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# COGNITIVE BIAS IN GERNERALISED ANXIETY DISORDER AND ITS RELATIONSHIP WITH THE EFFECT OF SSRI TREATMENT.

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**Cognitive bias in Generalised Anxiety Disorder and its relationship with the effect of Selective Serotonin Re-uptake Inhibitor (SSRI) treatment.**

## Abstract

The literature review examines the nature of Generalised Anxiety Disorder (GAD) with particular reference to cognitive models of anxiety, such as those proposed by Beck, Emery & Greenberger (1986) and Bower (1981). More recent cognitive theories of anxiety, such as those proposed by Williams, Watts, MacLeod and Mathews (1988, 1997) and Mogg & Bradley (1998) are also reviewed. These latter models suggest that either anxious mood is elicited by vigilance for threat stimuli (Williams et al., 1988, 1997) or that anxious individuals evaluate stimuli as threatening (Mogg & Bradley, 1998). The predictions made by these studies are examined with the empirical findings reported within the literature. The role of treatment in removing cognitive bias is also examined, and conclusions concerning the nature of cognitive bias in GAD are drawn.

The empirical study contains two sections. The first section is a longitudinal study that examines whether the level of cognitive bias before treatment can be used as an index to predict the level of anxious mood following treatment with Selective Serotonin Re-uptake Inhibitor (SSRI) medication. This part of the study also examines whether medication alters cognitive bias. The results suggest that neither attentional bias nor interpretive bias is a reliable predictor for the level of anxious mood following SSRI treatment. The SSRI medication did not appear to alter the level of attentional bias, but there was a significant reduction in the level of interpretive bias following treatment. The second part of the study, the cross-sectional part, examines whether there was a significant difference in the level of attentional and interpretive biases between the GAD group and a control group. The results suggest that there were significant differences in the level of interpretive bias between the two groups, but not in the level of attentional bias. These results are discussed and suggestions for further study identified.

# TABLE OF CONTENTS

**List of figures**..... vi

**List of tables**..... vi

**Acknowledgements**..... vii

**Literature Review: Cognitive bias in Generalised Anxiety Disorder**

**Abstract**..... 2

**Generalised Anxiety Disorder**

Definitions of Generalised Anxiety Disorder (GAD)..... 3

Prevalence..... 4

Co-morbidity..... 5

Age of onset..... 5

GAD as a disorder of worry..... 5

**Cognitive models of Anxiety**

Beck, Emery & Greenberger's schema model of anxiety..... 7

Bower's network theory..... 8

Williams, Watts, MacLeod & Mathews' Interactional model..... 10

Mogg & Bradley's Cognitive-motivational model..... 13

Summary..... 15

**Evidence of cognitive biases in Generalised Anxiety Disorder**

Evidence for an interpretive bias within anxiety..... 17

Summary of interpretive bias..... 20

Evidence for an attentional bias in anxiety..... 21

Attentional bias measured by modified Stroop task..... 21

Summary..... 23

Attentional bias measured by the visual probe task..... 24

Attentional bias for threat stimuli as a function of time.... 26

The vigilance-avoidance debate..... 28

Attentional bias for stimuli presented outside of conscious awareness..... 29

Content specificity of attentional bias in supra- and subliminal conditions..... 31

**Clinical implications of cognitive models**

Treatment of Generalised Anxiety Disorder

Cognitive-behavioural treatment..... 32

Summary of cognitive-behavioural treatment..... 34

Pharmacological treatment

Benzodiazepine treatment..... 35

Selective serotonin re-uptake inhibitor treatment..... 38

Summary of pharmacological treatment..... 40

**Conclusions**..... 41

**References**..... 44

**Empirical paper: Cognitive bias in Generalised Anxiety Disorder and its relationship with the effect of treatment**

**Abstract**..... 51

**Introduction**..... 52

**Method**

Design and overview..... 60

Participants..... 61

Materials

Diagnostic measures ..... 63

Self report measures..... 64

Materials for measuring cognitive bias..... 65

Procedure..... 68

**Results**

Longitudinal study results..... 75

Cross-sectional study results..... 79

**Discussion**..... 80

**Conclusion**..... 87

**References**..... 88

**Appendices**

**Appendix 1:** Notes to contributors – Behaviour Research & Therapy.... 101

**Appendix 2:** University of Southampton, Southampton &
S.W. Joint Ethics Committee Approval for Research..... 102

**Appendix 3:** Press Release..... 103

**Appendix 4:** Homophone stimulus words..... 104

**Appendix 5:** Modified Stroop stimulus words..... 105

**Appendix 6:** Participant information for GAD patients..... 106

**Appendix 7:** Consent form for GAD patients..... 107

**Appendix 8:** Information sheet and consent form for control participants..108

**Appendix 9:** Hamilton Anxiety Scale..... 109

**Appendix 10:** Hamilton Depression Scale..... 110

**Appendix 11:** Spielberger State-Trait Anxiety Inventory..... 111

**Appendix 12:** Penn State Worry Questionnaire..... 112

**Appendix 13:** Beck Depression Inventory..... 113

**Appendix 14:** Mogg & Bradley (1993) Worry scales..... 114

**Appendix 15:** Marlowe-Crowne Social Desirability scale..... 115

## List of Tables

**Table 1:** Paired t-Test comparing mood measures from Time 1 to Time 2..... 92
**Table 2:** Pearsons Correlation showing relationship between cognitive bias at Time 1 and Mood at Time 2..... 93
**Table 3:** Pearsons Correlation showing relationship between mood at Time 1 predict mood at Time 2..... 94
**Table 4:** Mean cognitive bias scores from Time 1 to Time 2 for GAD patients.. 95
**Table 5:** Demographic information for GAD and control groups..... 96
**Table 6:** Mood characteristics of GAD group and control group..... 97
**Table 7:** Characteristics of GAD patients and controls for cognitive bias..... 98

## List of Figures

**Figure 1:** Comparison of homophone bias between GAD patients and controls.. 99

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# Cognitive bias in Generalised Anxiety Disorder

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# Cognitive bias in Generalised Anxiety Disorder

## Abstract

This literature review examines the nature of Generalised Anxiety Disorder (GAD) in relation to various cognitive models, such as those proposed by Beck, Emery & Greenberger (1986) and Bower (1981). The review identifies the strengths and limitations of these models and it discusses the contributions that have been made by more recent cognitive theories of anxiety, such as those proposed by Williams, Watts, MacLeod and Mathews (1988, 1997) and Mogg & Bradley (1998). These latter models of information processing suggest that anxiety arises either as a result of attentional biases towards threat (Williams et al., 1988, 1997) or that anxious individuals have a bias to over-estimate the threat value of stimuli (Mogg & Bradley, 1998). This review examines the evidence contained in various empirical studies, which have sought to test the predictions made by these models.

