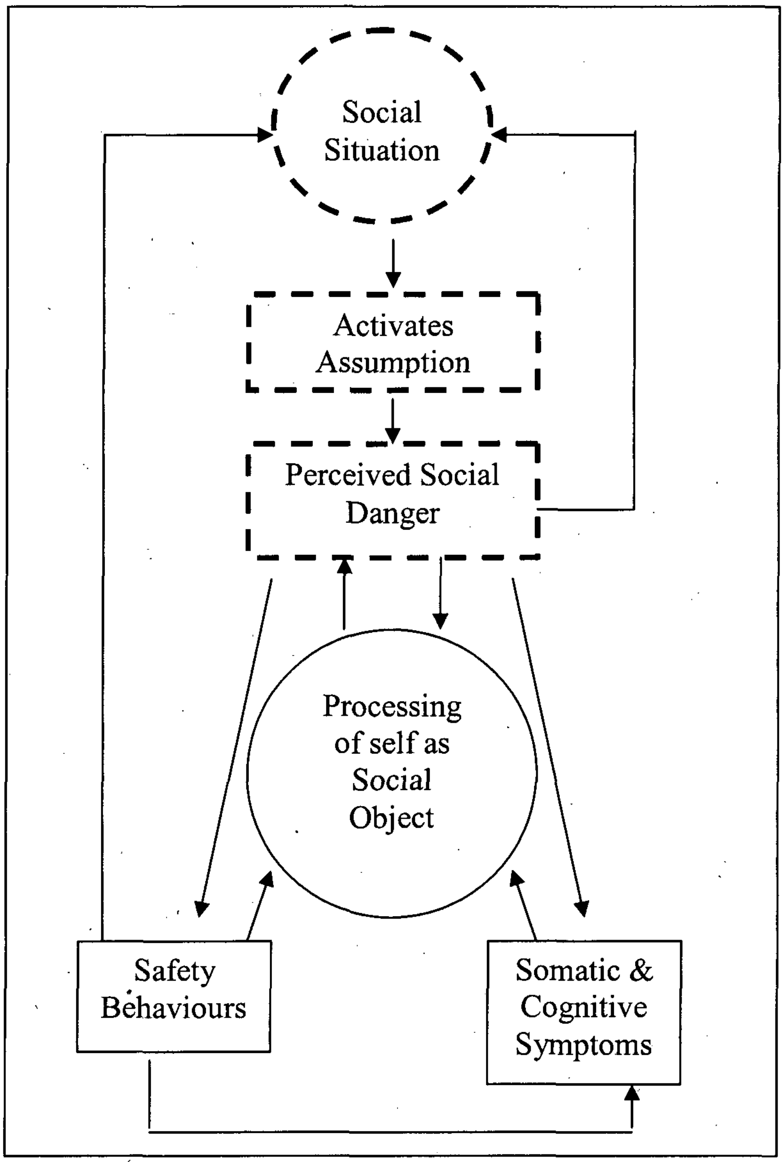

Figure 1. Clark & Wells' (1995) cognitive model of social phobia.

Clark and McManus (2002) extended predictions from Clark and Wells (1995), and proposed that social phobics will: interpret ambiguous events negatively; have a bias towards detecting negative social responses; have a reduction in resources to process external cues; selectively recall negative information and undertake protracted post-event processing.

Rapee and Heimberg (1997) also presented a cognitive model of social phobia (see figure 2), which shares some similarities with the Clark and Wells model. When in a social situation the authors predicted that a person with social phobia will generate a distorted image of their appearance. This representation is based upon attention to salient internal cues (eg. sweating, twitching, stammering) and external cues such as audience feedback. It is predicted that individuals with social phobia will detect external indicators of negative evaluation rapidly and find it difficult to disengage attention from them. It is also suggested that people with social phobia are more likely to appraise social cues with a negative bias. Representation of one's appearance is also thought to rely upon long-term memory, including prior experience and previous feedback, such as the memory of their appearance from mirrors. The model proposes that once this mental representation of self is generated, the individual formulates their perception of the audience's standard for performance. This standard is then compared against the individual's judgement of how the audience will evaluate them, which is predicted to be an underestimate in individuals with social phobia. The discrepancy between audience standard and perceived performance predicts the individual's view of the likelihood of negative evaluation. This elicits anxiety and therefore feeds into the mental representation of self and hence creates a vicious cycle.

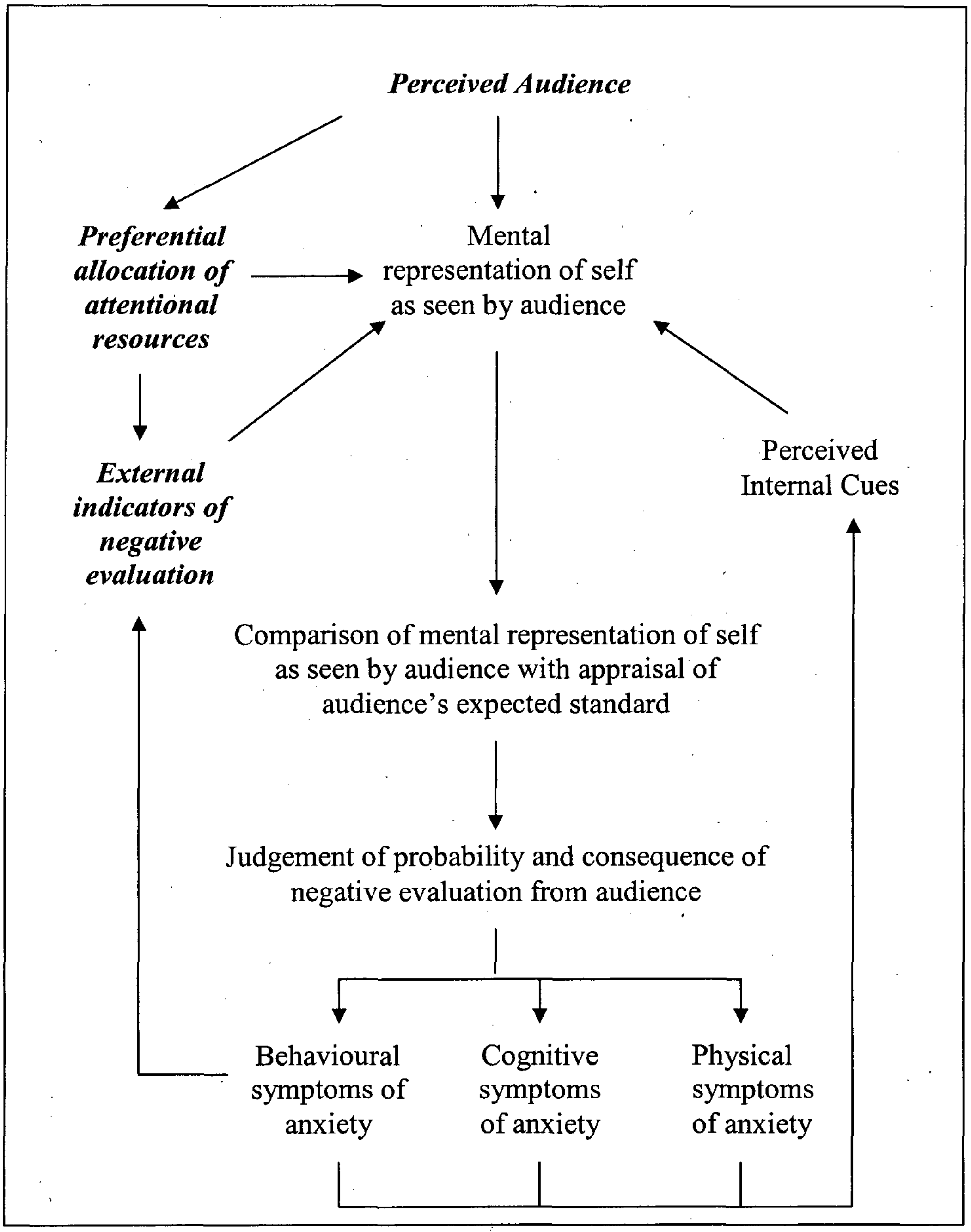

Figure 2. A model of the generation and maintenance of anxiety in social/evaluative situations (Rapee & Heimberg, 1997).

As with the Clark and Wells model, it is an initial appraisal of threat in response to a feared social situation that subsequently leads to further cognitive biases (e.g. self-focused attention, increased selective attention to external cues that signal negative evaluation). Thus specific models have similarities with general models of anxiety as they too predict that a maladaptive appraisal towards threat precedes biases in higher level cognition.

3.3 Neurobiological theory of anxiety

Neurobiological theorists have also proposed a mechanism thought to be central to the experience of anxiety. Based on much animal research, Le Doux (1995) proposed that a neural circuit, found to be involved in the experience of fear in rats, could also be involved in the experience of anxiety in humans. He argued that the amygdala and thalamic pathways are involved in automatic primary appraisals of threat. As such this neural network has been considered a plausible neural substrate that underlies the valence evaluation system described by Mogg and Bradley (1998). Le Doux described the processing involved in the primary appraisal of threat as automatic or 'quick and dirty', as it facilitates a rapid and coarse analysis of environmental threat. Le Doux argued that the amygdala is central in the experience of anxiety and through extensive projections to other neural regions including the thalamus and cortex, is able to influence high level cognitive operations e.g. selective attention, resolution of ambiguity and elaborative memory. As such, the amygdala and the distributed emotion/fear processing network could be considered to underlie both the valence evaluation and goal engagement mechanisms identified in the Mogg and Bradley model. As such, it too emphasises automatic threat evaluation as a precursor to cognitive and behavioural correlates of anxiety.

Ohman and Mineka (2001) also proposed that the amygdala was the central neural structure in fear elicitation. They argued that the activation of the amygdala is automatic, impenetrable to cognition and activated in aversive contexts; once triggered it cannot be deactivated by cognitive control. They suggested that the amygdala receives processed input from the thalamus and cortex and controls emotional output via the hypothalamus and brain stem nuclei.

Amaral (2002) proposed a working hypothesis of the function of the amygdala, suggesting that it is a 'protection device' that allows the organism to detect and avoid danger in the environment. The amygdala will co-ordinate a fear response typical for the species (eg. freeze or flee), which is activated via connections between the amygdala, thalamus and brainstem.

There has been a wealth of animal research undertaken to investigate the role of the amygdala in the experience of fear, particularly with rodents (Amaral, 2002; Davidson, 2002). Lesion studies with animals have allowed direct investigation of the association between discrete brain regions, behaviour and by inference, cognitive functioning. When investigating fear, animal models often rely upon a classical conditioning paradigm. This involves pairing a neutral and aversive stimuli to create a conditioned fear response to the previously neutral stimulus. It has been found in animal models that when the activity of the amygdala has been eradicated by a lesion or pharmacological disruption, the acquisition and extinction of fear is interrupted (Maren & Quirk, 2004). In addition, animal models have provided us with evidence that suggests the neural circuit involved in the expression of fear includes the dorsal prefrontal cortex, ventral orbital prefrontal cortex, amygdala, bed nucleus of the stria terminalis, hippocampus, locus coeruleus and raphe nuclei (Marcin & Nemeroff, 2003). In relation to social anxiety, Amaral (2002) has found that bilateral lesions of the amygdala in the social macaque monkeys results in a reduced fear response to objects and a socially uninhibited pattern of behaviour. Based on these findings, Amaral suggests that amygdala acts as a 'protective brake' in social phobia, which when dysfunctional (i.e. hyperactive) promotes greater fear response (i.e. appraisal of threat) and inhibited behaviour (i.e. social avoidance). The integration of neurobiological models of threat processing and cognitive formulations of anxiety has recently been formalised in a neurocognitive model of anxiety (Bishop, 2007) Specifically Bishop suggests that anxious individuals may have a hyperactive amygdala which increases the likelihood that mild threat stimuli are evaluated as threatening.

To provide evidence for the neurocognitive approach, the existence of appraisal biases in social phobia, the role of the amygdala in appraisal and an association between appraisal bias and the amygdala need to be demonstrated.

4. Evidence for appraisal biases in social phobia

It has been suggested in cognitive theories of social phobia that people with social anxiety appraise social cues with a negative bias (Clark & McManus, 2002; Rapee & Heimberg, 1997). The nature of the appraisal bias has been further specified by Mogg and Bradley (1998) in their description of the valence evaluation system as pre-attentive and operating outside of conscious awareness. For individuals with social phobia, if a social cue is appraised as threatening the goal engagement system is interrupted causing a re-direction of attentional resources. It is predicted that appraisal of threat to a social situation leads to cognitive biases such as increased self-focused attention and vigilance to external cues of negative evaluation (Rapee & Heimberg, 1997).

In recent years there has been much research generated into the area of cognitive biases in social phobia and anxiety more generally. For example, it has been found that socially anxious individuals direct attention towards social threat cues such as angry faces (Amir, Elias, Klumppa, & Przeworski, 2003; Mogg & Bradley, 2002; Musa, Lepine, Clark, Mansell, & Ehlers, 2003). In addition, attention appears to take a vigilant-avoidant style; negative social cues are attended to rapidly but, after brief observation, attention is directed away from the cue in an avoidant manner (Vassilopoulous, 2005; Mogg et al., 2004). Although there have been interesting findings from the attention literature, cognitive theory suggests a pre-attentive appraisal bias precedes (and modulates) other cognitive biases (e.g. attention). However, despite their theoretical importance, appraisal biases have received comparatively little empirical investigation.

In the empirical review that follows, data will be analysed from analogue studies that utilise sub-clinical samples to compare high and low socially anxious individuals and patient studies that compare individuals with social phobia typically to a non-anxious population

4.1 Social scenarios

Written scenarios regarding social situations have been used as a method to investigate appraisal biases in anxiety and social phobia. Generally the scenarios are ambiguous (e.g. the farmer gave Dave the sack) and participants are asked to endorse differently valenced (e.g. positive or negative) interpretations of the information. This approach has been used widely in early research of appraisal biases in anxiety, with findings suggesting that anxious individuals are more likely to appraise ambiguous sentences as threatening (Eysenck, Mogg, May, Richards, & Mathews, 1991). In social anxiety, Amin, Foa, & Coles (1998) utilised this paradigm by presenting participants with twenty-two ambiguous social and non-social scenarios. Each scenario was followed by three differently valenced interpretations: positive, negative and neutral, which participants were asked to rank order. The authors found that those with social phobia tended to choose more negative interpretations than the non anxious group and an obsessive compulsive disorder comparison group. Similarly, Voncken, Bogels, & de Vries (2003) found that social phobics were more likely than non-anxious controls to rate negative interpretations of scenarios as being more likely, consistent with a negative appraisal bias. Vassilopoulos (2006) presented high and low socially anxious individuals with unambiguous positive and mildly negative social situations and established appraisals by open questions and ratings of interpretations provided by the experimenter. Vassilopoulos found that high socially anxious individuals were more likely than the low anxiety group to appraise events as negative in both valence conditions. Wilson & Rapee (2005) presented participants with social and non-social scenarios that were either completed with a positive or negative description. Participants were asked to rate their belief in each interpretation. They found that individuals with social phobia were more likely than non-anxious controls to believe negative interpretations of negative social scenarios, but not positive scenarios.

Online inference refers to appraisals made at the time of encountering social information, which can be investigated using computerised reaction time tasks. Hirsch and Mathews (2000) utilised the online inference paradigm to investigate appraisal biases with lexical decision tasks. The *lexical decision* task utilises homographs, words that have the same spelling but both threatening and non-threatening meanings (eg. batter and punch). The homographs are used as primes and then followed by either a word or non-word, real words either relate to the threat or non-threat meaning of the prime. The prediction is that anxious individuals will take significantly less time to respond to negative interpretation of the ambiguous homographs. Participants read six realistic descriptions of a job interview and were asked to complete lexical decision tasks placed within the text, responding to words and non-words as quickly as possible. This is different to the classical scenario paradigms, as scenarios rely upon a retrospective rather than online appraisal. Cognitive theory would predict that biases in appraisal are made online. However, in this task Hirsch and Mathews did not observe any negative online inference biases by the high anxious group, only an absence of a positive bias in the low anxious group.

Amir, Beard, & Bower (2005) moved away from the concept of written scenarios and presented participants with videos that involved an actor walking up to the camera and commenting on an aspect of the participant's appearance. Participants had to rate how they would feel in that situation. Videos were of ambiguous, positive and negative valence. Those with social anxiety rated ambiguous videos more highly than non-anxious, high trait anxious and dysphoric control groups. Wenzel, Finstrom, Jordan, & Brendle (2005) also utilised videos, but presented six vignettes of positive, negative and neutral valence. Socially anxious individuals were found to make more negative interpretations of all videos, irrespective of valence. Taylor & Alden (2005) developed a more ecologically valid paradigm by creating a social interaction within the lab, with lab assistants inducing a positive or ambiguous social environment. Social phobia participants were not found to make negative interpretations of the social environment.

Evidence from off-line and on-line scenario paradigms suggests that both high social anxiety and social phobia groups appraise social scenarios with a negative bias. This provides evidence for cognitive theories that suggest a negative appraisal bias to social cues exists in social phobia and social anxiety.

4.2 Emotional faces

Another method in investigating appraisal bias is to utilise facial expressions as experimental stimuli, as they are a common social cue utilised by social phobics to judge audience evaluation (described by Rapee and Heimberg, 1997). Generally, faces are either ambiguous, positive or negative and participants are asked to endorse differently valenced interpretations, as with the scenario tasks. Winton, Clark & Edelmann (1995) presented high and low socially anxious individuals with pictures of neutral and negative facial expressions. Pictures were presented for 60 milliseconds and participants were asked to identify the affect of the facial expressions. In a second task, participants were asked to convey the overall emotion presented in short video clips. Winton et al., predicted, based upon cognitive theory, that the group high in social anxiety would show an enhanced ability to detect negative emotion. However, contrary to finding evidence of enhanced sensitivity, high socially anxious individuals were more likely to appraise all facial expressions negatively (i.e. a pervasive negative response bias)

Richards, French, Calder, Webb, Fox & Young (2002) utilised morphed faces as experimental stimuli. Morphed faces were created by combining or 'morphing' two facial expressions to various degrees (i.e. 90% fear: 10% sadness; 70% fear: 30% sadness). This allows the presentation of ambiguous information to be measured and controllable. Participants were asked to fixate on a central point of the screen, depicted using a cross, and were then presented with the face until a verbal response was made. Responses were prompted by the labelling of six emotions appearing on the screen. Richards et al. found that when fear was one of the component emotions, high socially anxious individuals showed increased sensitivity towards fear. However, there was no evidence of enhanced sensitivity for other negative emotions (e.g. anger), as would be predicted by cognitive theory.

Joormann and Gotlib (2006) also utilised morphed faces in their experimental design. They presented participants with 70 photographs of the same face that gradually expressed greater degrees of sadness, anger, fear and happiness. Participants were asked to press a key when they had identified the face, then a rating scale appeared where participants were asked to rate the facial expression. The authors found that the social phobia group required less intensity in the facial expression to detect angry faces than depressed patients or non-anxious individuals.

Yoon & Zinbarg (2007) presented participants with pairs of pictures, one as a cue and presented first, the other presented as a target. Cues consisted of question marks and angry, happy, disgust or neutral faces. Targets were either angry, happy or disgust faces. Prior to picture presentation participants were asked to undertake a 5-minute task, either writing a short essay or a speech. During the appraisal tasks participants were asked to press a response key as soon as they had decided upon the facial expression depicted in the picture. Appraisal was implied through reaction times. The authors found that high socially anxious individuals showed shorter reaction times in response to threatening faces following a neutral cue which was suggested to reflect their negative appraisal of neutral faces in social anxiety.

Gilboa-Schechtman, Presburger, Marom, & Hermesh (2005) presented participants with audiences of mixed facial expressions. The range of emotions expressed by the crowd included: extremely approving, moderately approving, neither approving nor disapproving, moderately disapproving (MD), and extremely disapproving (ED) They found that audiences with more disapproving faces were rated by social phobics with more negativity than controls. In addition, they found that the social phobia group had shorter response latencies in response to disapproving audiences, supporting Clark and McManus' prediction that there is a bias towards detection of negative social cues in social anxiety.

Evidence from appraisal research, that has utilised facial expressions as social cues, indicates that those with social phobia and social anxiety appraise emotional and ambiguous faces with a negative bias (Winton et al., 1995; Gilboa-Schectman et al., 2005). In addition, there is evidence to suggest that high social anxiety groups are sensitive to detecting faces depicting negative emotions (e.g. fear or anger) and that they are quicker at making appraisals of negative social cues (Richards et al., 2002; Joormann & Gotlib, 2006; Yoon & Zinbarg, 2007). This provides evidence for neurocognitive theory which suggests that threat appraisals are more likely and made rapidly in fear states (LeDoux, 1995).

One issue with appraisal research is the concept of response bias versus sensitivity. Response bias occurs when an individual repeatedly classifies stimuli with the same valence. However, sensitivity relates to the ability to discriminate between stimuli. While some studies have distinguished between these two aspects of task performance (e.g. Winton et al.) many studies to date have not. As such, future studies are encouraged to examine the component mechanisms involved in explicit appraisal. In addition, researchers should also use other indices of threat appraisal that might more accurately tap automatic, preattentive appraisal mechanisms that are proposed to operate implicitly and outside of awareness; and arguably precede the explicit appraisal biases studied to date.

5. The amygdala and appraisal

As noted earlier, neurocognitive theory predicts that the amygdala is the central neural structure involved in the appraisal of threat and that it may be oversensitive in individuals experiencing anxiety. While much of the research that has implicated the amygdala in fear acquisition, maintenance and expression has come from animal studies (see earlier), recent developments have enabled this amygdala functioning to be investigated in human participants.

5.1 The function of the amygdala in humans

In human studies, neuroimaging techniques such as functional MRI scans (fMRI) and positron-emission tomography (PET) allow the examination of the association between a neural structure and a cognitive process (Sarter, Bernston & Cacioppo, 1996). It has been found that individuals with specific amygdala damage have impairments in the recognition of facial expressions depicting fear, therefore indicating that the amygdala is involved in fear recognition (Broks, Young, Maratos, Coffey, Calder, Isaac, et al., 1998). In addition, patients with bilateral amygdala damage have been found to judge unfamiliar people to be more approachable and trustworthy than healthy volunteers (Adolphs, Tranel & Damasio, 1998). These neuropsychological studies describe patterns of behaviour in individuals with bilateral amygdala damage, which indicate difficulties in threat recognition and appraisal.

In a neuroimaging study, Whalen, Rauch, Etcoff, McInerney, Lee & Jenike (1998) found that individuals presented with masked fear faces, which could not be consciously perceived, therefore processed pre-attentively, showed activation of the amygdala. Carlsson, Petersson, Lundqvist, Karlsson, Ingvar, & Ohman (2004) found that phobics showed greater amygdala response to phobic or fear-relevant stimuli under conditions of non-awareness compared to neutral pictures. Increased amygdala activation in response to masked fear stimuli suggests that the amygdala is involved with pre-attentive fear processing consistent with the amygdala being a plausible neural substrate for the valence evaluation system described in the cognitive literature (Mogg & Bradley, 1998).

The research with human participants described here indicates that people with impaired amygdala function have difficulty with threat appraisal and fear recognition, suggesting an association between threat appraisal and the amygdala, which supports earlier animal data and models. This evidence suggests that the amygdala may also be involved in threat appraisal *biases* in anxiety. The neurocognitive approach not only predicts that the amygdala is central to threat appraisal, but that it is more highly activated in individuals experiencing anxiety. This increased activation is also predicted to be associated to more frequent appraisals of threat in anxious individuals. Individuals with social phobia are predicted to make more threat appraisals specifically to social cues, such as facial expressions. It is important to consider whether amygdala activation is associated with appraisal biases in social phobia.

5.2 Relationship between appraisal biases and the amygdala in social anxiety

Based upon neurocognitive theory, it is predicted that those high in social anxiety are more likely to make threat appraisals of social cues and this may be associated to the hypersensitivity of the amygdala. In a neuroimaging study, Birbaumer, Grodd, Diedrich, Klose, Erb, Lotze et al. (1998) found that individuals with social phobia had greater activation of the amygdala in response to neutral faces, compared to the non-anxious group. The response to neutral faces and negative faces were similar in the social phobia group, further, they were comparable to amygdala responses to negative stimuli in the non-phobic group. Thus the findings from the Birbaumer study indicate that processing of ambiguous information in social phobics is equivalent to threat stimuli, suggesting a processing bias towards ambiguous information. Similarly, Somerville, Kim, Johnstone, Alexander, & Whalen (2004) have found that high trait anxious individuals showed an increased amygdala response whilst viewing neutrally valenced pictures. The evidence suggests that the amygdala has a role in the processing of fear stimuli and that individuals with social anxiety appear to display similar amygdala activation to ambiguous and negative social stimuli. This further supports neurocognitive models that link cognitive biases in appraisal with heightened amygdala activity.

Research to investigate the predictions of the neurocognitive theory in relation to social phobia is limited. However, early evidence suggests that appraisal biases towards threat in response to social cues, specifically ambiguous stimuli, are associated to increased activation of the amygdala. Neuroimaging is not the only technique to investigate the association between the amygdala and appraisal biases. A method known as the startle technique may also be useful in investigating amygdala activation within cognitive experimental tasks.

6. The startle response: Providing evidence for neurocognitive theories of anxiety

6.1 The startle response

The startle reflex is an automatic response which consists of a rapid contraction of the orbicularis oculi muscle under the eye. It is argued that the startle response has a clear anatomical basis, with the amygdala projecting to the reticularis pontis caudalis which modulates the startle reflex (Shi & Davis, 2001). It provides a direct reflection of the activation of a fear circuit, particularly focused on the amygdala (Ohman & Mineka, 2001). Individuals who have undergone unilateral left temporal lobe lobectomy, including the amygdala, fail to show a startle response (Funayama, Grillon, Davis, & Phelps, 2001) and startle modulation is related to an increase in cerebral blood flow to amygdaloid-hippocampul region (Pissiota, Frans, Michelgard, Appel, Langstrom, Flaten et al., 2003). Experimentally, the startle reflex can occur in response to brief and intense auditory, tactile or visual stimuli. Typically, the startle response is modulated by a short acoustic (<50ms) white noise which is usually between 90 and 110 decibels. Contraction of the orbicularis oculi muscle is measured using an electromyogram (EMG), which involves placing two electrodes under one eye to measure the electrical activity generated by the movement of the muscle. The startle response is then analysed using either onset latency (typically 20-50ms), peak latency or amplitude, which is the measure used by most (Grillon & Bass, 2002).

As there is strong evidence that the startle response is reliant upon the function of the amygdala, it is a useful technique to investigate amygdala activation in anxiety. Based upon neuro-cognitive models of anxiety, it is predicted that those who are anxious will show greater amygdala activity and, therefore, startle response in concern-relevant situations compared to those with low levels of anxiety. Further, there is scope to investigate the association between cognitive biases and amygdala functioning. The startle technique could plausibly be paired with cognitive tasks, to act as a behavioural measure of amygdala functioning in response to contrasting cognitive styles. This is particularly relevant, as it has recently been argued that the startle response can be modified by cognitive and affective variables (Larsen, Norton, Walker & Stein, 2002).

As the next section will indicate, there are several methods available to investigate the association between anxiety, cognitive biases and the startle response.

6.2 Paradigms utilising the startle response

Affective modulation of the startle response involves presenting positive, neutral and negative slides or scenes with intermittent startle probes. It has reliably been found that unpleasant stimuli provoke a larger startle reflex than neutral stimuli (Lang, Bradley & Cuthbert, 1990; Ohman & Mineka, 2001). This pattern of findings can be explained by biphasic theory, which suggests that aversive cues activate the motivation system. More specifically, unpleasant cues are thought to activate the aversive motivational system which results in escape and defence behaviours, the startle response is thought to be a defensive reflex associated to this aversive motivational system (Bradley, Cuthbert & Lang, 1999). Using the affective modulation paradigm, it has been found that anxious individuals show greater startle reactivity in response to disorder-specific fear stimuli (eg. spiders) than to non- disorder specific stimuli (Lang, 1995). The benefit of the affective startle modulation paradigm is that it has the potential to assess emotional reactivity to contexts and cues that are of relevance to the aetiology and maintenance of psychopathology (Grillon & Bass, 2003). In addition, as cognitive theory suggests, affect is related to cognition. Thus, those with social phobia who are more likely to find social cues threatening would be predicted to have a greater affective startle response to these stimuli.

Fear-potentiated startle is another technique used to investigate startle reactivity in response to differing affective states. Fear-potentiated startle has been most extensively demonstrated using classical conditioning paradigms in which a neutral stimulus/context becomes associated with the delivery of an aversive unconditioned stimulus (electric shock). It has been reliably found in humans that startle amplitude is greater in the presence of the conditioned stimulus, consistent with the suggestion that fear-conditioning has resulted in greater potentiated fear responses to the conditioned stimulus/context (CS+) than a control stimulus (CS-) (Grillon & Davis, 1997). Therefore, the startle response is greater in humans when they experience fearful situations. The difficulty with this paradigm is that it relies upon the use of aversive stimuli, which causes ethical implications when trying to apply it to a psychiatric population. The benefit of fear-potentiated startle is that it has provided evidence of the role of fear in modulating the startle response.

The *light-dark startle* is another example of fear-potentiated startle, it is a technique that involves individuals sitting in light and dark conditions whilst exposed to startle probes, it is thought that darkness provokes a fear response in humans. It has been found that this technique can facilitate the startle response, with darkness inducing a fear response and potentiating amygdala activity and thus greater startle responsivity (Grillon, Merikangas, Dierker, Snidman, Arriaga, Kagan et al., 1999; Grillon, Pellowski, Merikangas, & Davis, 1997). Again, neuro-cognitive theory would predict that trait anxious individuals would be particularly sensitive to manipulations of contextual fear and would therefore show greater amygdala activity/startle modulation to darkness than those low in anxiety. Indeed, it has been found that Vietnam war veterans with post-traumatic stress disorder have a larger startle response to darkness than non-traumatised individuals (Grillon, Morgan, Davis & Southwick, 1998).

The startle response is associated to the activation of a sub-cortical fear network thought to be involved in the appraisal of fear and modulating behavioural responses (eg. fight or flight). As such, it could prove to be a useful index of amygdala-driven appraisal biases proposed to characterise anxiety disorders such as social phobia.

6.3 Appraisal biases and the startle response

As previously described, the affect modulation paradigm utilises differently valenced stimuli (i.e. pictures) to induce varying affect states in participants. As neuroimaging data suggests, socially anxious individuals are likely to interpret ambiguous social cues as threatening. With the startle paradigm it is predicted that threat appraisal will induce negative affect and therefore a larger startle response will be expected compared to the non-anxious group. The affect modulation paradigm has been the most widely used in the study of social phobia.

Blumenthal, Chapman & Muse (1995) found, using an affect modulation paradigm, that a social encounter potentiates the startle response in those with high levels of social anxiety. These findings indicate an initial appraisal bias towards threat in response to a social situation leads to an augmented startle response, indicating greater activation of the amygdala. Cornwell, Johnson, Berardi, & Grillon (2006) created a virtual reality environment, where the participant felt as though they were stood centre stage in-front of an audience about to give a speech or count backwards. Those high in trait social anxiety were found to have greater startle reactivity in response to conditions of social-evaluative threat (ie. anticipation of giving a speech). Perception of social-evaluative threat is thought to be generated by biased appraisal of social cues towards threat. Similarly, Panayiotou & Vrana, (1998) asked individuals high and low in social anxiety to undertake digit recall tasks under conditions of evaluative or non-evaluative instructions in self-focused and non self-focused attention conditions. The authors found that self-focus conditions led to greater startle reactivity in the high socially anxious group consistent with proposals from Clark & McManus that emphasise self-focus and the construction of self images taken from the observer perspective as a primary source of threat in socially anxious individuals. Taken together this evidence suggests that the startle response is potentiated in the context of feared social situations, indicating that threat appraisal may be associated to activation of the amygdala.