Since cognitive models of anxiety propose that cognitive bias plays a role in the development and maintenance of anxious mood, this review examines whether treatment of GAD is associated with a reduction in the level of cognitive bias. The findings from these studies and their implications for cognitive models of anxiety are discussed.

*Keywords:* Generalised Anxiety Disorder; interpretive bias; attentional bias; treatment

## Introduction.

*Definition.*

Generalised Anxiety Disorder (GAD), as defined by the American Psychiatric Association Diagnostic Statistical Manual 4th Edition (DSM-IV; APA, 1994), is a condition characterised by excessive and uncontrollable worry accompanied by physical symptoms of anxiety. Individuals with GAD have usually experienced excessive worry which occurs on the majority of days over a period of at least six months, and which is associated with a range of events or activities unrelated to another Axis I disorder. Once the worry has been elicited, the intensity and excessive nature of the worry causes significant distress or impairment in social or occupational functioning, resulting in the individual experiencing at least three of the following symptoms: fatigue; difficulty in concentration; irritability; muscle tension; feeling restless or keyed up, and difficulties in sleeping.

Unlike other disorders, such as specific phobias, the generalised nature of GAD anxiety is not elicited by discrete or easily identifiable environmental stimuli, nor is it brought about by panic attacks. Instead, individuals with GAD report pervasive worry across a wide range of seemingly unrelated situations and this sometimes makes it difficult to recognise the disorder. DSM-IV (APA, 1994) suggests that GAD may initially present in a clinical setting as an inability to relax, muscle tiredness, or feeling on edge. The worry component may be difficult to detect in routine medical practice, but problematic worry is seen as the key feature of the disorder. Since there are often a wide range of concerns within GAD, the

disorder has sometimes been conceptualised as being a disorder of 'free-floating anxiety'.

Conceptualisations of GAD have also been drawn from wider, more diverse, literatures which suggest that highly anxious individuals are likely to have high levels of state and trait anxiety (Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983). Spielberger et al. (1983) suggest that state anxiety is a relatively variable mood state, which reflects the demands of a given situation. On the other hand, trait anxiety is considered to be:

*'A relatively stable individual difference in the disposition to perceive a wide range of situations as threatening or dangerous'* (Spielberger et al., 1983; pp 39).

Individuals within the normal population who have high trait anxiety are considered to have a tendency to respond in an anxious manner towards potentially threatening situations. High levels of trait anxiety have been identified as being a risk factor for developing clinical anxiety, when the individual is under stressful situations (Williams et al., 1988). This vulnerability factor has led Rapee (1991) to suggest that GAD represents the extreme end of high trait anxiety spectrum.

*Prevalence.*

GAD is a common and long-standing condition (Mahe & Balough, 2000) and is thought to affect around 3% of the adult population which increases to 5% for the lifetime prevalence (DSM-IV; 1994).

*Co-morbidity.*

It is estimated that around eighty-two percent of GAD cases meet the diagnostic criteria for other anxiety or depressive disorders (Brown & Barlow, 1992), with social and simple phobia being the most common comorbid anxiety disorders. Around 42 percent of GAD patients also meet the diagnostic criteria for major depression (Brawner-Mintzer, Lydiard, Emmanuel, Payer, Roberts, Jarrel et al., 1993), with co-morbidity often resulting in greater levels of disability and associated functional impairment (Wittchen, et al., 2000; Kessler, Dupont & Berglund, 1999).

*Age of onset.*

The age of onset of GAD differs from that of other anxiety disorders, with the majority of cases first presenting aged 35-45 years (Wittchen, Carter, Pfisster, 2000), although the gradual onset of the anxiety problems experienced in GAD means that eighty percent of GAD patients are unable to recall a specific onset (Rapee, 1991).

*GAD as a disorder of worry.*

Borkovec & Inz (1990); Carter, Johnson & Borkovec (1986) have defined the essential features of worrying in GAD as being uncontrollable, verbal, negative and affect-laden, which are aimed primarily at problem solving. Thus, worry can be thought of as a conscious and attention-oriented process, with patients believing that worry can positively avert future possible threat (Carter et al., 1986).

The content of worries within GAD have been found to be associated with a range of day-to-day issues, such as health, finance and inter-personal relationships as well as minor adversive consequences (Butler, Gelder, Hibbert, Cullingham & Klimes, 1987). Although the focus of these worries are in themselves nothing unusual, the subsequent intensity and the level of uncontrollability are considered to be the pathological elements within GAD (Tallis, Davey, & Capuzzo, 1994). Since GAD is characterised by exaggerated or unrealistic concerns, cognitive formulations of GAD are well placed to identify the distorted information processing that is hypothesised to be associated with such an anxious state. Cognitive models of anxiety have therefore sought to understand the cognitive processes involved within GAD, which have subsequently provided useful understandings to explain how these states may be maintained over time.

This review will now turn to examine four such cognitive models.

## Cognitive models of anxiety.

The cognitive models of anxiety contained within the literature seek to explain the processes involved with the aetiology and maintenance of anxious cognitions within such individuals. Aetiological factors within this part of the literature refer to refer to the individual's style of *apprasing* incoming stimuli. Thus, once a stimulus has been evaluated as threatening then this will interrupt subsequent attentional processes. The maintenance of anxious mood is thought to occur when attentional processes are subsequently *directed* towards detecting further threat stimuli, which maintains anxious mood. This section will examine

four cognitive models that are considered to be of importance within the information processing literature.

**Beck, Emery & Greenberger (1986) Schema Model of Anxiety.**

Beck et al., (1986) suggest that the individual has cognitive representations of propositions, rules, assumptions and formulae concerning themselves, which are organised into schemas. Schemas are considered to be functional structures of prior knowledge which are held within long-term memory, which subsequently guide the screening, encoding, storing of incoming information, as well as providing cues for retrieving stored information (Beck & Clark, 1988). This schema-based informational processing model proposes that schemas are organised into groups that reflect the broad motivational interests of the individual. The theory suggests that anxious individuals are characterised by danger schemata which causes information to be interpreted as threatening or dangerous. This biased interpretation subsequently causes the individual to overestimate potential danger which leads to the belief that the world is a dangerous place.