7. Conclusions

Many theories regarding the aetiology and maintenance of social phobia have been proposed. Cognitive theorists propose that social phobia is maintained by an individual's appraisal bias towards threat when confronted with a social situation. This appraisal bias is thought to be the precipitating factor for a number of characteristics of social phobia such as: self-focused attention; greater levels of attention to external social cues; perceived negative evaluation from others and negative perception of self (Rapee and Heimberg, 1997). In addition, neuroscientists propose that the amygdala is central to threat appraisal in anxiety (Ohman & Mineka, 2001), with its role being to detect and avoid danger (Amaral, 2002). What has become evident to researchers is that neurobiological theories, suggesting that the amygdala is the central structure involved in threat appraisal, map onto contemporary cognitive theories of anxiety. Cognitive theorists argue that the valence evaluation system is critical for pre-attentive threat appraisal (Mogg & Bradley, 1998), while neurobiological evidence implicates the amygdala. Neurocognitive theory draws together neurobiological and cognitive theory to suggest that the amygdala is the neural structure at the centre of a fear network, which is responsible for threat appraisal and as such, predicts that greater activation of the amygdala will be associated to a bias towards negative appraisal of mild threat.

Evidence from cognitive paradigms (Amin et al., 1998; Amir et al., 2005; Vonken et al., 2003; Wenzel et al., 2005) and facial expression research (Yoon & Zinberg, 2007; Gilboa-Schechtman et al., 2005), suggest that a negative appraisal bias towards ambiguous social threat cues exists with individuals high in social anxiety.

Lesion data suggests that individuals with impaired amygdala function have difficulties in threat appraisal and recognition (Broks et al., 1998; Adolphs et al., 1998). Neuroimaging studies have revealed that the pre-attentive appraisal of fear is related to specific activation of the amygdala, suggesting that it is the neural structure involved in the appraisal of threat. Considering the predicted association between appraisal bias and amygdala activation, it has been found, using neuroimaging techniques, that processing of ambiguous information is related to increased activity of the amygdala indicating an appraisal bias towards threat (Birbaumer et al.,1998; Somerville et al., 2004). Startle probe paradigms have also been employed to investigate the relationship between amygdala activation and appraisal biases and benefit from relative cost-effectiveness, limited intrusiveness, while providing a robust measure of fear behaviours potentiated by the sub-cortical fear network.

Bishop (2007) suggested that the strength of amygdala activation in response to threat cues (i.e. emotional faces in social anxiety) will influence the level of explicit threat appraisal. Evidence for this approach is limited, therefore further research is required to reliably demonstrate the association between amygdala function and the threat appraisal biases observed in social anxiety. It would be informative to further investigate the utility of startle paradigms in assessing the predictions of neurocognitive theory. While findings from initial studies are promising, for example potentiated startle in response to increased social evaluation and during self-focussed attention, it remains necessary to investigate to what extent fear networks are potentiated in response to disorder - specific social cues (e.g. facial expressions) in social anxiety.

References

Adolphs, R., Tranel, D., Damasio, A.R. (1998). The human amygdala in social judgment. *Nature 393*(6684), 470-474.

Amaral, D. (2002). The primate amygdala and the neurobiology of social behavior: implications for understanding social anxiety . . *Biological Psychiatry, 51*(1), 11-17.

Amin, N., Foa, E. B., & Coles, M. E. (1998). Negative interpretation bias in social phobia. *Behaviour Research and Therapy, 36*(10), 945-957.

Amir, N., Beard, C., Bower, E. . (2005). Interpretation Bias and Social Anxiety *Cognitive Therapy and Research, 29*(4), 433-443.

Amir, N., Elias, J., Klumppa, H., Przeworski, A. (2003). Attentional bias to threat in social phobia: facilitated processing of threat or difficulty disengaging attention from threat? . *Behaviour Research and Therapy, 41*(11), 1325-1335

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders (3rd ed.).* Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th ed.).* Washington, DC: American Psychiatric Association.

Beck, A. T. (1976). *Cognitive Therapy and the Emotional Disorders*. New York: International Universities Press.

Beck, A. T., & Clark, D. A. (1997). An information processing model of anxiety: Automatic and strategic processes. *Behaviour Research and Therapy, 35*(1), 49-58.

Beck, A. T., Emery, G., & Greenberg, R.L. . (1985). *Anxiety Disorders and Phobias: A cognitive perspective*. New York: Basic Books.

Birbaumer, N., Grodd, W., Diedrich, O., Klose, U., Erb, M., Lotze, M., Schneider, F., Weiss, U., Flor, H. (1998). fMRI reveals amygdala activation to human faces in social phobics. *NeuroReport, 9*(6), 1223-1226.

Bishop, S. J. (2007). Neurocognitive mechanisms of anxiety: an integrative account. *Trends in Cognitive Sciences, 11*(7), 307-316.

Blumenthal, T. D., Chapman, J. G., & Muse, K. B. (1995). Effects of Social Anxiety, Attention, and Extroversion on the Acoustic Startle Eyeblink Response. *Personality and Individual Differences, 19*(6), 797-807.

Broks, P., Young, A.W., Maratos, E.J., Coffey, P.J., Calder, A.J., Isaac, C.L., Mayes, A.R., Hodges, J.R., Montaldi, D., Cezayirli, E., Roberts, R., Hadley, D. (1998). Face processing impairments after encephalitis: amygdala damage and recognition of fear *Neuropsychologia, 36*(1), 59-70

Carlsson, K., Petersson, K. M., Lundqvist, D., Karlsson, A., Ingvar, M., & Ohman, A. (2004). Fear and the amygdala: Manipulation of awareness generates differential cerebral responses to phobic and fear-relevant (but nonfeared) stimuli. *Emotion, 4*(4), 340-353.

Clark, D. M., McManus, F. (2002). Information processing in social phobia. *Biological Psychiatry, 51*(1), 92-100.

Clark, D. M., Teasdale, J.D. (1982). Diurnal variation in clinical depression and accessibility of memories of positive and negative experiences. *Journal of Abnormal Psychology, 91*(2), 87-95.

Clark, D. M., Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, Liebowitz, M.R., Hope, D.A., Schneier, F.R. (Ed.), *Social Phobia: Diagnosis, Assessment and Treatment* (pp. 69-93). New York: Guilford Press.

Cornwell, B. R., Johnson, L., Berardi, L., & Grillon, C. (2006). Anticipation of public speaking in virtual reality reveals a relationship between trait social anxiety and startle reactivity. *Biological Psychiatry, 59*(7), 664-666.

Davidson, R. (2002). Anxiety and affective style: role of prefrontal cortex and amygdala *Biological Psychiatry, 51*(1), 68 - 80.

Eysenck, M. W., Mogg, K., May, J., Richards, A., Mathews, A. (1991). Bias in interpretation of ambiguous sentences related to threat in anxiety. *Journal of Abnormal Psychology, 100*(2), 144-150.

Fehm, L., Pelissolo, A., Furmark, T., and Wittchen, H. (2005). Size and burden of social phobia in Europe. *European Neuropsychopharmacology, 15*(4), 453-462

Funayama, E. S., Grillon, E.S., Davis, M., Phelps, E.A. (2001). A double dissociation in the affective modulation of startle in humans: Effects of unilateral temporal lobectomy *Journal of Cognitive Neuroscience, 13*, 721-729.

Furmark. (2002). Social phobia: overview of community studies. *Acta Psychiatrica Scandinavica, 105*(2), 84-93.

Gilboa-Schechtman, E., Presburger, G., Marom, S., & Hermesh, H. (2005). The effects of social anxiety and depression on the evaluation of facial crowds. *Behaviour Research and Therapy, 43*(4), 467-474.

Greenberger, D., & Padesky, C.A. . (1995). *Mind Over Mood*. New York: Guilford Press.

Grillon, C., Baas. (2002). Comments on the use of the startle reflex in psychopharmacological challenges: impact of baseline startle on measurement of fear-potentiated startle *Psychopharmacology, 164*(2), 236-238.

Grillon, C., & Davis, M. (1997). Effects of stress and shock anticipation on prepulse inhibition of the startle reflex. *Psychophysiology, 34*(5), 511-517.

Grillon, C., Dierker, L., & Merikangas, K. R. (1998). Fear-potentiated startle in adolescent offspring of parents with anxiety disorders. *Biological Psychiatry, 44*(10), 990-997.

Grillon, C., Merikangas, K. R., Dierker, L., Snidman, N., Arriaga, R. I., Kagan, J., et al. (1999). Startle potentiation by threat of aversive stimuli and darkness in adolescents: a multi-site study. *International Journal of Psychophysiology, 32*(1), 63-73.

Grillon, C., Pellowski, M., Merikangas, K. R., & Davis, M. (1997). Darkness facilitates the acoustic startle reflex in humans. *Biological Psychiatry, 42*(6), 453-460.

Hirsch, C. A., Mathews, A. (2000). Impaired positive inferential bias in social phobia. *Journal of Abnormal Psychology, 109*(4), 705-712.

Jarrard, L. E. (1993). On the role of the hippocampus in learning and memory in the rat. *Behavioral and Neural Biology, 60*(1), 9-26.

Joormann, J., Gotlib, I.H. (2006). Is This Happiness I See? Biases in the Identification of Emotional Facial Expressions in Depression and Social Phobia. *Journal of Abnormal Psychology, 115*(4), 705-714.

Judd, L. L. (1994). Social phobia: A clinical overview. *Journal of Clinical Psychiatry, 55*(6), 5-9.

Lang, P. J. (1995). The emotion probe. Studies of motivation and attention. *American Psychologist, 50*(5), 372-385.

Lang, P. J., Bradley, M.M., Cuthbert, B.N. (1990). Emotion, attention, and the startle reflex.. *Psychological Review, 97*(3), 377-395.

Larsen, D. K., Norton, G.R., Walker, J.R., Stein, M.B. (2002). Analysis of startle responses in patients with panic disorder and social phobia *Cognitive Behaviour Therapy, 31*(4), 156-169.

Leahy, R. L., Holland, S.J.. (2000). *Treatment Plans and Interventions for Depression and Anxiety Disorders*. New York: Guilford Press.

LeDoux, J. E. (1995). Emotion: Clues from the brain. *Annual Review of Psychology, 46*, 209-235.

MacLeod, C., Mathews, A., Tata, P. (1986). Attentional bias in emotional disorders. *Journal of Abnormal Psychology, 95*(1), 15-20.

Maren, S., & Quirk, G. J. (2004). Neuronal signalling of fear memory. *Nature Reviews Neuroscience, 5*(11), 844-852.

Mathews, A., & Mackintosh, B. (1998). A cognitive model of selective processing in anxiety. *Cognitive Therapy and Research, 22*(6), 539-560.

Mogg, K., & Bradley, B. P. (2002). Selective orienting of attention to masked threat faces in social anxiety. *Behaviour Research and Therapy, 40*(12), 1403-1414.

Mogg, K., Bradley, B.P. . (1998). A cognitive-motivational analysis of anxiety *Behaviour Research and Therapy 36*(9), 809-848.

Mogg, K., Mathews, A., Weinman, J. (1987). Memory bias in clinical anxiety. *Journal of Abnormal Psychology, 96*(2), 94-98.

Mogg, K., Philippot, P., & Bradley, B. P. (2004). Selective attention to angry faces in clinical social phobia. *Journal of Abnormal Psychology, 113*(1), 160-165.

Musa, C., Lepine, J. P., Clark, D. M., Mansell, W., & Ehlers, A. (2003). Selective attention in social phobia and the moderating effect of a concurrent depressive disorder. *Behaviour Research and Therapy, 41*(9), 1043-1054.

Ohman, A., Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning *Psychological Review, 108*(3), 483-522.

Panayiotou, G., Vrana, S.R. (1998). Effect of self-focused attention on the startle reflex, heart rate, and memory performance among socially anxious and nonanxious individuals *Psychophysiology, 35*, 328-336

Pissiota, A., Frans, O., Michelgard, A., Appel, L., Langstrom, B., Flaten, M. A., et al. (2003). Amygdala and anterior cingulate cortex activation during affective startle modulation: a PET study of fear. *European Journal of Neuroscience, 18*(5), 1325-1331.

Rapee, R. M. (1995). Descriptive psychopathology of social phobia. In R. G. Heimberg, Liebowitz, M.R., Hope, D.A., Schneier, F.R. (Ed.), *Social Phobia: Diagnosis, Assessment and Treatment* (pp. 41-68). New York: Guilford Press.

Rapee, R. M., Heimberg, R.G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741-756

Richards, A., French, C.C., Calder, A.J., Webb, B., Fox, R., Young, A.W. (2002). Anxiety-related bias in the classification of emotionally ambiguous facial expressions. *Emotion, 2*(3), 273-287.

Sarter, M., Berntson, G.G., Cacioppo, J.T. (1996). Brain imaging and cognitive neuroscience. *American Psychologist, 51*(1), 13-21.

Schneier, F. R., Johnson, J., Hornig, C.D., Liebowitz, M.R., Weissman, M.M.. (1992). Social phobia. Comorbidity and morbidity in an epidemiologic sample. *Archives of General Psychiatry, 49*(4), 282-288.

Shi, C., Davis, M. (2001). Visual pathways involved in fear conditioning measured with fear-potentiated startle: Behavioral and anatomic studies. *The Journal of Neuroscience, 21*(24), 9844–9855.

Somerville, L. H., Kim, H., Johnstone, T., Alexander, A. L., & Whalen, P. J. (2004). Human amygdala responses during presentation of happy and neutral faces: Correlations with state anxiety. *Biological Psychiatry, 55*(9), 897-903.

Taylor, C. T., & Alden, L. E. (2005). Social interpretation bias and generalized social phobia: the influence of developmental experiences. *Behaviour Research and Therapy, 43*(6), 759-777.

Vassilopoulos, S. P. (2005). Social anxiety and the vigilance-avoidance pattern of attentional processing. *Behavioural and Cognitive Psychotherapy, 33*(1), 13-24.

Vassilopoulos, S. P. (2006). Interpretation and judgmental biases in socially anxious and nonanxious individuals. *Behavioural and Cognitive Psychotherapy, 34*(2), 243-254.

Voncken, M. J., Bogels, S. M., & de Vries, K. (2003). Interpretation and judgmental biases in social phobia. *Behaviour Research and Therapy, 41*(12), 1481-1488.

Wells, A. (1997). *Cognitive therapy of anxiety disorders: A practical manual and conceptual guide.* Chichester: John Wiley & Sons.

Wenzel, A., Finstrom, N., Jordan, J., & Brendle, J. R. (2005). Memory and interpretation of visual representations of threat in socially anxious and nonanxious individuals. *Behaviour Research and Therapy, 43*(8), 1029-1044.

Whalen, P. J., Rauch, S.L., Etcoff, N.L., McInerney, S.C., Lee, M.B., Jenike, M.A. . (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge *The Journal of Neuroscience, 18*(1), 411-418.

Williams, J. M. G., Watts, F.N., MacLeod, C, Mathews, S.A. (1988). *Cognitive Therapy and Emotional Disorders*. New York: John Wiley & Sons.

Wilson, J. K., & Rapee, R. M. (2005). Interpretative biases in social phobia: Content specificity and the effects of depression. *Cognitive Therapy and Research, 29*(3), 315-331.

Winton, E. C., Clark, D.M., Edelman, R.J. (1995). Social anxiety, fear of negative evaluation and the detection of negative emotion in others *Behaviour Research and Therapy, 33*(2), 193-196

Yoon, K. L., & Zinbarg, R. E. (2007). Threat is in the eye of the beholder: Social anxiety and the interpretation of ambiguous facial expressions. *Behaviour Research and Therapy, 45*(4), 839-847.

EMPIRICAL PAPER

Effect of Darkness, Neutral and Fear Faces on the Startle Reflex in Social Anxiety

Rebecca Lee

Prepared for submission to Behaviour Research & Therapy- see Appendix B for instructions to authors.