The central tenet of the Beck et al (1986) model suggests that anxious individuals erroneously interpret information as bearing significant threat to individual well-being. Thus, the model proposes that the difference between normal and pathological anxiety is that individuals with pathological anxiety consistently overestimate threat within the environment, whereas non-anxious individuals hold more accurate perceptions of threat. Beck et al. suggest that along with over-estimating potential threat, anxious individuals also under-estimate personal coping resources within such situations. This leads to the development of anxious mood.

Consequently the Beck et al (1986) model suggests that both interpretation and coping biases serve to compound the individual's anxious mood, which lead to the maintenance of anxious mood. Beck et al., suggest that anxious mood is associated with cognitive distortions, fear-related beliefs, threatening images and automatic thoughts. Beck et al., (1986) propose that the biased appraisal of information as threatening is a factor that elicits a much wider response, which includes affective, physiological and behavioural components. These factors lead the individual to prepare for the fight or flight response.

In summary, the Beck et al (1986) cognitive model of anxiety proposes that anxious individuals demonstrate a tendency to erroneously attach threatening interpretations to innocuous stimuli within the environment. The central concept within this theory rests on the notion that danger schemata filter new information in a biased manner which then leads to the elicitation and maintenance of anxious mood. Thus, this model predicts biases throughout information processing in GAD, including attention, interpretive and memory biases which favour threat-related material. However, evidence within the experimental literature suggests that anxiety disorders are characterised by attentional biases (e.g. Mathews & MacLoed, 1985), but not memory biases (e.g. MacLeod, Mathews & Tata, 1986).

**Bower's (1981) Associative Network Theory.**

Bower (1981) proposes an associative network theory which suggests that once a certain affective state is elicited, then a wide network of corresponding representations become active. The theory suggests that cognitions and emotions are represented as nodes within a memory system, and that emotion-relevant

information is activated when the individual experiences a particular affective state. The model suggests that the network of interconnecting representations become active when a particular mood is experienced, thus representations of threat or sadness are activated when anxious or depressive mood is elicited, respectively. The theory suggests that old associations are recalled faster and new representations become stored more easily as they become congruently encoded with the present emotional state. This spreading activation within the memory network consequently affects perceptions and evaluations, causing new information to be encoded in a biased manner that is consistent with the active network of representations.

An advantage of Bower's (1981) model is that other researchers have confirmed that mood-congruent information is more likely to be recalled, particularly when the affective level is high (e.g. Eich, 1995). However, the limitations that apply to the Beck et al., model also apply to Bower's model, inasmuch that evidence exists for attentional biases within anxiety, but there is little supporting evidence for memory biases within anxious individuals. Although the Bower (1981) model proposes a mechanism for the maintenance of anxious or depressed mood over time, a potential disadvantage is that it does not specify the aetiological mechanism associated with anxious/depressed mood in the first instance. It therefore appears that this model could be strengthened further by being able to account for these processes.

More recent models of information processing in anxiety and depression have sought to further identify the mechanisms involved in production in these

mood states, such as the Williams et al. (1988, 1997) and the Mogg & Bradley (1998) models. This review will now turn to examine these models in turn.

**The Integrative Model (Williams, Watts, MacLeod & Mathews; 1988, 1997).**

Williams et al (1988, 1997) propose that anxious individuals are vigilant for threat within the environment. The difference between this model and the schema model proposed by Beck et al (1986) is that this model proposes that a number of important cognitive functions take place before the information reaches conscious awareness. Williams et al (1988, 1997) suggest that highly anxious individuals demonstrate a pre-conscious bias for negative information, which means that anxious individuals perceive and classify stimuli as threatening, even before the stimuli have entered conscious awareness. This bias in early-stage processing is considered to be aetiological in the development of anxious mood. Thus, Williams et al. (1997) suggest that the Affective Decision Mechanism (ADM) is responsible for determining the threat-value of the incoming stimuli at the preconscious stage. This mechanism is thought to be heavily influenced by the level of anxiety that the individual is experiencing at the particular time (e.g. level of state anxiety). If the individual is in a highly anxious state, then the ADM is more likely to attach or 'tag' a high threat value to new incoming stimuli. Stimuli that are assigned such tags are identified by the Resource Allocation Mechanism (RAM) which controls attention. The RAM is therefore responsible for deciding whether the individual's attention should remain on their current task or whether it should be interrupted and reallocated to the source of the perceived threat. The RAM is proposed to be affected by high levels of trait anxiety (Spielberger et al., 1983), which is

considered to be an enduring characteristic of the individual. According to Williams et al., (1988, 1997) high levels of trait anxiety are associated with an over-active RAM, which causes attention to be disproportionately allocated to threat cues. The allocation of attention towards threat cues is proposed to maintain anxious mood, since the individual continues to detect threatening stimuli within the environment. Consequently, a high degree of information perceived to be threat-related is more likely to enter into conscious awareness. Williams et al. therefore propose that these mechanisms are faulty within anxious individuals, which causes biases in selective attention to threat.

The Integrative model suggests that when individuals are not in a state of high anxiety, then the ADM is less likely to evaluate stimuli as threatening. On these occasions the ADM does not issue a high-threat tag, which means that the RAM does not interrupt the individual's attention, leaving the individual free to focus upon their original task. In fact Williams et al (1988) suggest that on these occasions, the RAM might actually divert the individual's attention away from the location of the minor threat towards more neutral or positive stimuli, which is likely to serve as a protective factor for the maintenance of positive mood. in addition, they propose that low trait anxious individuals have a permanent tendency to direct attention away from threat cues and that this avoidance increases as state anxiety or threat value increases.

Williams et al. (1988, 1997) propose that the cognitive processes underlying anxiety differ from those in depression. They suggest that depressed mood is associated with some of the same mechanisms associated with anxiety, in that the ADM assesses the level of negativity (rather than threat). Similarly the RAM then

allocates processing resources towards the origin of the negative stimuli, causing greater elaboration and maintenance of depressed mood. The Williams et al. model suggests that depressed mood is mainly characterised by a bias in the elaboration process in memory, rather than in the allocation of attention towards negative stimuli. Subsequently, it is proposed that negative representations are stored and accessed more easily, resulting in depressed individuals recalling more negative memories (e.g. Teasdale & Fogarty, 1979) and this is thought to maintain depressed mood. The Williams et al (1997) model differs from Beck et al.'s (1986) and Bower's (1981) models, which suggest that there is an emotion-congruent attentional bias for threat and negative stimuli within anxiety and depression respectively. However, the Williams et al (1997) and Mogg & Bradley (1998) models distinguish between attentional bias for anxiety and memory bias for depression.

In summary, the Integrative model suggests that anxious and depressed moods are characterised by different cognitive processes, which are associated with attention and elaboration biases. The key difference between this model and the Beck et al (1986) model is that the former hypothesises that attentional bias causes and maintains anxious mood whereas Beck et al. (1986) suggest that there is a general bias within all parts of processing, be it within attention or memory. The evidence supporting these notions will be examined later. A further potential strength of the Williams et al model is that it defines the differences associated with pre-and post- conscious processing and how these specific processes are associated with the aetiology and maintenance of anxious mood. However, there is a potential limitation to the model concerning the notion that low-anxious individuals orient

their attention away from the source of the threat. Since anxiety is considered to have an evolutionary function (Öhman, 1986), the model does not take account of how such avoidance might become maladaptive if the severity of the threat were to increase, since continual avoidance shown in low trait anxious individuals could result in injury or death. Thus, this model seems to apply mainly to very mild threat stimuli (e.g. processing of threat words) but not the more real-life threats.

**The Cognitive-motivational model of anxiety (Mogg & Bradley, 1998).**

Mogg & Bradley (1998) suggest that vigilance towards threatening stimuli has an evolutionary function, since this helps the individual to detect potential sources of threat within the environment. When such threat has been detected, anxiety can be perceived as motivating the individual to take appropriate action to ensure survival.

Mogg & Bradley (1998) suggest that there are two mechanisms associated with the aetiology of anxious and depressed mood, which are described as the Valence Evaluation System and the Goal Engagement System. Firstly, it is suggested that Valence Evaluation System (VES) is concerned with assessing the level of threat associated with incoming stimuli. This system is concerned with a very rapid registration of threat, which can occur outside of awareness. This system operates to quickly evaluate the valence of stimuli within the environment and it determines whether stimuli present a general threat to the individual. The distinction between normal and pathological anxiety is thought to occur as a result of the VES over-estimating the threat value of stimuli. Mogg & Bradley (1998) suggest that individual differences in the reactivity of the VES are associated with

levels of trait anxiety, with such individuals being more likely to perceive relatively harmless stimuli within the environment as being threatening. Once the VES has evaluated the level of threat, the Goal Engagement System (GES) then responds accordingly. For example, when information is appraised as having high threat value, the GES interrupts attention focused towards current goals and orients attention towards the threat, which maintains anxious mood. On the other hand, if the threat is low then the GES does not interrupt attention, allowing the individual to avoid attending to mild threat. The cognitive-motivational account of anxiety suggests that valence evaluation and subsequent goal engagement is a normal and adaptive process. Pathological anxiety, however, is thought to occur when the GES becomes falsely activated by stimuli that have being incorrectly labelled as threatening.

According to the cognitive-motivation model, the reason why depressed individuals often fail to show an attentional bias in the same manner as anxious individuals is because the Goal Engagement System operates differently within this disorder. Mogg et al. (1998) suggest that depression (or anxiety with comorbid depression) is characterised by low levels of external goal engagement which lowers the vigilance for external threat cues. Consequently, individuals with depression, with or without anxiety, may fail to show a bias for external threatening stimuli.

In summary, the Mogg & Bradley cognitive-motivational account provides a useful framework to consider the mechanisms involved within the aetiology and maintenance of anxious and depressed mood. The model suggests that anxious individuals have a bias for evaluating stimuli in a threatening manner in the first

instance, when compared to non-anxious individuals. The advantage of the Mogg & Bradley (1998) model over the Williams et al.'s model is that it is clearly couched within the context of an evolutionary framework. This evolutionary perspective also explains the function of this motivational state, which demonstrates the almost inextricable link between anxiety and human experience. A second strength of the Cognitive-motivational model offers a hypothesis to why differences in attention occur between anxiety and depression, whereas the Williams et al.'s (1988, 1997) model does not seem to offer such insight.

Both the Williams et al.'s (1988, 1997) and the Mogg & Bradley (1998) model draw upon conceptualisations of state and trait anxiety, with the latter being identified as a key factor in contributing to pathological anxiety states. The two models differ at the stage at which trait anxiety is proposed to have its effect since Williams et al., suggest that once threatening information has been identified, trait anxiety influences the *direction* of attention, with high-trait anxious individuals directing attention towards the threat and low-trait anxious individuals directing their attention away from the threat. The Mogg & Bradley model, however suggests that trait factors are primarily associated with the initial *appraisal* of the stimuli as being threatening in the first instance.

**Section summary.**

The four models discussed within this section have sought to explain the mechanisms involved in the elicitation and maintenance of affective states, particularly those of anxiety and depression. Beck et al., (1986) schema model

suggests that, in anxiety, information is screened and encoded by danger schema for threat cues. The Bower (1981) network theory suggests that networks of cognitive representations of emotion-related information become activated once an individual is in an anxious state. Thirdly, the Integrative model (Williams et al., 1988, 1997) suggests that high state anxiety influences the direction of initial attention, towards the source of threat. The Cognitive-motivational account of anxiety (Mogg & Bradley, 1998) suggests that highly anxious individuals evaluate stimuli as more threatening, which then has the effect of directing attention towards the potential threat cue.

The four cognitive models outlined have the central tenet that anxiety arises as a result of biased processing of threat stimuli including biases in appraisal and selective attention. However, each model proposes that this occurs in a slightly different manner. Thus, these four models predict that anxious individuals demonstrate an 'interpretation bias', and 'attentional bias' for threat cues. This literature review will now turn to consider the evidence for both of these types of bias in turn.