Effect of Darkness, Neutral and Fear Faces on the Startle Reflex in Social Anxiety

Abstract

Recent cognitive models of social phobia propose that individuals with social anxiety will have biases in threat appraisal when processing social information. Recent efforts to integrate cognitive theory of threat processing to functional neuroanatomy have implicated the amygdala. The recent neurocognitive account of anxiety suggests that individuals with social phobia will make biased appraisals of social cues due to a hypersensitivity of the amygdala.

The present study investigated predictions from neurocognitive theory, which suggest social cues potentiate neural systems involved in fear and anxiety more than less specific fear stimuli. Sub-cortical appraisal of social and non- social cues in high and low social anxiety (HSA vs. LSA) was investigated by measuring startle magnitude and latency to fear and neutral faces (social cues) and light and dark patches (non-social cues).

The present study found startle potentiation to the dark condition in comparison to light and faces, in both the HSA and LSA groups. No individual differences were found in the processing of social cues, thus predictions from the neurocognitive approach were not supported. In comparison to previous findings, it was concluded that faces may not have appropriate contextual relevance to potentiate sub-cortical threat appraisal.

1. Introduction

Social phobia is an anxiety disorder characterised as a marked and persistent fear of social situations that could cause embarrassment (DSM-III; American Psychiatric Association, 1980). The disorder is prevalent, with findings suggesting that between 7% and 13% of the population meet the criteria for diagnosis in a lifetime (Furmark, 2002). The consequences of experiencing social phobia can be pervasive, with a large proportion of people experiencing subsequent co-morbid difficulties such as depression and substance misuse (Schneier, Johnson, Hornig, Liebowitz, & Weissman, 1992). It has also been indicated that individuals with social phobia may have difficulty in fulfilling their potential academically or occupationally (Judd, 1994). Social phobia is therefore a significant issue for the individuals that experience these problems, mental health services and wider society. A better understanding of the factors involved in the aetiology and maintenance of this disorder would help generate effective treatment protocols and inform service provision.

Cognitive theorists (e.g. Clark & McManus, 2002; Rapee & Heimberg, 1997) suggest that social phobia is characterised by a range of cognitive biases (i.e. biases in information processing) that maladaptively distort socially anxious individuals appraisal, attention and memory of feared social situations. It is proposed that these cognitive biases potentiate perceptions of threat and danger when an anxious individual enters a feared social situation and prevents them from disconfirming pre-existing social evaluative concerns. Cognitive models of social anxiety propose that when confronted with a social situation, a person with social phobia will appraise the situation as threatening and be vigilant for both external threatening social cues (e.g. angry faces) and internal cues that signal anxious apprehension (e.g. self-focus on physiological symptoms such as sweating, changes in heart-rate/breathing). Specifically threat appraisal is proposed to trigger increased allocation of attention to external and internal cues that further exacerbate the individual's anxiety. General cognitive models of threat processing in anxiety explicitly link biases in threat appraisal with higher level biases such as selective attention and interpretation of ambiguity.

Recent cognitive theories suggest that the bias towards appraisal of threat in individuals with social phobia is pre-attentive, therefore outside conscious awareness and an individual's control. Cognitive theorists argue that this pre-attentive appraisal bias is the principle vulnerability risk factor for anxiety (Mogg & Bradley, 1998).

In recent years efforts have been made to integrate cognitive accounts of threat processing biases (e.g. appraisal), with an increasing understanding of the functional neuroanatomy involved in normal fear and pathological anxiety. Neuroscientists have suggested that the amygdala is the neural structure responsible for the appraisal of threat (LeDoux, 1995; Ohman & Mineka, 2001)., This follows much animal research reporting that the amygdala is the fundamental structure in the experience of fear in animals (Amaral, 2002; Davidson, 2002; Maren & Quirk, 2004). One of the latest theories to be proposed, that has implications for understanding the aetiology of social phobia, is a general neuro-cognitive model of anxiety (Bishop, 2007). Bishop explicitly links hypersensitivity of the amygdala and related structures with biases in information processing in anxiety. When applied to social anxiety, Bishop would predict that socially anxious individuals' negative biases in perception and attention result from potentiation in the amygdala.

It has been found that high socially anxious groups have been found to interpret ambiguous, positive and negative social scenarios with a negative bias (Amin, Foa & Coles, 1998; Vonken, Boegels, & deVries, 2003; Vassilpoulos; 2006; Wilson & Rapee, 2005). Though consistent with predictions from cognitive models, it is unclear whether written social scenarios can accurately characterise the contextual social cues that feature in feared social situations. (e.g. facial expressions). Studies utilising contextually relevant faces as stimuli have revealed that high socially anxious individuals appraise both negative and neutral facial expressions as negative (Winton, Clark & Edelmann, 1995) and are better able to detect fear in morphed ambiguous faces (i.e. two emotional facial expressions morphed together; Richards, Calder, Webb, Fox, & Young, 2002). In addition, when asked to make an appraisal of a series of differently valenced facial expressions, high social anxiety groups have been found to be quicker to classify angry faces (Joormann & Gotlib, 2006). Taken together, this data supports predictions from cognitive models that suggest social anxiety is characterised by maladaptive biases in stimulus appraisal.

As noted earlier neurobiological theorists suggest that the amygdala is responsible for threat appraisal. In humans, lesions of the amygdala lead to impairments in the recognition of facial expressions depicting fear and attenuated fear responsivity to unfamiliar people (Brok, Young, Maratos, Coffey, Calder, Isaac et al., 1998; Adolphs, Tranel & Damasio, 1998). Neuroimaging studies have found that fear or phobia related stimuli presented outside of conscious awareness (using visual masking techniques) generate increased amygdala activation (Whalen, Rauch, Etcoff, McInerney, Lee & Jenike, 1998; Carlsson, Petersson, Lundqvist, Karlsson, Ingvar, & Ohman, 2004). This is consistent with the view that threat-related stimuli enjoy prioritised pre-attentive processing. Furthermore, neuroimaging data has revealed greater amygdala activity in high social anxiety groups in response to ambiguous faces comparable to the activity observed in response to aversive faces (Birbaumer, Grodd, Diedrich, Klose, Erb, Lotze et al., 1998; Somerville, Kim, Johnstone, Alexander & Whalen, 2004).

Thus, taken together there is evidence to suggest that appraisal biases exist in social phobia, that the amygdala appears to be involved in the appraisal of threat, and that the amygdala is hyperactive when socially anxious individuals process social cues. Futhermore, this evidence supports the proposal that amygdala dysfunction in anxiety might underlie the negative appraisal of social cues observed in cognitive studies of social anxiety, and enhanced fearfulness in social situations.

While fMRI studies have linked amygdala hyperactivity with processing negative information, the extent to which potentiated amygdala activity increases the subjective experiences of fear and the co-ordination of fear behaviours (e.g. activation of the flight-flight response) remains to be clarified. As such researchers have supplemented fMRI paradigms with methods that examine behavioural components of fear-related amygdala hyperactivity.

The startle response is a cross-species primitive reflex consisting of a rapid sequential muscle contraction that serves a protective function, reducing the risk of organ injury, and acting as a behavioural interrupt thereby enabling an organism to deal with possible threat (Graham, 1979). In humans, rapid eye closure is one of the most reliable components of the defensive cascade that constitutes the startle (Landis & Hunt, 1939; cited from Grillon & Baas, 2003). Startle reflexes (such as eyeblink) are elicited when individuals are exposed to aversive (brief and intense) auditory, visual, or tactile stimuli. In human studies, brief bursts (up to 50 ms) of white noise with high intensity (e.g. 90 – 110 dB) are often used to elicit startle responses which are then typically quantified in terms of response amplitude and peak latency.

Given the involvement of the amygdala in emotion processing and its role in the modulation of defensive responses such as startle, research has increasingly used startle magnitude to index sub-cortical stimulus appraisal processes. Central to such *affective startle modulation paradigms* is the notion that when the sub-cortical fear-network is activated (i.e. when unpleasant or threatening information has been identified) defensive startle responses are primed (potentiated). In contrast, when the fear-network is less active, startle reflexes are inhibited (e.g. Lang, Bradley, & Cuthbert, 1990). Though limited research has employed the startle paradigm in anxiety, two studies have found that high social anxiety groups show greater startle potentiation, to a social situation where they are anticipating social-evaluative threat and under conditions of self-focused attention (Cornwell, Johnson, Berardi & Grillon, 2006; Panayiotou & Vrana, 1998).

Cognitive theory predicts that appraisal biases are a risk factor for social phobia (Mogg & Bradley, 1998). Given that appraisal biases can be considered to result from amygdala hyperactivity, it has been proposed that a generalized amygdala potentiation (in the absence of disorder/concern-relevant cues) could be a plausible neurobiological risk factor for a range of anxiety disorders. In a study of adolescents at high and low risk of developing an anxiety disorder, Grillon utilised a technique called light-dark startle to investigate generalized startle potentiation in different participant groups. The technique involves delivering a startle probe whilst the participant is in light or darkness in the laboratory. Results suggest that darkness facilitates the startle response in college students (Grillon, Pellowski, Merikangas & Davis, 1997) and that this response is greater still in a war veteran group with a diagnosis of post-traumatic stress disorder (PTSD) in contrast to non-PTSD comparison groups (Grillon, Morgan, Davis & Southwick, 1998). While these results suggest that generalized amygdala potentiation (as measured by light-dark startle) is evident in a range of anxiety disorders, to date no study has examined this in social anxiety.

Further investigation is required to further clarify the extent to which neurobiological structures involved in fear responding are activated in response to disorder specific cues in social anxiety, as proposed by neuro-cognitive models. Startle studies thus far have investigated potentiation in the fear-network in anticipation of social evaluation and during self-focus, but no study has examined whether cues encountered within a feared social situation potentiate neural systems involved in threat processing in social anxious individuals.

1.1 The current study

The aims of the present study were two-fold: Firstly to examine whether cues that are considered to be of relevance to the concerns of individuals with social anxiety (i.e. faces) potentiate neural systems involved in fear and anxiety (i.e. the amygdala). Secondly, to examine whether more generalized amygdala hyperrexitability (as measured by light-dark) is characteristic of social anxiety.

In the present study University students, split into high and low socially anxious groups completed a modified fear-potentiated startle task. High and low socially anxious individuals were administered acoustic startle probes during the presentation of social cues (negative fear and neutral facial expressions) a non-social grey patch and in darkness. Fear faces were used in preference to other negative expressions (e.g. angry expressions), as imaging studies have reliably shown evidence of amygdala involvement in the processing fear faces.

Given that anxiety is proposed to be characterised by inappropriate exaggeration of normal fear responses (Barlow, 1988) and a tendency to evaluate stimuli as threatening (Mathews & Mackintosh, 1998; Mogg & Bradley, 1998; Bishop, 2007) it is predicted that:

1. High socially anxious individuals will demonstrate greater startle potentiation (as indexed by increased startle magnitude and reduced startle latency) in response to social cues (particularly negative fear expressions) relative to non-social cues (light patch) compared low anxious individuals.
2. High socially anxious individuals will be characterised by elevated levels of generalized amygdala reactivity to contextual manipulations of non-specific threat (i.e. elevated startle in dark relative to light conditions) compared to low anxious individual.

2. Method

2.1 Design

The study employed a mixed design, with between and within factor variables. The between factors variable was group, low versus high social anxiety. The within factors variable was stimulus with four levels: light, dark, neutral faces and fear faces. The dependent variables were startle response magnitude (peak amplitude) and startle latency.

2.2 Participants

One-hundred and forty-seven undergraduate students were screened using the brief version of the Fear of Negative Evaluation Scale (18 male, 129 female; mean age 20.4; mean sFNE 35.9). Those individuals scoring below thirty or above forty were deemed eligible to participate, which corresponds to the 30th and 65th percentiles (Stopa & Clark, 2001). This method of screening is consistent with methods used previously (Garner, Mogg & Bradley, 2006), it provides an enriched sample, therefore excluding individuals that lie in the mid-range for social anxiety. This enabled low and high anxiety groups to be formed using a median split based on the extended version of the FNE, which was completed by participants at the test session. This method of splitting groups was favoured to minimise the quantity of participant exclusions required following the test session. Of those screened, one-hundred were deemed eligible (16 male, 84 female; mean age 20.5; mean sFNE 36.6), thirty-six of whom agreed to participate in the study (7 male, 29 female; mean age 21.7; mean sFNE 34.8). Due to difficulties in participant recruitment in a previous design, three participants who had previously taken part in the study without undertaking the screening measure met the criteria for inclusion in the current study (i.e. scoring above the 65th percentile or below the 30th percentile). Thus the total enriched sample consisted of thirty-nine participants (9 male, 30 female; mean age 21.5).

Participants were offered two participation credits for undertaking the screening questionnaire and eight participation credits, £10 and travel expenses for attending the subsequent test session. The research credit scheme operates within the University's School of Psychology. The aim is that research participation will enhance undergraduates learning experience (students are asked to accrue seventy-five research credits in their first year which provides 1.25% of their first year mark or forty-eight credits in their second year which is 1.5% of the year mark).

2.3 Materials

2.3.1. Experimental task

Conductance sites for the startle response (electromyography; EMG), were prepared using a surgical spirit and water solution and abrasive pads specifically designed to remove non-conductive skin cells. For EMG recording, two 4-millimetre silver-silver chloride (Ag-AgCl) re-usable electrodes were filled with conductive electrode gel and attached to the skin using double-sided adhesive collars. Psychophysiological data was acquired using a Biopac MP100 System in combination with AcqKnowledge 3.8.1 software.

Experimental stimuli consisted of light and dark patches and pictures of faces expressing either fear or neutral facial expressions. Neutral and fearful facial expressions from four male and four female models were selected from the NimStim face set (MacArthur Foundation Research Network on Early Experience and Brain Development, 2002)[1]. Images were resized to 506 x 650 pixels, mounted on a grey background (RGB values = 128, 128,128 respectively) and presented in greyscale, see Figure 3. The grey patch had RGB values = 128, 128,128 respectively and the dark patch had RGB values = 0,0,0. Patches were prepared using Microsoft Paint (2001, Microsoft Cooperation).

Visual stimuli, startle probes and Biopac event triggers were presented using Inquisit version II software (Millisecond software, 2002) within Windows

[1] Development of the MacBrain Face Stimulus Set was supported by the MacArthur Foundation Research Network; please contact Nim Tottenham (tott0006@tc.umn.edu) for further information.

XP on a Pentium III 1.2 GHz PC. Visual stimuli were presented to the participant through head-mounted eMagin Z800 VGA goggles. EMG data was acquired using AcqKnowledge 3.8.1 software (Biopac Systems, 2004) on a Pentium III 3.0 GHz PC. Participants completed all tasks in a dimly lit testing cubicle.