## Evidence of cognitive biases.

The literature contains widespread evidence which suggests that anxious individuals process information in a biased manner, which the literature refers to as 'cognitive bias'. This term can be further divided into two further terms: 'interpretive bias' and 'attentional bias'. 'Interpretive bias' refers the tendency of anxious individuals to selectively interpret ambiguous stimuli as threatening. Secondly, 'attentional bias' refers to the allocation of visual attention towards

threatening words or pictorial stimuli over simultaneously presented neutral/positive stimuli.

Since clinically anxious individuals are characterised by high levels of state and trait anxiety, there are advantages of examining some of the non-clinical studies with high trait anxious individuals, since these studies are able to disentangle the theoretical effects of state and trait anxiety upon cognitive bias. Thus, the following section will refer to both clinical and non-clinical studies.

**Evidence for an interpretive bias within anxiety.**

Given that normal daily living is associated with interpreting a range of impoverished or ambiguous stimuli, a continually threatening or negative interpretation of such stimuli is likely to cause and maintain anxious mood. The appraisal of stimuli as threatening is therefore a key factor within cognitive models of anxiety (e.g. Beck et al., 1986). In order to establish whether anxious individuals appraise and encode information in a more threatening manner, when compared to non-anxious controls, an homophone task has been devised by Eysenck, MacLeod & Mathews (1987). They presented a list of threat-related homophones words, which are words that share the same sound but have different meanings (e.g. : die/dye, moan/mown, groan/grown) to anxious and non-anxious individuals to examine whether, upon hearing the word, they wrote down the threatening or neutral version of the word. Eysenck et al., (1987) report that there was a significant correlation between the level of anxiety and the tendency to select the threatening version of the homophone. This finding suggests that anxious individuals encode their environment in a particularly threatening way, and this

finding is consistent with the Beck et al. (1986), Bower et al (1981) Eysenck (1997), Mogg & Bradley (1998) and Williams et al. (1997) models of information processing. Although this study yielded interesting results, some caution is advised since the study used a relatively small amount of participants (n=16), which limits the generalisability of the findings. A further limitation of this study is that it did not contain a measure of social desirability; individuals high in social desirability often under-report potentially negative results. A third limitation of the study is that since the study used anxious individuals it is difficult to establish whether an interpretive bias occurs as a result of high state anxiety or high trait anxiety. With these limitations in mind, Mathews, Richards & Eysenck (1989) examined this question further by using three groups of 14 subjects, which comprised of a clinically anxious group, a recovered-anxious group and a matched control group with no history of anxiety problems. The investigation sought to examine whether the tendency to select the more threatening interpretation of a homophone was a function of an enduring cognitive vulnerability to anxiety, or whether it related more closely to current anxious mood. The use of the recovered-anxious group enabled these various factors to be explored in greater depth and is seen as a particular strength of the investigation.

The results of the Mathews et al (1989) study suggest that, whilst controlling for social desirability, anxious individuals perceived significantly more threatening interpretations when compared to the non-anxious control group. This finding suggests that a clear interpretive bias exists within anxious individuals. Interestingly, the amount of threat interpretations reported by the recovered-anxious group was not significantly different from the non-anxious control group, which

suggests that interpretive bias might be associated with high levels of state anxiety. However, given that the level of interpretive bias in the recovered-anxious group was not significantly different from the highly anxious group either, it becomes difficult to draw definitive conclusions concerning the relative importance of high levels of trait and state variables.

Similar result from the homophone task have also been reported by Mogg, Bradley, Miller, Potts, Glenwright & Kentish (1994) who found that anxious individuals produce more threatening interpretations. Their analyses suggested that trait, rather than state, anxiety was responsible for this effect, although similar limitations to these results are highlighted given the difficulties of disentangling the effects of these two variables. Furthermore, Mogg et al. (1994) also report that interpretive bias was determined by levels of social desirability (study 1), but conflicting results are also presented and discussed from a further study (study 2), which reports that interpretive bias is associated with anxiety rather than social desirability.

Whilst acknowledging these potential limitations, a further study of interpretive bias has been reported by MacLeod (1990). Using the homophone task, MacLeod attempted to overcome the difficulties associated with disentangling state and trait variables by manipulating the level of state anxiety within high and low trait anxious individuals. MacLeod (1990) reports that the proportion of threat-related spellings in high trait anxiety individuals rose as a direct of function of increases in state anxiety. On the other hand, low state anxiety individuals were found to produce fewer threat related interpretations when state anxiety increased, which suggests that such individuals show an avoidance of threat when state

anxiety increases, which might serve as a protective mechanism in low trait anxiety individuals. Consequently, MacLeod (1990) proposes that cognitive bias arises as a result of an interaction between trait and state anxiety variables, with high trait anxious individuals being most at risk of developing an interpretive bias when their levels of state anxiety are also high.

Although the homophone task provides a useful insight into the nature of biased interpretation within anxious states, the literature does identify some limitations associated with it. The discrepant findings between the relative influences of state anxiety or social desirability lead Mogg et al (1994) to suggest that anxious individuals may have a tendency to report interpretations that are consistent with their mood at the time, or that their responses may be influenced by some kind of general response bias (e.g. to say negative words). A second criticism of the interpretive bias task is that some of the homophones are consistently spelt in a threatening manner by both anxious and non-anxious subjects. It appears that three particular homophones (guilt vs. gilt, skull vs. scull, and liar vs. lyre) are almost universally spelt in a negative manner amongst all individuals. Consequently, the over-reporting of the negative version of these words reduces the number of stimuli words that yield useful information from fourteen to eleven.

**Summary of interpretive bias.**

The evidence outlined above concerning interpretive bias has suggested that both clinically anxious and non-clinical individuals with high trait and state anxiety are significantly more likely to interpret ambiguous information in a negative manner. Evidence from Mathews et al (1989) suggests that currently anxious

individuals report more negative interpretations and this is also reported by Mogg et al (1994), although the effects of state and trait anxiety remain unclear. The work completed by MacLeod (1990) suggests that interpretive bias operates as a result of trait anxiety with the effects of this being amplified when the individual has high levels of state anxiety. The opposite pattern is thought to be true for low trait individuals. The task, however, does have some limitations with some authors suggesting that it is difficult to establish whether it is an interpretive- or response-bias that accounts for the results. The evidence for interpretive bias within anxious individuals appears to be consistent with the cognitive models of anxiety outlined previously, particularly those outlined by Beck et al., (1986), Bower (1981) and Mogg & Bradley (1998).

**Evidence for an attentional bias in anxiety.**

There is now considerable evidence within the literature that clinically anxious individuals selectively allocate processing resources towards threat stimuli (e.g.: Mathews & MacLeod, 1985; MacLeod, Mathews & Tata, 1986; Mogg, Mathews & Eysenck, 1992). These findings suggest that individuals with high levels of anxiety orientate their attention towards threat stimuli within the environment, significantly more than individuals with lower levels of anxiety. The consequence of such orienting is that such individuals attend more readily to environmental threat cues, which in turn, serves to maintain anxious mood.

**Attentional bias measured by the modified Stroop task.**

A number of studies within the literature have confirmed that anxious individuals preferentially attend to threatening stimuli. For example, the modified-Stroop task has been extensively employed within experimental psychopathology. Based on the findings of Stroop (1935), who suggested that individuals experience difficulties in naming the colour of ink that a word is printed when the content of the word refers to a different colour, the degree of disruption experienced within the task can be identified. For example, when the word 'red' is printed in blue ink the latency between stimulus onset and colour-naming can be taken as an indication of the degree of interference or distraction experienced. Using this finding, the Stroop task has been modified to detect whether colour-naming latencies occur when threatening words are shown to anxious individuals, when compared to neutral or positive words.

A number of studies using a modified version of the Stroop have indicated that anxious individuals experience disproportionate amounts of distraction, compared to individuals without anxiety problems. Individuals with GAD are reported to take longer to name the colours of threatening words (such as *cancer* or *collapse*), than neutral words (such as *carpet*) when compared to healthy controls (Mathews & MacLeod, 1985; Mogg, Mathews & Weinman, 1989; Mogg, Bradley, Williams & Mathews, 1993a; Mathews & Klug, 1993, Bradley, Mogg, Millar & White, 1995). These findings suggest that anxious individuals attend to threat information more readily than non-anxious individuals and such findings are consistent with cognitive formulations of anxiety (e.g.: Beck et al., 1986). However, Bradley, Mogg, Millar & White (1995) report GAD patients with

comorbid depression did not demonstrate an attentional bias for information presented on the modified Stroop task.

Since clinically anxious individuals are characterised by high levels of both state and trait anxiety levels (Spielberger et al., 1983), it is difficult to disentangle the relative effects of these variables on attentional bias. Consequently, some studies have examined non-clinical high trait anxiety individuals, when state anxiety has been variable. In an attempt to understand this relationship further, MacLeod & Rutherford (1992) examined high- and low-anxiety trait students on two occasions during the university term: once when exam stress was low and once when exam stress was high. MacLeod & Rutherford (1992) report that when exam anxiety was low, (and hence low levels of state anxiety), there were no differences in the level of attentional bias shown between high and low trait anxious students, with neither group showing attentional bias for exam-related threat words. However, when state anxiety was high, the high trait students demonstrated an attentional bias for exam-related threat words, compared to low-trait students, which suggests cognitive bias arises as a result of an interaction between high levels of state and trait anxiety.

**Summary.**

There is now extensive evidence within the literature that individuals with high levels of anxiety preferentially attend to threat stimuli over neutral stimuli. Consistent with cognitive theories of anxiety, these findings are taken to suggest that attentional bias plays an important part in the maintenance of anxious mood.

There is also some evidence to suggest that high trait non-clinically anxious individuals also show preferential attention for threat stimuli although differences between studies have emerged, since opposing data suggest that cognitive bias is either unaffected by levels of trait anxiety, or it is affected by state anxiety alone or is affected by a combination of state and trait anxiety.

**Attentional bias measured by the Visual-probe task.**

In contrast to the modified-Stroop task, where a delay in colour-naming is taken to indicate attentional bias, so the speed in which individuals respond to visual probes has also been taken as a marker of cognitive bias. The Dot-probe task, or as it has been more recently termed: the visual-probe task, has been adapted from an experimental psychology framework. The visual-probe task is based on the assumption that direction of an individual's attention can be measured from the speed at which individuals respond to visual probes that occur in various locations (MacLeod et al, 1986). The response speeds to probes will be shorter if they occur in an attended, rather than unattended area of the individual's visual field (Posner, Snyder & Davidson, 1980). Consequently, the allocation of attention towards particular stimuli can be measured by the speed at which the individual responds to probes that appear either in place of the attended stimulus, or the unattended stimulus.

Evidence from visual-probe studies suggest that GAD patients responded faster to probes that replace threat words rather than neutral words (MacLeod et al., 1986; Mogg et al 1992, 1995), which confirms the nature of biased processing within high levels of anxiety. Attentional bias has also been examined using

pictures of threatening faces, since such pictures are considered to be a particularly ecologically valid measures of attention., since there is virtually universal cross-cultural agreement on the six basic facial expressions of happiness, surprise, fear, anger, disgust and sadness (Ekman, Sorneson & Friesen, 1969; Ekman & Friesen, 1971). These findings suggest that facial expressions might have an important evolutionary function (Dimberg & Öhman, 1996). Consequently, there appear to be advantages to using such stimuli over word stimuli.

In an important paper, MacLeod, Mathews & Tata (1986) used 16 'generally anxious' patients and 16 matched controls in an attempt to measure the allocation of attentional resources. MacLeod et al (1986) presented pairs of negative and neutral words on a computer screen for 500 ms and the participants were required to detect a small probe that appeared in the location of one of the words following the ending of the display. MacLeod et al (1986) found that highly anxious individuals responded faster to the dot-probes that occurred in place of threatening words rather than neutral words. This finding suggests that highly anxious individuals direct their attention significantly more towards threat information which thereby intensifies anxious mood.