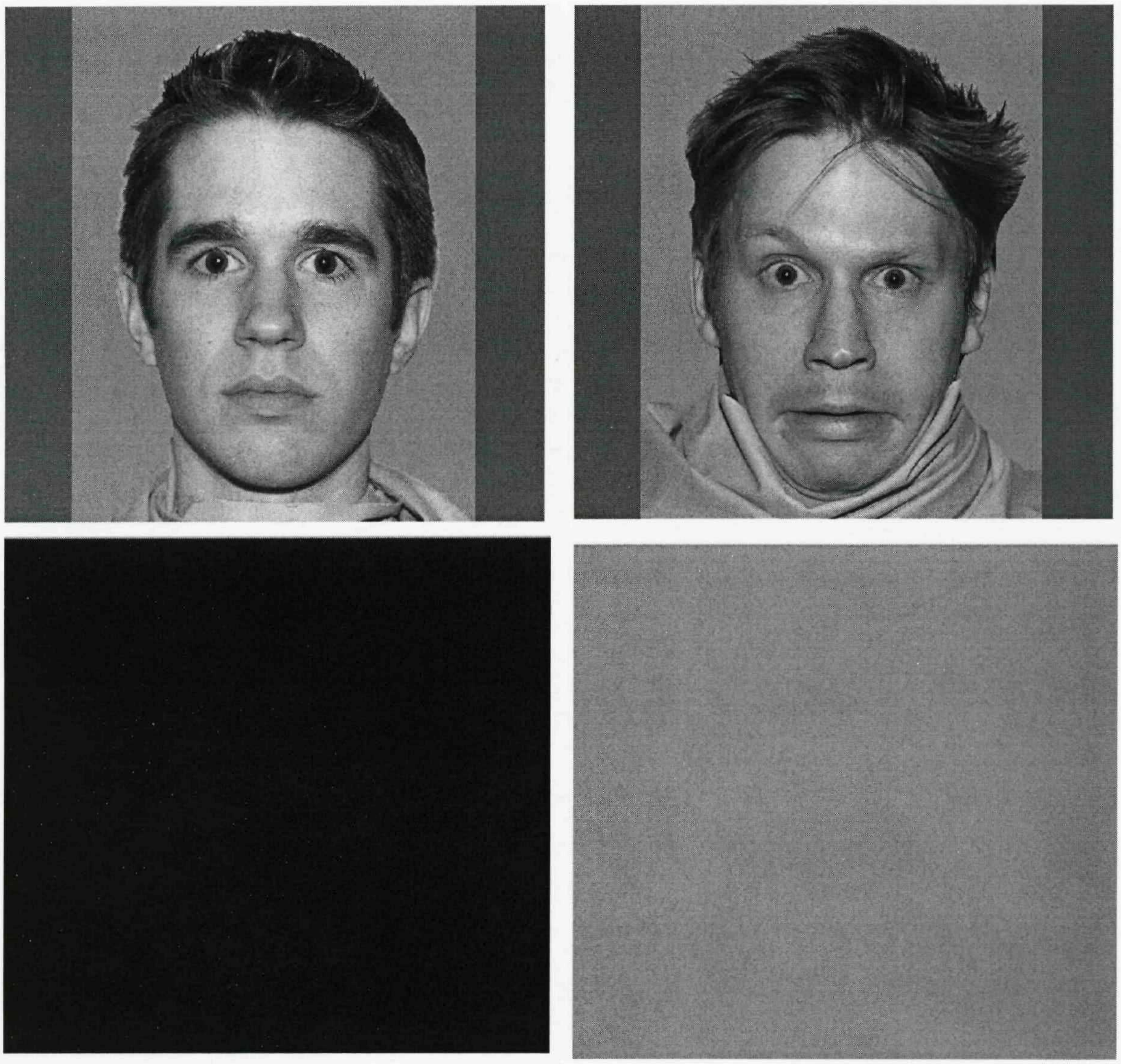

Figure 3. Sample social and non-social stimuli.

The acoustic startle probe was a 50 ms burst of 96 dB white noise with near instantaneous rise/fall time. Startle probes were produced using a white noise generator, presented through DR-3A head-phones (Sony, Japan) and were calibrated prior to each session using a 1405C sound level meter (Dawe Instruments Limited, UK). Eye blink startle response was measured electro-myographically from the orbicularis oculi, using Ag/AgCl electrodes and electrolyte (Biopac System, 2004). Electro-myographic (EMG) signal was sampled at 1000 Hz throughout the task using AcqKnowledge 3.8.1 software (Biopac Systems, 2004) on a Pentium III 3.0 GHz PC.

2.3.2 Screening measure

The *brief fear of negative evaluation scale* (BFNE; Leary, 1983) is a 12-item short-form version of the full FNE. In contrast to the FNE, the BFNE requires responses on a 5-point Likert scale from 'not at all characteristic of me' to 'extremely characteristic of me'. BFNES scores range from 12-60. It has been found to have good reliability and validity (Duke, Krishnan, Faith, & Storch, 2006) and has been used as a screening tool in social anxiety research (Brown & Stopa, 2007).

2.3.3. Questionnaire measures

The *fear of negative evaluation scale* (FNE; Watson & Friend, 1969) is a 30-item self-report scale requiring yes/no responses, assessing concerns regarding negative evaluation from others.

The *Beck depression inventory-II* (BDI-II; Beck and Steer, 1987) is a 21-item self-report measure of depression used widely within psychological research. It assesses cognitive-affective and somatic symptoms of depression over a two-week period using a multiple choice approach, scores range between 0 and 63. The measure has been found to have good internal consistency and concurrent validity within a college student population (Storch, Roberti, & Roth, 2004).

The *Spielberger state-trait anxiety inventory* (STAI; Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983) is a 40-item self-report scale, with 20 items assessing transient anxiety based in the present (state) and 20 assessing anxiety in general (trait). Responses are based on a 4-point Likert scale, scores range from 20 to 80. This measure has been found to have high discriminant and convergent validity with other measures of anxiety (Speilberger et al., 1983).

The *Liebowitz social anxiety scale* (LSAS; Liebowitz, 1987) is a scale that is designed to assess fear and avoidance of performance and social interaction situations. The 24-item self-report measure requires respondents to rate each scenario (either performance or social interaction) on a scale of 0 to 3 for fear (none to severe) and avoidance (never to usually). The scale has been found have good internal consistency and convergent validity (Heimberg, Horner, Juster, Safren, Brown, Scheiner, & Liebowitz, 1999).

The *social phobia inventory* (SPIN; Connor, Davidson, Churchill, Tupier, Sherwood, Fog, & Weisler, 1998) is a 17-item self-report measure to assess fear, avoidance and physiological discomfort to a number of social situations. Respondents rate each item on a scale of 0 to 4 (not at all to extremely) to indicate distress. Scores range between 0 and 68. The measure has been found to have good test-re-test reliability, internal consistency, convergent and divergent validity (Connor et al., 1998).

2.4 Procedure

There were two approaches to recruiting undergraduate students. One method was to advertise on a University webpage aimed at recruiting research participants, students would respond to the advert by making e-mail contact with a researcher. The other method of recruitment was to place the screening questionnaire within a study area in the School of Psychology for students to complete and post into a locked drop-box. Contact details were recorded on the screening questionnaire, if the participants were eligible they were e-mailed a copy of the information sheet and asked to make contact if they wished to participate in the study.

The test session took place in the University Department. Participants were introduced to the equipment being used in the laboratory, particularly to the electrodes and how they would be utilised to enable informed consent. A brief visual acuity test was then completed to ensure participants had normal or corrected–to-normal vision. To prepare for EMG recording, the area underneath the right eye was cleaned using a surgical spirit and water solution to remove any excess oils, dirt or make-up and then the skin was lightly exfoliated using an abrasive pad. The startle response (or EMG recording) refers to the reflex of the orbicularis oculi muscle under the eye. Two 4-millimetre silver-silver chloride (Ag-AgCl) re-usable electrodes were filled with conductive electrode gel and attached to the skin using double-sided adhesive collars. The electrodes placed approximately 0.8 cm below the pupil and outer canthus of the left eye (Fridlund & Cacioppo, 1986; Tassinary & Cacioppo, 2000). The virtual reality visor and headphones were then placed, as comfortably as possible, on the participants head ensuring that the stimuli could be seen on the visor's screen and that the EMG electrodes underneath the eye were not disturbed. The instructions for the tasks were presented via a computer to the virtual reality visor and were also read aloud to the participant. Participants were asked not to move or speak during the task, unless they wanted to stop, to ensure minimum movement and interference with the psychophysiological recording. The experimenter remained in the room with participants due to the nature of the test equipment, and sat at a computer behind a screen throughout the task.

Participants sat through a 1-minute baseline period during which they were asked to look at a centrally presented white fixation cross. The habituation period followed in which participants were again asked to look at the fixation point for 65-seconds during which time 3 startle probes (50ms burst of white noise) were presented at 10, 30 and 50 seconds. The main task consisted of 48 trials; 12 presentations of light, dark, neutral faces and fear faces. For each stimulus type, 8 trials were paired with a startle probe and 4 without. On each trial the fixation cross was displayed for 2 seconds and was then followed by the presentation of the stimulus for 4.2 seconds. On startle trials, the acoustic startle probe was delivered 3 seconds following stimulus onset. Each trial was followed by a 10-second inter-trial interval. Following the experimental task, participants were asked to complete the collection of questionnaires.

The startle task was presented after two other tasks, (a visual probe and perceptual discrimination task, both containing different face stimuli to those used in the present study), which are not reported here as they addressed different theoretical questions and hypotheses to those of the present study.

3. Results

3.1 Group characteristics

Participants (who had been screened using the short-FNE to favour extreme social anxiety scorers) were allocated to the LSA or HSA group according to whether their scores on the full FNE (completed under standardized conditions) were below, or above, the sample median. The LSA group (*n* = 20; 5 male, 15 female) had FNE scores of less than or equal to 9 and the HSA group (*n* = 19; 4 male, 15 female) had FNE scores of 10 or more.

Questionnaire measures, age and years in education were entered into independent samples t-tests, with group as the independent variable. Significant differences were found between the low and high anxiety groups on all measures of social anxiety and mood (refer to Table 1). There were no significant differences between groups in age or years of education.

Table 1.

Characteristics of high and low anxiety groups.

	Low social anxiety (n= 20)		High social anxiety (n= 19)			
	M	SD	M	SD	*t(35)*	*p*
FNE	5.16	3.32	24.2	5.11	-13.57	<.001
BDI-II	3.11	2.11	12.83	8.52	-4.82	<.001
STAI-T	30.58	4.61	47.0	8.53	-7.34	<.001
STAI-S	28.89	4.93	40.83	11.59	-4.12	<.001
LSAS	20.16	9.42	47.72	24.2	-4.61	<.001
SPI	6.05	3.99	21.39	13.81	-4.64	<.001
Age	22.26	4.07	21.17	3.24	.904	.372
Education (Years)	14.89	1.82	15.05	1.70	-.277	.783
Male:Female Ratio		5:15		4:15		

Note. FNE = Fear of Negative Evaluation Scale; BDI-II = Beck Depression Inventory version II; STAI-T = Spielberger Anxiety Inventory-Trait; STAI-S = Spielberger Anxiety Inventory- State; LSAS: Liebowitz Social Anxiety Scale; SPI = Social Phobia Inventory.

3.2 Data preparation

EMG signal was filtered using a Finite response (30-500 Hz) bandpass filter, and subsequently rectified and integrated with a constant of 50 Hz. Startle response amplitude was defined as the difference in amplitude between the mean EMG in the 50 ms prior to the startle response, and the maximum EMG response between 20 ms and 250 ms after startle probe presentation. Latency of peak startle response was also recorded. Mean startle response amplitudes in each condition are displayed in Table 2. Mean startle response latencies in each condition are displayed in table 3. Kolmogorov-Smirnov tests indicated that the distributions of startle amplitudes for each condition did not differ significantly from normality. Startle amplitudes were also winsorised for further analysis, thus startle responses greater than 3 SDs above each participants mean startle responses were substituted with a value equal to the participants mean + 3 SD (Bernat, Patrick, Benning & Tellegen, 2006). Winsorising the startle data allowed the influence of outliers, which may have been generated through blinks or movement, to be minimised.

3.2 Peak startle

Mean peak startle responses were entered into a 2 x 4 mixed design ANOVA, with group (low vs. high social anxiety) as a between subjects factor and stimuli (dark, light, neutral face and fear face) as the within-subjects factor. Results revealed a significant main effect of stimulus, $F(3,105)=3.08$ $p< .05$ (effect size was moderate; partial eta squared .081, with adequate power; observed power .706).

Table 2.

Mean startle amplitude for each stimulus type x social anxiety group.

	Low socially anxious (n =19)		High socially anxious (n = 18)	
	M	SD	M	SD
Light	47.3	40.5	30.8	25.4
Dark	52.2	43.7	37.2	24.4
Neutral faces	47.4	43.4	30.8	24.9
Fear faces	48.2	46.5	32.5	26.5

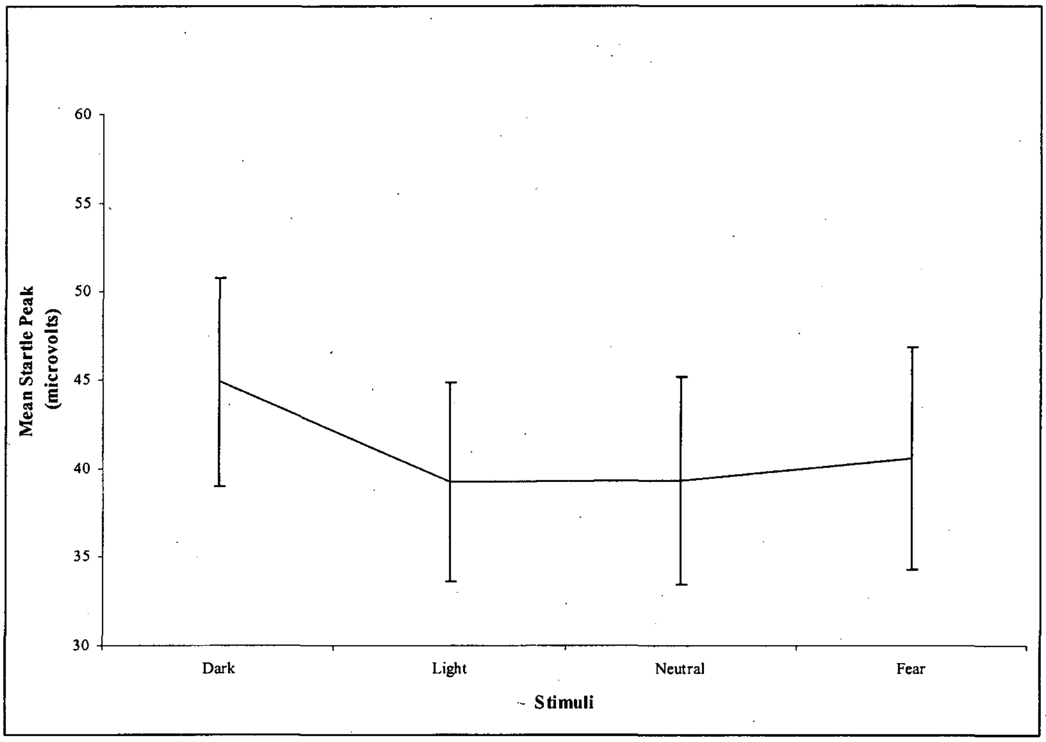

Figure 4.

Main effect of stimulus (startle amplitude).

3.3 Startle latency

Mean startle latencies were entered into a 2 x 4 mixed design ANOVA, with group (low vs. high social anxiety) as a between subjects factor and stimulus (dark, light, neutral face and angry face) as the within-subjects factor. A significant main effect of stimulus was not found, [F(3,105)=1.06 p=.369] and neither was an interaction effect between group and stimulus [F(3,105)= 2.22, p=.091].

3.4 Post-hoc tests

Bonferonni post-hoc paired comparisons indicated that participants mean peak startle response for the dark condition (M= 44.88, SD= 35.99) was significantly greater than their peak startle responses in the light condition (M= 39.24, SD= 34.52), to neutral faces (M= 39.32, SD= 36.15) and fear faces (M= 40.57, SD= 38.42), ps< .05. However all other comparisons were non-significant i.e. no significant differences between neutral faces, fear faces and light. The interaction between anxiety group and stimulus was non-significant [F(3,105)= 0.063, p= .979]. The main effect of group was non-significant [F(1,35)= 1.93, p= .174].[2]

[2] Startle amplitudes were winsorized in favour of within-subject z transformations as this allowed startle amplitudes to be compared between groups. However, it is reassuring to note that the pattern of within-subjects effects following z-transformation is similar to that reported in the main text and that obtained when using log transformed data. Specifically, z-transformed startle amplitudes were larger in the dark condition compared to all other conditions F(3,105)= 6.542, p<.001. Again, there was no main effect of group [F(1,35)= .007, p= .935] or significant group by stimulus interaction [F(3,105)= .325, p=.806]

Correlations between self-report measures of social, trait and state anxiety and depression with mean peak startle and startle latency scores for each stimulus condition were non-significant. To examine whether self-report anxiety was associated with greater startle in the dark condition *relative to* the light condition, bias scores, equal to the mean startle response in dark minus mean startle response in the light were computed for each participant. Similar bias scores were computed for the fear vs. neutral face contrast (i.e mean startle response to fear faces minus mean startle response to neutral faces) and the face (pooled across fear and neutral) vs. light condition contrast. All correlations between these bias scores and self-report measures of social anxiety were non-significant. Similar analyses of bias scores for startle latency data were also non-significant.

Table 3

Mean startle latency for each stimulus type x social anxiety group.

	Low socially anxious (n =19)		High socially anxious (n = 18)	
	M	SD	M	SD
Light	104.24	13.76	100.85	4.94
Dark	100.54	7.08	102.7	8.34
Neutral faces	105.18	16.03	102.44	6.44
Fear faces	104.29	13.18	101.35	5.0

3.5 Supplementary analyses: The influence of gender on startle response

Supplementary analyses examined whether the gender of the facial expression had any impact upon startle response as conceivably, male faces might convey significantly more threat to the predominantly female sample, than female faces. Peak startle data was entered into a 2 x 2 x 2 mixed design ANOVA, with group (low vs. high social anxiety) as a between subjects factor, face emotion (neutral vs. fear) and face gender (male vs. female) as within subjects factors. No main effect of gender was found $F(1,35)=.052$, $p=.821$, nor were there any interactions between face gender and group $F(1,35)=.051$, $p=.822$ or emotion and face gender $F(1,35)< 1$, $p=.984$.[3]

4. Discussion

The aim of the present study was to utilise the startle technique, a behavioural index of sub-cortical appraisal, to investigate the predictions from integrated neurocognitive theories of anxiety which suggest that cues relevant to social concerns compared to less specific fear stimuli potentiate neural systems involved in fear and anxiety (i.e. the amygdala) in those with social anxiety. The sub-cortical appraisal of social and non- social cues in high and low social anxiety (HSA vs. LSA) was investigated by measuring startle magnitude and latency to fear and neutral faces (social cues) and light and dark patches (non-social cues).

The main findings from the present study can be summarised as follows: (i) Participants, irrespective of anxiety group, had larger startle amplitudes to dark patches compared to grey patches (i.e. light), neutral and fear faces. (ii) There were no differences between anxiety groups in startle amplitude or latency

[3] Due to the low number of males recruited in the present study it was not appropriate to enter participant gender as an additional between-subjects factor.

in response to the dark patches. (iii) There were no differences between groups in startle amplitude or latency to social cues (i.e. faces) relative to non-social cues (i.e. patches). (iv) There were also no differences in the HSA group in startle amplitude or latency between neutral and fear faces. These findings will be discussed with reference to key predictions and findings from related studies.

4.1 Startle potentiation to social stimuli

HSA individuals were not found to have a potentiated startle response to social cues (neutral and fear faces) in comparison to LSA individuals. This does not support cognitive theory of social phobia that predicts social anxiety leads to a maladaptive appraisal of threat to social cues (Clark & McManus, 2002; Rapee & Heimberg, 1997). As the startle response is a behavioural index of sub-cortical appraisal, it was predicted that the HSA group would have a potentiated startle response to social cues, particularly fear faces, due to appraisal activating the sub-cortical fear circuit. The neurocognitive approach makes predictions that threat appraisal may be associated to potentiation of the amygdala (Bishop, 2007), the findings from the present study do not support these predictions. The findings are not consistent with previous research either, which has found that HSA groups explicitly appraise emotional faces as more threatening than LSA groups (Richards et al., 2002; Joormann & Gotlib, 2006; Winton et al., 1995) and that conditions of social-evaluative threat and self-focused attention potentiate the startle response in HSA groups (Cornwell et al., 2006; Panayiotou & Vrana, 1998). Neuroimaging data has also revealed that HSA groups have increased amygdala activation in response to phobia related cues such as emotional faces (Birbaumer et al., 1998; Somerville et al., 2004). In addition, phobia specific cues potentiate startle in spider phobics (Hamm, Cutherbert, Globisch, & Vaitl, 1997; Globisch, Hamm, Esteves, & Ohman, 1999) and groups with personal injury fears (Hamm et al., 1997). Therefore, theoretical predictions and previous research suggests that elevated startle potentiation to face cues in the current sample of high socially anxious individuals might have been expected.

The previous studies which have reported the use of the startle technique to investigate sub-cortical processing in social phobia utilised contextual methods to induce social anxiety, which is in contrast to the present study that used specific social cues (i.e. faces). For example, Cornwell et al. (2006) induced fear of negative evaluation by introducing participants to a virtual reality environment which simulated standing centre stage whilst participants anticipated giving a speech, whereas Panayiotou & Vrana (1998) asked participants to undertake a digit recall task under conditions of self-focused and non-self focused attention. Each of these studies revealed elevated startle potentiation in HSA individuals in response to the induced social situations. It is plausible that these contextual manipulations elicited more potent fear responses which resulted in the sub-cortical appraisal of threat and potentiated startle response in socially anxious individuals than did the presentation of discrete social cues in the current study. If these predictions are correct, it would be expected that state anxiety would be higher in the HSA groups exposed to contextual manipulations compared to the group in the present study. Consistent with the present study, Panayiotou and Vrana (1998) also measured state anxiety using the Spielberger self-report questionnaire, however they did not report mean scores so comparisons cannot be made across groups.

It is plausible that contextual cues induce self-focused attention, which would be consistent with proposals from Clark & McManus (2002) which emphasise the role of self-focus in the experience of social anxiety. During self-focussed attention individuals with social anxiety construct self images taken from the observer perspective (i.e. how the audience may view them), this is thought to be a primary source of threat in socially anxious individuals. This suggests that it may be the observer perspective taken by individuals with social anxiety that is troublesome, therefore facial expressions from a field perspective (i.e. looking at external cues) may not induce such a potent fear response and sub-cortical appraisal of threat. Having an experimenter remain within the laboratory, as with the present study, could mimic conditions that induce self-focused attention due to the problems with experimenter bias in such situations. However, the disadvantage of the present study is this was not controlled for, so the influence of the experimenter cannot be established. Future research may need to further investigate sub-cortical appraisal in social anxiety using contextually relevant paradigms as discrete social cues (i.e. faces) may not be sufficient to promote the activation of the appraisal system (i.e. the amygdala).

Research into attention biases in social phobia has revealed that HSA groups tend to have a vigilant-avoidant pattern of attention towards threat, initially scanning the environment for threat cues but rapidly directing attention away from cues such as emotional faces. It has been found that avoidance from the emotional face tends to occur between 500ms and 1250ms (Mogg, Philpott & Bradley, 2004; Vassilopoulos, 2005). In the present study faces were presented for a period of 3000ms and in light of the vigilance-avoidance hypothesis it is plausible that the HSA group were avoiding eye gaze towards the faces at the time the startle probe was delivered, therefore eradicting any differences between groups in sub-cortical processing. As such future studies are encouraged to monitor the extent to which participants engage with task instructions and do not adopt cognitive strategies (e.g. attentional avoidance) that confound experimental manipulations and results.

Interestingly, recent research has sought to clarify the time course of threat processing as measured by startle-probe designs. Globisch et al. (1999) presented spider phobics with fear-relevant and irrelevant slides for durations of between 150ms and 6-seconds. Startle probes were delivered at five different time points during picture presentation, startle responses were observed at 300ms and were maintained at all later probe times. This evidence suggests that the startle response is not ameliorated over longer presentation trials due to avoidance of fear-related pictures. While it is unclear whether avoidance strategies were used in the present study, the presentation of images through the head-mounted virtual reality headset was likely to dissuade eye-movements to regions outside the display. Furthermore, participant eye closure during picture onset was not observed or reported by any participants at debrief.

The present study investigated individual differences between high and low social anxiety groups which were formed by undertaking a median split of an enriched sample of university students based upon the Fear of Negative Evaluation Scale (FNE; Watson & Friend, 1969). The use of analogue samples to investigate cognitive biases in social phobia has been recommended by Stopa and Clark (2001), as the psychological processes in social anxiety are essentially analogous to patients with social phobia. Therefore, it is a fruitful way to investigate the processes which underlie social phobia and an approach which has widely been employed (Hirsch & Clark, 2004). It has to be considered whether there was sufficient power with the present analogue sample to be able to infer differences which may exist in a clinical sample. Post-hoc power was reported as .706, which was deemed adequate. However, this may not have been sufficient to observe relationships between startle magnitude and potential confounding variables (e.g. depression) in the post-hoc correlations. T-tests revealed a significant difference between groups on a measure of depression (Beck Depression Inventory-II) with mean scores of 3.11 (low anxiety group) compared to 12.83 (high anxiety group). Correlations between BDI-scores and peak startle and startle latency responses were found to be non-significant, however a non-significant result may have been due to insufficient power. It is possible that depression did influence startle responding, particularly as a score of twelve on the BDI indicates mild to moderate depression. In terms of power, it would have been beneficial to undertake an a priori power calculation to determine the sample size required, however individual differences in appraisal between high and low anxiety groups have consistently been found with similar sample sizes in cognitive bias research (Hirsch & Clark, 2004).

In addition, it has to be considered whether the method of splitting the groups provided adequate difference to produce individual differences in startle magnitude. Panayiotou and Vrana (1998) did not use the FNE as a measure and although Cornwell et al., used the FNE they had a correlational design with one group (median FNE score of 11). However, Winton et al. (1995) found significant group differences in the appraisal of emotional faces when HSA and LSA groups were formed using FNE scores and extensive evidence of attentional biases to face cues has been revealed in sub-clinical analogue designs. The average FNE scores appear similar between the present and comparison HSA group (24.2 vs. 22.5) and LSA groups (5.2 vs. 5.3). This seems to suggest that there was adequate difference between the anxiety groups in the present study. Additionally, Stopa and Clark (2001) recommend that low social anxiety groups should have a mean of seven or below on the FNE and high anxiety groups a mean of twenty or above, which has been met with the present study.

In the present study participants were presented with 32 startle probes across the 48 trials, which were consistently delivered 3 seconds after stimulus onset. While it is possible that the number of startle probes delivered resulted in habituation which may have ameliorated individual differences in emotion potentiated startle, the number of startles and rate of presentation is consistent with previous research (Lang, Bradley & Cuthbert, 1998)

4.2 Startle potentiation to the light-dark paradigm

It was found in the present study that both HSA and LSA groups had a potentiated startle in response to dark patches compared to emotional faces (neutral and fear) and grey patches, but that there were no significant differences between groups. Cognitive theory of social phobia predicts that HSA groups would specifically appraise social cues with a threat bias (Clark & McManus, 2002; Rapee & Heimberg, 1997), thus from a neurocognitive perspective amygdala hyper-arousal would only occur in relation to sub-cortical appraisal of threat to social cues in the HSA group (Bishop, 2007). The findings from the present study were inconsistent with these theoretical predictions, as the HSA group showed a potentiated startle response to non-social than social threat (i.e. dark vs emotional faces). Recent evidence suggests that anxiety groups (i.e. PTSD, high risk groups) show a potentiated startle response to dark compared to non-anxious or low risk groups (Grillon et al., 1998). Thus, a competing hypothesis was that individuals with vulnerability to social anxiety would show a greater startle potentiation to a manipulation of non-specific contextual threat (i.e. darkness) than low anxious controls.

The findings from the present study did not support this hypothesis either, as there were no group differences in startle potentiation to the dark. A recent study revealed that darkness facilitates the startle response in college students without anxiety disorders (Grillon et al., 1997), thus the findings from the present study suggest that the LSA group showed a potentiated startle response to the dark as found with college students and that this was also consistent with the HSA group. This indicates that the HSA group processed the dark condition similarly to the LSA group, implying that sub-cortical hypersensitivity to non-specific contextual threat is not characteristic of social anxiety, contrary to predictions from cognitive and neurocognitive theory.

4.3 Implications for further research

The findings from the present study provide little evidence for the neurocognitive approach and cognitive theory of social phobia, which predict a hypersensitivity of the amygdala in response to sub-cortical appraisal of threat to social cues (Bishop, 2007; Clark & McManus, 2002; Rapee & Heimberg, 1997). It has been found previously that HSA groups appraise social cues such as faces with a threat bias (Joormannn & Gotlib, 2006; Richards et al., 2002; Winton et al., 1995) and that processing emotional faces is related to increased amygdala activation in social anxiety (Birbaumer et al., 1998; Somerville et al., 2004). As the startle response is a behavioural index of sub-cortical processing, it was anticipated that a potentiated startle would have been observed in response to social cues in the HSA group. The startle technique is a useful measure to investigate the behavioural manifestations of sub-cortical threat appraisal, as it is a reliable component of the defensive cascade serving a protective function to individuals. Evidence suggests that the startle response is potentiated in social anxiety in response to phobia-specific tasks that induce self-focused attention or risk of negative evaluation from others (Cornwell et al., 2006; Panayiotou & Vrana, 1998). It is possible that the facial expression stimuli utilised in the present study were not salient to the concerns of individuals with elevated but sub-clinical levels of social anxiety and therefore did not produce sub-cortical appraisal of threat in the HSA group. As such future research is encouraged to use more contextually relevant designs that include cues with more contextual relevance. It would be of benefit to further utilise the startle technique to further understand the cues that potentiate the defensive cascade in social anxiety.

References

Adolphs, R., Tranel, D., Damasio, A.R. (1998). The human amygdala in social judgment. *Nature 393*(6684), 470-474.

Amaral, D. (2002). The primate amygdala and the neurobiology of social behavior: implications for understanding social anxiety . . *Biological Psychiatry, 51*(1), 11-17.

Amin, N., Foa, E. B., & Coles, M. E. (1998). Negative interpretation bias in social phobia. *Behaviour Research and Therapy, 36*(10), 945-957.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders (3rd ed.).* Washington, DC: American Psychiatric Association.

Barlow, D. H. (1988). *Anxiety and its disorders.* New York: Guildford.

Beck, A.T., & Steer, R.A. (1987). *Beck Depression Manual.* San Antonio, TX: Psychological Corporation.

Bernat, E., Patrick, C.J., Benning, S.D., & Tellegen, A. (2006). Effects of picture content and intensity on affective physiological response. *Psychophysiology, 43 (1),* 93-103.

Birbaumer, N., Grodd, W., Diedrich, O., Klose, U., Erb, M., Lotze, M., Schneider, F., Weiss, U., Flor, H. (1998). fMRI reveals amygdala activation to human faces in social phobics. *NeuroReport, 9*(6), 1223-1226.

Bishop, S. J. (2007). Neurocognitive mechanisms of anxiety: an integrative account. *Trends in Cognitive Sciences, 11*(7), 307-316.

Broks, P., Young, A.W., Maratos, E.J., Coffey, P.J., Calder, A.J., Isaac, C.L., Mayes, A.R., Hodges, J.R., Montaldi, D., Cezayirli, E., Roberts, R., Hadley, D. (1998). Face processing impairments after encephalitis: amygdala damage and recognition of fear. *Neuropsychologia, 36*(1), 59-70.

Brown, M.A., & Stopa, L. (2007). The spotlight effect and illusion of transparency in social anxiety. *Journal of Anxiety Disorders, 21*(6), 804-819.

Carlsson, K., Petersson, K. M., Lundqvist, D., Karlsson, A., Ingvar, M., & Ohman, A. (2004). Fear and the amygdala: Manipulation of awareness generates differential cerebral responses to phobic and fear-relevant (but nonfeared) stimuli. *Emotion, 4*(4), 340-353.

Clark, D. M., McManus, F. (2002). Information processing in social phobia. *Biological Psychiatry, 51*(1), 92-100.

Connor, K. M,, Davidson, J. R. T., Churchill, L. E., Sherwood, A., Foa, E. B., & Weisler, R. H. (2000). Psychometric properties of the Social Phobia Inventory (SPIN): a new self-rating scale. *Br J Psychiatry,* 176, 379–386.

Cornwell, B. R., Johnson, L., Berardi, L., & Grillon, C. (2006). Anticipation of public speaking in virtual reality reveals a relationship between trait social anxiety and startle reactivity. *Biological Psychiatry, 59*(7), 664-666.