The results from several dot-probe studies that have used faces as stimuli have found that non-clinical anxious individuals respond faster to probes that replace threatening faces, rather than neutral or positive faces (Bradley, Mogg, Falla & Hamilton, 1998; Mogg & Bradley, 1999). On the other hand, individuals with low anxiety and depression attended to the positive and neutral faces more so than the threatening faces, which suggests that such individuals show an avoidance of such pictures (Bradley, Mogg, Miller, Bonham-Carter, Fergusson, Jenkins & Parr,

1997). Taken together, these results provide evidence for attentional bias for threatening faces in non-clinical high trait anxious individuals. Similar evidence for attentional bias towards threat faces has also been identified within GAD patients (Bradley, Mogg, White, Groom, de Bono, 1999; Mogg, Millar & Bradley, 2000), although GAD patients with comorbid depression did not show such attentional bias (Mogg, Miller & Bradley, 2000). These findings suggest that anxiety and depression are characterised by different types of information processing, as suggested by the Williams et al., (1988, 1997) model and the Mogg & Bradley (1998) model. Interestingly, the Bradley et al (1999) study suggests that GAD patients also demonstrated an attentional bias *towards* happy faces, during the second half of the task. It is not immediately apparent why this result should have emerged, since these findings are not predicted by either the Cognitive-motivational model (Mogg & Bradley, 1998), or the Integrative model (Williams, 1988, 1997). Bradley et al., (1999) suggest that this might occur as a result of anxious individuals using this strategy in order to regulate their mood. Another possibility for this result is based on the finding that the degree of positive bias was associated with the level of social anxiety, which suggests that socially anxious individuals might interpret an approaching, smiling individual to represent social threat.

**Attentional bias for threat stimuli as a function of time.**

The findings that individuals with GAD respond faster to probes that replace threat stimuli are consistent with an attentional bias oriented towards threat stimuli. The visual-probe studies have used different stimulus durations and revealed that individuals with clinical anxiety disorders are vigilant for threat

stimuli across stimuli durations of 500 ms (e.g. MacLeod et al 1986; Bradley et al, 1999), and at 1000 ms duration (Mogg et al., 1995). However, other studies report that attentional bias is not found at 1000 ms durations (Mogg, Miller & Bradley, 2000), nor at 1250 ms (e.g. Bradley et al, 1999). These discrepant findings have led to a closer examination of initial orienting and maintenance of attention upon threat stimuli. Using a method of monitoring the direction of actual eye movements, along with the normal manual reaction time procedure, Bradley, Mogg & Millar (2000) report that the direction of the initial shift in gaze was associated with the RT measure of attentional bias at the 500 ms duration, which suggests that the manual RT to visual probes is a valid index of initial orienting. The discrepant findings reported above for longer durations may have arisen as a result of multiple shifts in attention between the stimuli. Consequently, the individual might have initially directed attention to the threat stimulus, but shifted attention and may have been looking at the neutral stimuli towards the end of these longer stimulus durations. In an attempt to overcome these limitations, Mogg, et al. (2000) employed an eye movement tracking device, which determines direction of overt orienting.

Mogg et al. (2000) examined whether there were differences in attention bias for threatening faces in patients with GAD (without concurrent depression) and a bias for sad faces in patients with depression, by employing a visual probe task along with an eye-gaze tracking device. They report that patients with GAD were more likely to look initially at the threatening faces, when compared to the depressed group or the non-clinical control group, who did not demonstrate an attentional bias. Moreover, the GAD group shifted their gaze more quickly towards

the threat stimuli, compared to the other two groups. Interestingly, there was virtually no correlation (close to zero) between the data obtained at 1000 ms. Mogg et al. (2000) suggest that patients with GAD initially orient attention towards threat, but over a period of 1000 ms such individuals do not consistently focus their attention upon such threat, as indicated by manual responses to visual probes. These findings help differentiate initial orienting between the maintenance of attention upon stimuli exposed for longer.

**The vigilance-avoidance debate.**

The results from the above studies suggest that there is an attentional bias for threat information in anxious individuals, along with the lack of attentional bias within depressed individuals, which seems consistent with the Williams et al. (1997) and the Mogg & Bradley (1998) models of anxiety and depression.

The studies outlined within this section are also relevant to the proposal of a vigilance-avoidance pattern of attention, previously identified by Mogg, Mathews & Weinman (1989), and Williams, Watts, MacLeod & Mathews (1988). This suggests that anxious individuals initially direct their attention towards threat cues, but then avoid prolonged exposure to it in an attempt to regulate anxious mood. However, although the studies reviewed indicate an initial attentional bias to threat, there is, as yet, no evidence of avoidance. However, given the relatively limited range of findings using long stimulus durations, it remains unclear whether a vigilance-avoidance pattern of processing occurs within anxious individuals, or not. Consequently, the vigilance-avoidance debate appears to be an unresolved issue within the literature.

The discussion of attentional studies has thus far only examined stimuli that are presented within conscious awareness. Given that several models predict preconscious biases (e.g. Williams et al., 1988) it is interest to examine whether there are differences in the bias shown for stimuli shown outside of awareness. This literature review will now turn to consider this evidence.

**Attentional bias for stimuli presented outside of conscious awareness.**

There has been broad interest with perception outside of conscious awareness for a number of years (see Dixon, 1981 for a review of early preconscious processing studies). Dixon (1981) suggests that :

> 'since he (the participant) remains oblivious to the contingency between stimulus and response, he cannot avoid demonstrating even such contingencies as might reveal normally repressed aspects of psychopathology' (p168).

Thus, the presentation of stimuli at the subliminal level appears to serve two main purposes. Firstly, it helps to establish the stage of processing at which attentional biases occur, since it helps to disentangle the effects of conscious and effortful shifts in attention either towards or away from the threat stimuli. The response given to the presentation of subliminal material is therefore considered to be a measure of automatic processing that is outside of voluntary control (McNally, 1995).

Mathews and MacLeod (1986) suggest that the free-floating nature of anxiety seen within GAD may be associated with unconscious and automatic processing for threat cues within the environment, since generally anxious individuals might become anxious following the detection of threat that they had

detected outside of conscious awareness. Consequently, the trigger of the anxiety might not always be readily identifiable, which would lead the individual to experience seemingly unrelated feelings of tension and anxiety in a range of situations. It is therefore of interest to examine the role of preconscious processing when assessing attentional bias within anxious states. Before this notion is examined further, it is important to note that the terminology used within this part of the literature is diverse, with the terms: subthreshold, preconscious and subliminal being used interchangeably; this review will primarily use the latter term.