Davidson, R. (2002). Anxiety and affective style: role of prefrontal cortex and amygdala *Biological Psychiatry, 51*(1), 68 - 80.

Fridlund, A.J. & Cacioppo, J.T. (1986). Guidelines for human electromyographic research. *Psychophysiology, 23 (5),* 567-589.

Furmark. (2002). Social phobia: overview of community studies. *Acta Psychiatrica Scandinavica, 105*(2), 84-93.

Garner, M., Mogg, K., & Bradley, B.P. (2006). Orienting and maintenance of gaze to facial expressions in social anxiety. *Journal of Abnormal Psychology, 115 (4),* 760-770.

Globisch, J., Hamm, A.O., Esteves, F., & Ohman, A. (1999). Fears appear fast: Temporal course of startle reflex potentiation in animal fearful subjects. *Psychophysiology, 36,* 66-75.

Graham, F. K. (1979). Distinguishing among orienting, defense, and startle reflexes. In: Kimmel, H. D., van Olst, E. H., & Orlebeke, J. F. (1979). *The Orienting Reflex in Humans*. An International Conference Sponsored by the Scientific Affairs Division of the North Atlantic Treaty Organization Erlbaum, Hillsdale, NJ, (pp. 37–167).

Grillon, C., & Davis, M. (1997). Effects of stress and shock anticipation on prepulse inhibition of the startle reflex. *Psychophysiology, 34*(5), 511-517.

Grillon, C., Dierker, L., & Merikangas, K. R. (1998). Fear-potentiated startle in adolescent offspring of parents with anxiety disorders. *Biological Psychiatry, 44*(10), 990-997.

Hamm, A.O., Cuthbert, B.N., Globisch, J., & Vaitl, D. (1997). Fear & the startle reflex: Blink modulation and autonomic response patterns in animal & mutilation fearful subjects. *Psychophysiology, 31*(1), 97-107.

Heimberg, R.G., Horner, K.J., Juster, H.R., Safren, S.A., Brown, E.J., Schneiner, F.R., & Liebowitz, M.R. (1999). Psychometric properties of the Liebowitz Social Anxiety Scale. *Psychological Medicine, 29*(1), 199-212.

Joormann, J., Gotlib, I.H. (2006). Is This Happiness I See? Biases in the Identification of Emotional Facial Expressions in Depression and Social Phobia. *Journal of Abnormal Psychology, 115*(4), 705-714.

Judd, L. L. (1994). Social phobia: A clinical overview. *Journal of Clinical Psychiatry, 55*(6), 5-9.

Lang, P. J., Bradley, M.M., Cuthbert, B.N. (1990). Emotion, attention, and the startle reflex. . *Psychological Review, 97*(3), 377-395.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1998). Emotion and motivation: Measuring affective perception. *Journal of Clinical Neurophysiology,* 15, 397-408.

Leary, M. R. (1983). A brief version of the fear of negative evaluation scale, *Personality and Social Psychology Bulletin,* 9, 371–376.

LeDoux, J. E. (1995). Emotion: Clues from the brain. *Annual Review of Psychology, 46*, 209-235.

Liebowitz, M.R. (1987). Social phobia. *Modern Problems of Pharmacopsychiatry, 22,* 141-173.

Maren, S., & Quirk, G. J. (2004). Neuronal signalling of fear memory. *Nature Reviews Neuroscience, 5*(11), 844-852.

Mathews, A., & Mackintosh, B. (1998). A cognitive model of selective processing in anxiety. *Cognitive Therapy and Research, 22*(6), 539-560.

Mogg, K., Bradley, B.P. . (1998). A cognitive-motivational analysis of anxiety *Behaviour Research and Therapy 36*(9), 809-848.

Mogg, K., Philippot, P., & Bradley, B. P. (2004). Selective attention to angry faces in clinical social phobia. *Journal of Abnormal Psychology, 113*(1), 160-165.

Ohman, A., Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning *Psychological Review; 108*(3), 483-522.

Panayiotou, G., Vrana, S.R. (1998). Effect of self-focused attention on the startle reflex, heart rate, and memory performance among socially anxious and nonanxious individuals *Psychophysiology, 35*, 328-336

Rapee, R. M., Heimberg, R.G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy, 35*(8), 741-756

Richards, A., French, C.C., Calder, A.J., Webb, B., Fox, R., Young, A.W. (2002). Anxiety-related bias in the classification of emotionally ambiguous facial expressions. *Emotion, 2*(3), 273-287.

Schneier, F. R., Johnson, J., Hornig, C.D., Liebowitz, M.R., Weissman, M.M. . (1992). Social phobia. Comorbidity and morbidity in an epidemiologic sample. *Archives of General Psychiatry, 49*(4), 282-288.

Somerville, L. H., Kim, H., Johnstone, T., Alexander, A. L., & Whalen, P. J. (2004). Human amygdala responses during presentation of happy and neutral faces: Correlations with state anxiety. *Biological Psychiatry, 55*(9), 897-903.

Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-TraitAnxiety Inventory.* CA: Consulting Psychologists Press.

Storch, E.A., Roberti, J.W., & Roth, D.A. (2004). Factor structure, concurrent validity, and internal consistency of the Beck Depression Inventory Second Edition in a sample of college students. *Depression & Anxiety, 19*(3), 187-189.

Stopa, L., & Clark, D.M. (2001). Social phobia: Comments on the viability and validity of an analogue research strategy and British norms for the Fear of Negative Evaluation Questionnaire. *Behavioural and Cognitive Psychotherapy, 29,* 423-430.

Tassinary, L.G., & Cacioppo, J.T. (2000). The skeletomotor system: Surface Electromyogramphy. In J.T. Cacioppo, L.G. Tassinary, & G.G. Bernston. *Handbook of Psychophysiology.* New York: Cambridge University Press.

Vassilopoulos, S. P. (2005). Social anxiety and the vigilance-avoidance pattern of attentional processing. *Behavioural and Cognitive Psychotherapy, 33*(1), 13-24.

Vassilopoulos, S. P. (2006). Interpretation and judgmental biases in socially anxious and nonanxious individuals. *Behavioural and Cognitive Psychotherapy, 34*(2), 243-254.

Voncken, M. J., Bogels, S. M., & de Vries, K. (2003). Interpretation and judgmental biases in social phobia. *Behaviour Research and Therapy, 41*(12), 1481-1488.

Watson, D., & Friend, R. (1969). Measurement of social evaluative anxiety. *Journal of Consulting and Clinical Psychology*, 33, 448–457.

Whalen, P. J., Rauch, S.L., Etcoff, N.L., McInerney, S.C., Lee, M.B., Jenike, M.A. . (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge *The Journal of Neuroscience, 18*(1), 411-418.

Wilson, J. K., & Rapee, R. M. (2005). Interpretative biases in social phobia: Content specificity and the effects of depression. *Cognitive Therapy and Research, 29*(3), 315-331.

Winton, E. C., Clark, D.M., Edelman, R.J. (1995). Social anxiety, fear of negative evaluation and the detection of negative emotion in others *Behaviour Research and Therapy, 33*(2), 193-196

List of Appendices

Appendix A

Clinical Psychology Review Guide for Authors

CLINICAL PSYCHOLOGY REVIEW

Guide for Authors

SUBMISSION REQUIREMENTS: Authors should submit their articles electronically via the Elsevier Editorial System (EES) page of this journal (http://ees.elsevier.com/cpr). The system automatically converts source files to a single Adobe Acrobat PDF version of the article, which is used in the peer-review process. Please note that even though manuscript source files are converted to PDF at submission for the review process, these source files are needed for further processing after acceptance. All correspondence, including notification of the Editor's decision and requests for revision, takes place by e-mail and via the Author's homepage, removing the need for a hard-copy paper trail. Questions about the appropriateness of a manuscript should be directed (prior to submission) to the Editorial Office, details at URL above. Papers should not exceed 50 pages (including references).

Submission of an article implies that the work described has not been published previously (except in the form of an abstract or as part of a published lecture or academic thesis), that it is not under consideration for publication elsewhere, that its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published elsewhere in the same form, in English or in any other language, without the written consent of the Publisher.

FORMAT: We accept most wordprocessing formats, but Word, WordPerfect or LaTeX are preferred. Always keep a backup copy of the electronic file for reference and safety. Save your files using the default extension of the program used.

Please provide the following data on the title page (in the order given).

Title. Concise and informative. Titles are often used in information-retrieval systems. Avoid abbreviations and formulae where possible.

Author names and affiliations. Where the family name may be ambiguous (e.g., a double name), please indicate this clearly. Present the authors' affiliation addresses (where the actual work was done) below the names. Indicate all affiliations with a lower-case superscript letter immediately after the author's name and in front of the appropriate address. Provide the full postal address of each affiliation, including the country name, and, if available, the e-mail address of each author.

Corresponding author. Clearly indicate who is willing to handle correspondence at all stages of refereeing and publication, also post-publication. **Ensure that telephone and fax numbers (with country and area code) are provided in addition to the e-mail address and the complete postal address.**

Present/permanent address. If an author has moved since the work described in the article was done, or was visiting at the time, a 'Present address' (or 'Permanent address') may be indicated as a footnote to that author's name. The address at which the author actually did the work must be retained as the main, affiliation address. Superscript Arabic numerals are used for such footnotes.

Abstract. A concise and factual abstract is required (not exceeding 200 words). This should be typed on a separate page following the title page. The abstract should state briefly the purpose of the research, the principal results and major conclusions. An abstract is often presented separate from the article, so it must be able to stand alone. References should therefore be avoided, but if essential, they must be cited in full, without reference to the reference list.

STYLE AND REFERENCES: Manuscripts should be carefully prepared using the Publication Manual of the American Psychological Association, 5th ed., 1994, for style. The reference section must be double spaced, and all works cited must be listed. Please note that journal names are not to be abbreviated.

Reference Style for Journals: Cook, J. M., Orvaschel, H., Simco, E., Hersen, M., and Joiner, Jr., T. E. (2004). A test of the tripartite model of depression and anxiety in older adult psychiatric outpatients, *Psychology and Aging*, 19, 444-45.

For Books: Hersen, M. (Ed.). (2005). Comprehensive handbook of behavioral assessment (2 Volumes). New York: Academic Press (Elsevier Scientific).

TABLES AND FIGURES: Present these, in order, at the end of the article. High-resolution graphics files must always be provided separate from the main text file (see http://ees.elsevier.com/cpr for full instructions, including other supplementary files such as high-resolution images, movies, animation sequences, background datasets, sound clips and more).

PAGE PROOFS AND OFFPRINTS: When your manuscript is received by the Publisher it is considered to be in its final form. Proofs are not to be regarded as 'drafts'. One set of page proofs will be sent to the corresponding author, to be checked for typesetting/editing. No changes in, or additions to, the accepted (and subsequently edited) manuscript will be allowed at this stage. Proofreading is solely the authors' responsibility.
The Publisher reserves the right to proceed with publication if corrections are not communicated. Please return corrections within 3 days of receipt of the proofs. Should there be no corrections, please confirm this.

COPYRIGHT: Upon acceptance of an article, authors will be asked to transfer copyright (for more information on copyright, see http://www.elsevier.com). This transfer will ensure the widest possible dissemination of information. A letter will be sent to the corresponding author confirming receipt of the manuscript. A form facilitating transfer of copyright will be provided.
If excerpts from other copyrighted works are included, the author(s) must obtain written permission from the copyright owners and credit the source(s) in the article. Elsevier has forms for use by authors in these cases available at

www.elsevier.com/locate/permissions phone: (+44) 1865 843830, fax: (+44) 1865 853333, e-mail: permissions@elsevier.com

NIH voluntary posting policy US National Institutes of Health (NIH) voluntary posting (" Public Access") policy Elsevier facilitates author response to the NIH voluntary posting request (referred to as the NIH "Public Access Policy", see http://www.nih.gov/about/publicaccess/index.htm) by posting the peer-reviewed author's manuscript directly to PubMed Central on request from the author, 12 months after formal publication. Upon notification from Elsevier of acceptance, we will ask you to confirm via e-mail (by e-mailing us at NIHauthorrequest@elsevier.com) that your work has received NIH funding and that you intend to respond to the NIH policy request, along with your NIH award number to facilitate processing. Upon such confirmation, Elsevier will submit to PubMed Central on your behalf a version of your manuscript that will include peer-review comments, for posting 12 months after formal publication. This will ensure that you will have responded fully to the NIH request policy. There will be no need for you to post your manuscript directly with PubMed Central, and any such posting is prohibited.

Appendix B

Behaviour Research and Therapy Guide for Authors

BEHAVIOUR RESEARCH AND THERAPY

An International Multi-Disciplinary Journal

Guide for Authors

For full instructions, please visit http://ees.elsevier.com/brat

Aims and Scope

Behaviour Research and Therapy encompasses all of what is commonly referred to as cognitive behaviour therapy (CBT). The major focus is on the following: experimental analyses of psychopathological processes linked to prevention and treatment; the development and evaluation of empirically-supported interventions; predictors, moderators and mechanisms of behaviour change; and dissemination of evidence-based treatments to general clinical practice. In addition to traditional clinical disorders, the scope of the journal also includes behavioural medicine. The journal will not consider manuscripts dealing primarily with measurement, psychometric analyses, and personality assessment.

The Editor and Associate Editors will make an initial determination of whether or not submissions fall within the scope of the journal and are of sufficient merit and importance to warrant full review.

Submission to the journal prior to acceptance Authors can submit their articles electronically via the Elsevier Editorial System (EES) page of this journal http://ees.elsevier.com/brat. The system automatically converts source files to a single Adobe Acrobat PDF version of the article, which is used in the peer-review process. Please note that even though manuscript source files are converted to PDF at submission for the review process, these source files are needed for further processing after acceptance. All correspondence, including notification of the Editor's decision and requests for revision, takes place by e-mail and via the Author's homepage, removing the need for a hard-copy paper trail.

Online submission is strongly preferred but authors can, in special cases, also submit via mail. Four copies of the manuscript, including one set of high-quality original illustrations, suitable for direct reproduction, should be submitted to **Professor G. T. Wilson, Psychological Clinic at Gordon Road, Rutgers, The State University of New Jersey, 41C Gordon Road, Piscataway, New Jersey, 08854-8067, USA.** Email: brat@rci.rutgers.edu. (Copies of the illustrations are acceptable for the other sets of manuscripts, as long as the quality permits refereeing.)

Submission of an article implies that the work described has not been published previously (except in the form of an abstract or as part of a published lecture or academic thesis), that it is not under consideration for publication elsewhere, that its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published elsewhere in the same form, in English or in any other language, without the written consent of the Publisher.

Presentation of manuscript Please write your text in good English (American or British usage is accepted, but not a mixture of these). Italics are not to be used for expressions of Latin origin, for example, in vivo, et al., per se. Use decimal points (not commas); use a space for thousands (10 000 and above). Print the entire manuscript on one side of the paper only, using double spacing and wide (3 cm) margins. (Avoid full justification, i.e., do not use a constant right-hand margin.) Ensure that each new paragraph is clearly indicated. Present tables and figure legends on separate pages at the end of the manuscript. If possible, consult a recent issue of the journal to become familiar with layout and conventions. Number all pages consecutively.

Provide the following data on the title page (in the order given).

Title. Concise and informative. Titles are often used in information-retrieval systems. Avoid abbreviations and formulae where possible.

Author names and affiliations. Where the family name may be ambiguous (e.g., a double name), please indicate this clearly. Present the authors' affiliation addresses (where the actual work was done) below the names. Indicate all affiliations with a lower-case superscript letter immediately after the author's name and in front of the appropriate address. Provide the full postal address of each affiliation, including the country name, and, if available, the e-mail address of each author.

Corresponding author. Clearly indicate who is willing to handle correspondence at all stages of refereeing and publication, also post-publication. **Ensure that telephone and fax numbers (with country and area code) are provided in addition to the e-mail address and the complete postal address.**

Present/permanent address. If an author has moved since the work described in the article was done, or was visiting at the time, a 'Present address' (or 'Permanent address') may be indicated as a footnote to that author's name. The address at which the author actually did the work must be retained as the main, affiliation address. Superscript Arabic numerals are used for such footnotes.

Abstract. A concise and factual abstract is required (maximum length 200 words). The abstract should state briefly the purpose of the research, the principal results and major conclusions. An abstract is often presented separate from the article, so it must be able to stand alone. References should therefore be avoided, but if essential, they must be cited in full, without reference to the reference list.

Keywords. Immediately after the abstract, provide a maximum of 6 keywords, to be chosen from the APA list of index descriptors. These keywords will be used for indexing purposes.

Abbreviations. Define abbreviations that are not standard in this field at their first occurrence in the article: in the abstract but also in the main text after it. Ensure consistency of abbreviations throughout the article.

N.B. Acknowledgements. Collate acknowledgements in a separate section at the end of the article and do **not**, therefore, include them on the title page, as a footnote to the title or otherwise.

Shorter Communications This option is designed to allow publication of research reports that are not suitable for publication as regular articles. Shorter Communications are appropriate for articles with a specialized focus or of particular didactic value. Manuscripts should be between 3000 - 5000 words, and must not exceed the upper word limit. This limit includes the abstract, text, and references, but not the title pages, tables and figures.

Arrangement of the article Subdivision of the article. Divide your article into clearly defined and numbered sections. Subsections should be numbered 1.1 (then 1.1.1, 1.1.2), 1.2, etc. (the abstract is not included in section numbering). Use this numbering also for internal cross-referencing: do not just refer to 'the text.' Any subsection may be given a brief heading. Each heading should appear on its own separate line.

Appendices. If there is more than one appendix, they should be identified as A, B, etc. Formulae and equations in appendices should be given separate numbering: (Eq. A.1), (Eq. A.2), etc.; in a subsequent appendix, (Eq. B.1) and so forth.

Acknowledgements. Place acknowledgements, including information on grants received, before the references, in a separate section, and not as a footnote on the title page.

Figure legends, tables, figures, schemes. Present these, in this order, at the end of the article. They are described in more detail below. High-resolution graphics files must always be provided separate from the main text file (see Preparation of illustrations).

Specific remarks Tables. Number tables consecutively in accordance with their appearance in the text. Place footnotes to tables below the table body and indicate them with superscript lowercase letters. Avoid vertical rules. Be sparing in the use of tables and ensure that the data presented in tables do not duplicate results described elsewhere in the article.

Preparation of supplementary data. Elsevier accepts supplementary material to support and enhance your scientific research. Supplementary files offer the author additional possibilities to publish supporting applications, movies, animation sequences, high-resolution images, background datasets, sound clips and more. Supplementary files supplied will be published online alongside the electronic version of your article in Elsevier Web products, including ScienceDirect: http://www.sciencedirect.com. In order to ensure that your submitted material is directly usable, please ensure that data is provided in one of our recommended file formats. Authors should submit the material in electronic format together with the article and supply a concise and descriptive caption for each file. For more detailed instructions please visit our artwork instruction pages at ⇒http://www.elsevier.com/artworkinstructions.

References Responsibility for the accuracy of bibliographic citations lies entirely with the authors

Citations in the text: Please ensure that every reference cited in the text is also present in the reference list (and vice versa). Any references cited in the abstract must be given in full. Unpublished results and personal communications should not be in the reference list, but may be mentioned in the text. Citation of a reference as 'in press' implies that the item has been accepted for publication.

Citing and listing of web references. As a minimum, the full URL should be given. Any further information, if known (author names, dates, reference to a source publication, etc.), should also be given. Web references can be listed separately (e.g., after the reference list) under a different heading if desired, or can be included in the reference list.

Text: Citations in the text should follow the referencing style used by the American Psychological Association. You are referred to the Publication Manual of the American Psychological Association, Fifth Edition, ISBN 1-55798-790-4, copies of which may be ordered from http://www.apa.org/books/4200061.html or APA Order Dept., P.O.B. 2710, Hyattsville, MD 20784, USA or APA, 3 Henrietta Street, London, WC3E 8LU, UK. Details concerning this referencing style can also be found at http://humanities.byu.edu/linguistics/Henrichsen/APA/APA01.html.

List: References should be arranged first alphabetically and then further sorted chronologically if necessary. More than one reference from the same author(s) in the same year must be identified by the letters "a", "b", "c", etc., placed after the year of publication.

Examples: Reference to a journal publication: Van der Geer, J., Hanraads, J. A. J., & Lupton R. A. (2000). The art of writing a scientific article. Journal of Scientific Communications, 163, 51-59.

Reference to a book: Strunk, W., Jr., & White, E. B. (1979). The elements of style. (3rd ed.). New York: Macmillan, (Chapter 4).

Reference to a chapter in an edited book: Mettam, G. R., & Adams, L. B. (1994). How to prepare an electronic version of your article. In B. S. Jones, & R. Z. Smith (Eds.), Introduction to the electronic age (pp. 281-304). New York: E-Publishing Inc.

Note that journal names are not to be abbreviated.

Preparation of illustrations

Submitting your artwork in an electronic format helps us to produce your work to the best possible standards, ensuring accuracy, clarity and a high level of detail.

General points

• Always supply high-quality printouts of your artwork, in case conversion of the electronic artwork is problematic.
• Make sure you use uniform lettering and sizing of your original artwork.
• Save text in illustrations as "graphics" or enclose the font.
• Only use the following fonts in your illustrations: Arial, Courier, Helvetica, Times, Symbol.
• Number the illustrations according to their sequence in the text.
• Use a logical naming convention for your artwork files, and supply a separate listing of the files and the software used.
• Provide all illustrations as separate files and as hardcopy printouts on separate sheets.
• Provide captions to illustrations separately.
• Produce images near to the desired size of the printed version.

For more detailed instructions please visit our artwork instruction pages at http://www.elsevier.com/artworkinstructions. You are urged to visit this site; some excerpts from the detailed information are given here.

*Formats*Regardless of the application used, when your electronic artwork is finalised, please "save as" or convert the images to one of the following formats (Note the resolution requirements for line drawings, halftones, and line/halftone combinations given below.):

EPS: Vector drawings. Embed the font or save the text as "graphics".
TIFF: Colour or greyscale photographs (halftones): always use a minimum of 300 dpi.
TIFF: Bitmapped line drawings: use a minimum of 1000 dpi.
TIFF: Combinations bitmapped line/half-tone (colour or greyscale): a minimum of 500 dpi is required.
DOC, XLS or PPT: If your electronic artwork is created in any of these Microsoft Office applications please supply "as is".

Line drawings Supply high-quality printouts on white paper produced with black ink. The lettering and symbols, as well as other details, should have proportionate dimensions, so as not to become illegible or unclear after possible reduction; in general, the figures should be designed for a reduction factor of two to three. The degree of reduction will be determined by the Publisher. Illustrations will not be enlarged. Consider the page format of the journal when designing the illustrations. Photocopies are not suitable for reproduction. Do not use any type of shading on computer-generated illustrations.

Photographs (halftones) Please supply original photographs for reproduction, printed on glossy paper, very sharp and with good contrast. Remove non-essential areas of a photograph. Do not mount photographs unless they form part of a composite figure. Where necessary, insert a scale bar in the illustration (not below it), as opposed to giving a magnification factor in the legend. Note that photocopies of photographs are not acceptable.

Copyright Upon acceptance of an article, authors will be asked to sign a ?Journal Publishing Agreement?? (for more information on this and copyright see http://www.elsevier.com/copyright). Acceptance of the agreement will ensure the widest possible dissemination of information. An e-mail (or letter) will be sent to the corresponding author confirming receipt of the manuscript together with a 'Journal Publishing Agreement? form or a link to the online version of this agreement.
If excerpts from other copyrighted works are included, the author(s) must obtain written permission from the copyright owners and credit the source(s) in the article. Elsevier has preprinted forms for use by authors in these cases: contact Elsevier?s Rights Department, Oxford, UK: phone (+44) 1865 843830, fax (+44) 1865 853333, e-mail permissions@elsevier.com. Requests may also be completed online via the Elsevier homepage (http://www.elsevier.com/locate/permissions).

Proofs When your manuscript is received by the Publisher it is considered to be in its final form. Proofs are not to be regarded as 'drafts'. One set of page proofs will be sent to the corresponding author, to be checked for typesetting/editing. No changes in, or additions to, the accepted (and subsequently edited) manuscript will be allowed at this stage. Proofreading is solely your responsibility. The Publisher reserves the right to proceed with publication if corrections are not communicated. Return corrections within 3 days of receipt of the proofs. Should there be no corrections, please confirm this.

OffprintsTwenty-five offprints will be supplied free of charge. Additional offprints and copies of the issue can be ordered at a specially reduced rate using the order form sent to the corresponding author after the manuscript has been accepted. Orders for reprints (produced after publication of an article) will incur a 50% surcharge.

NIH voluntary posting policy
US National Institutes of Health (NIH) voluntary posting (" Public Access") policy
Elsevier facilitates author response to the NIH voluntary posting request (referred to as the NIH "Public Access Policy", see http://www.nih.gov/about/publicaccess/index.htm) by posting the peer-reviewed author's manuscript directly to PubMed Central on request from the author, 12 months after formal publication. Upon notification from Elsevier of acceptance, we will ask you to confirm via e-mail (by e-mailing us at NIHauthorrequest@elsevier.com) that your work has received NIH funding and that you intend to respond to the NIH policy request, along with your NIH award number to facilitate processing. Upon such confirmation, Elsevier will submit to PubMed Central on your behalf a version of your manuscript that will include peer-review comments, for posting 12 months after formal publication. This will ensure that you will have responded fully to the NIH request policy. There will be no need for you to post your manuscript directly with PubMed Central, and any such posting is prohibited.

Appendix C

School of Psychology Ethics Committee Approval Letter

UNIVERSITY OF
Southampton
School of Psychology

28th April 2008

Confirmation of Ethical Approval

I hereby confirm that Rebecca Lee's study "Information Processing Biases in Anxiety" received ethical approval from the School of Psychology's ethics committee on 04th April 2007, reference CLIN/04/46.

Barbara Seiter

Academic Administrator
School of Psychology
Direct tel: +44 (0)23 8059 5578
email: bs1c06@soton.ac.uk

School of Psychology, University of Southampton, Highfield Campus, Southampton SO17 1BJ United Kingdom

Appendix D

Information Sheet for Participants

Date: 20.8.07. Version 2

Information Processing Study (Control Participants)

Processing of Emotional Information in Anxiety
Information Sheet for Research Participants

You are being invited to take part in a research study. Before you decide whether or not to take part, it is important for you to understand why we are conducting this research and what it will involve. Please take time to read the following information carefully and discuss it with family and friends if you wish. Do not hesitate to ask us if there is anything that is not clear, or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

Who are we?
The research team consists of Rebecca Lee and Caroline Gamble (Trainee Clinical Psychologists), Dr Matt Garner (Lecturer in Psychology), Dr David Baldwin (Honorary Consultant Psychiatrist / Reader in Psychiatry) and Professors Karin Mogg and Brendan Bradley (Professors of Psychology). This research project is being undertaken by Rebecca and Caroline for their dissertations, as part of the Doctorate in Clinical Psychology training course.

What is the purpose of this study?
This study aims to develop a better understanding of the relationship between emotions, thinking and attention to different types of information. You have been chosen because we need to study the responses of a sample of the general population and compare these to the responses of people with high levels of anxiety and their non-anxious first degree relatives. Specifically, we are looking for volunteers who have not experienced, or required treatment for, psychiatric problems in the past.

Do I have to take part?
It is up to you to decide whether or not you wish to take part. If you do decide to take part you will be given this information sheet to keep and asked to sign a consent form. Your participation is voluntary and you may withdraw your participation at any time. If you are a student in the School of Psychology and you choose not to participate, there will be no consequences to your grade or to your treatment as a student in the School. If you have any questions please ask a member of the research team on your first meeting, or contact us at rjl205@soton.ac.uk or cjg105@soton.ac.uk.

What will happen to me if I decide to take part?
You will initially be asked to come for a screening interview to check your suitability for the study and this should take approximately 30 minutes.

If applicable, you will then be asked to return for another session, which will last between 75 and 90 minutes. In this testing session you will be asked to complete a number of computer tasks and questionnaires. One task will involve you looking at a series of faces on a computer screen and classifying the emotion shown on the face using the keypad. Another task will involve you observing a number of pictures of faces on the computer screen, and responding when you see a specific marker on the screen. During this task your eye movements will also be monitored.

The final computer task involves looking at pictures while sounds are presented through headphones. Throughout this task various physical responses (e.g. skin conductance, heart rate and muscle tension) will be measured. This will involve placing 6 small electrodes on your skin (2 electrodes on two of your fingers, 1 electrode on each wrist, and two electrodes just beneath one of your eyes). These electrodes have a comfortable plastic case and allow us to monitor changes in heart rate, muscle tension and skin conductance.

In addition, we will ask you whether we can take a sample of saliva from your mouth, which would allow us to look at small specific parts of your DNA (genes). We would only examine your DNA in order to see if you have a certain type of gene that has been shown to influence results on the computer tasks that you will be asked to complete. For this reason, we will be unable to give you any information about your DNA. Your sample is kept anonymous and once it has been tested it will be destroyed.

In approximately 8 weeks time we will invite you back to repeat the computer tasks described above. This will again take about 75-90 minutes.

Your travel expenses for all appointments will be reimbursed.

Will my taking part in this study be kept confidential?
All information that is collected about you during the course of the research will be kept strictly confidential and your name will not be used when analysing the data obtained. Personal information will not be released to or viewed by anyone other than researchers involved in this project.

What will happen to the results of the research?
A report of the findings of the study will be written and useful findings will be submitted for publication in scientific journals. Results of this study will not include your name or any other identifying characteristics.

A summary of the results will be made available on request.

Who is organising the research?
This study is being organised by the University of Southampton.

Who has reviewed the study?
The National Research Ethics Service (Oxford Panel C) and the University of Southampton School of Psychology Ethics Committee have both reviewed the study.

Who can I contact?
If you have questions about your rights as a participant in this research, or if you feel that you have been placed at risk, you can contact the Chair of the Ethics Committee, Department of Psychology, University of Southampton, Southampton, SO17 1BJ. Phone: (023) 8059 3995.

Appendix E

Consent Form

Participant Identification Number for this Study:

Statement of Consent

I ______________________ have read the attached information sheet.
[participant's name]

Please initial box

1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions. ☐

2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. ☐

3. I agree to have a saliva sample taken to obtain DNA for use in this research project only. I understand that my saliva sample and extracted DNA will be destroyed at the end of the study. I understand that non-agreement does not exclude me from completing the computer tasks/questionnaires. ☐

4. I agree to take part in the above study. ☐

______________________	______________	______________
Name of Participant	Date	Signature
______________________	______________	______________
Name of Researcher	Date	Signature

1 for participant; 1 for researcher;

Appendix F

Debriefing Sheet

Participant Debriefing Sheet

Processing of Emotional Information in Anxiety

Debriefing Sheet

You have just taken part in a study designed to measure how mood affects what we notice and pay attention to, and how we interpret ambiguous information. We were interested to know how this was different when you were shown emotional pictures e.g. happy, angry or expressionless faces in the computer tasks.

Our mood can change from day to day, however, for some people, their thoughts or feelings may trouble them on a more regular basis. If you found any of the questions you were asked distressing, there are several sources of advice which are available and which may prove helpful in dealing with these feelings. These include Dr David Baldwin, Honorary Consultant Psychiatrist at the Department of Psychiatry and your General Practitioner.

We hope that our results will help us to better understand how mood affects attention and interpretation, and therefore also thinking and judgement. This in turn may be useful for the future development of strategies to help change the patterns of attention and interpretation that are thought to contribute to and maintain high levels of anxiety.

Please feel free to ask questions or make comments on any aspect of this study.

Thank you for your help.

If you have any further questions please contact Rebecca Lee (rjl205@soton.ac.uk) or Caroline Gamble (cjg105@soton.ac.uk).

Caroline Gamble and Rebecca Lee,
Department of Clinical Psychology,
School of Psychology,
University of Southampton,
SO17 1BJ