Various investigations have examined whether attentional bias can be detected for negative stimuli which is shown very briefly and then masked, which prevents conscious processing (subliminal). This question has been investigated using the Modified Stroop and the dot-probe tasks. Firstly, GAD patients have been found to have an attentional bias for negative words presented subliminally in the modified Stroop task. This task involves presenting a stimulus word for a duration of 14 ms, which is then immediately followed by a string of random letters which acts as a mask. Most individuals report only seeing the random letters and are not consciously aware of any stimuli that precede the mask, when given an awareness check. The results from various studies have reported that individuals with GAD experience disproportionate amounts of colour-naming distraction for words that are presented subliminally, even though they showed no evidence of awareness (e.g. Mogg, et al., 1993a; Bradley, et al., 1995). Findings of preattentive attentional bias have also been found in highly anxious non-clinical individuals (e.g. MacLeod & Rutherford, 1992; Mogg, Kentish, Bradley, 1993b; Mogg, Bradley, de Bono &

Painter, 1997). These results suggest that highly anxious individuals demonstrate an attentional bias for threat stimuli even thought they are unable to tell whether any stimuli have been presented. Mogg, Bradley, Williams & Mathews (1993a) report that the attentional bias shown by the GAD patients for subliminal stimuli was as strong as those for information shown supraliminally. These findings suggest that preconscious processing plays an important role in mediating initial orienting towards threat stimuli.

**Content specificity of attentional bias in supra- and subliminal conditions.**

Evidence from supraliminal studies suggests that the attentional bias shown towards particular stimuli is in keeping with the individual's primary concerns. For example, GAD patients who were primarily characterised by social concerns attended significantly more to social-threat related words, whereas those concerned more with physical concerns demonstrated a greater bias for physical-threat related words (Mogg et al., 1992). The finding that attentional biases are linked with the individual's primary concerns has also been identified within other anxiety disorders other than GAD, for example individuals with a spider phobia attended to spider-related words (Watts, Sharrock & Trezise, 1986; Mathews & Sebastian, 1992), social phobia patients attended to social-threat words (e.g. Mattia, Heimberg & Hope (1993) and panic patients attended to catastrophe words (e.g. McNally, Reimann, & Kim, 1990). These findings indicate that vigilance for threat in supraliminal studies is a specific process relevant to individual concerns.

The literature suggests that when similar material is presented subliminally, attentional bias within anxious individuals was found to be associated with

negative stimuli in general (e.g.: Mogg et al. 1993a; Mathews & MacLeod, 1994), rather than the specific content of the stimuli. These findings therefore suggest that a rather crude and preliminary level of information processing operates at the preconscious level to evaluate the valence of the stimuli.

The findings outlined above suggests that there is now considerable evidence for attentional bias within anxious mood states and this exists for both word and picture stimuli. The data also suggest that subliminal processing is associated with a basic evaluation of negative valence, whereas supraliminal processing is associated with a more refined and specific type of processing and this reflects the individual's person concerns.

## CLINICAL IMPLICATIONS OF COGNITIVE MODELS.

### Treatment of Generalised Anxiety Disorder: The effect on cognitive bias

Since cognitive formulations of anxiety have suggested that cognitive biases cause and maintain pathological anxiety, it is of interest to examine whether such biases are reduced by treatment.

### Cognitive-behavioural treatment of GAD.

The aim of cognitive-behaviour therapy (CBT) is to identify and modify dysfunctional beliefs and representations that are associated with the maintenance of anxious mood (e.g. Beck et al., 1986). Beck et al. (1986) suggest that such treatment can be undertaken by helping the individual to identify distorted cognitions that are associated with danger or risk. CBT proceeds by helping the individual to challenge these cognitions and to identify more accurate beliefs

instead. The more adaptive beliefs can be behaviourally tested and can ultimately be substituted for the dysfunctional belief itself. The cognitive model suggests that once these more adaptive beliefs are in place, then the individual will be able to appraise risk more accurately which will lead to the decline of anxious mood.

Mathews, Mogg, Kentish & Eysenck (1995) report that a seven session course of anxiety management training was effective in removing attentional bias within GAD patients. They report that the anxiety management training, which consisted of relaxation, cognitive coping strategies and graded exposure, was associated with a significant reduction of the level of bias shown towards threat related stimuli at both the supraliminal stage. This reduction in bias was also accompanied by a reduction in the level of anxious thoughts, along with an overall reduction in anxious mood. A follow-up study of GAD patients by Mogg, et al. (1995) twenty months later revealed that the effects of the treatment were still present and that there was no evidence of attentional bias. Similar results indicating the effectiveness of psychological treatment for removing the cognitive bias in other anxiety disorders have been reported, such as: social phobia (Mattia, Heimberg & Hope, 1993), spider phobia (Watts, McKenna, Sharrock & Trezise, 1986), and obsessive compulsive disorder (Foa & McNally, 1986). These results combine to suggest that psychological treatment is efficacious in removing cognitive bias.

These studies suggest that cognitive biases are amenable to change when treated with psychological intervention. However, since cognitive bias also occurs at the subliminal level, how does psychological treatment affect biases that occur outside of awareness? The evidence outlined above suggests that psychological

treatment is effective in removing subliminal biases (e.g. Mathews et al. 1995), although the mechanism associated with this change is unclear. Theoretically, it could be suggested that the change occurs as an indirect result of changes in state and/or trait variables, rather than the explicit challenging of anxious thoughts or beliefs. Since trait anxiety is conceptualised as a relatively enduring individual characteristic, the reduction in the level of state anxiety after treatment (Mathews et al., 1995) suggests that cognitive bias arises as an interaction between high trait and high state anxiety variables. It has therefore been suggested that both subliminal and supraliminal biases occur as a result of an interaction between high state and trait anxiety levels (MacLeod & Mathews, 1988, MacLeod & Rutherford, 1992; Mathews et al., 1995). However, some other studies within the literature have suggested that cognitive bias might arise solely out of state anxiety alone (e.g.: Wienstein & Nutt, 1995). The role of state and trait variables and potential interactions is debated within the wider literature.

**Summary of cognitive-behavioural treatment on cognitive bias.**

The evidence outlined above suggests that cognitive bias can successfully be reduced following treatment with CBT. These findings also suggest that a reduction in anxious mood and thoughts is accompanied by a reduction in cognitive bias. The results from these studies suggest that cognitive bias may arise as an interaction of high trait anxiety and high state anxiety variables. It is necessary however, to discuss a potential limitation to these studies, since anxious patients are required to complete the same task at baseline and again at follow-up. This type of design introduces the possibility of practice effects as well as the fact that the

patients become more familiar with the testing conditions. It is therefore possible to propose that the differences in state anxiety are due to procedural familiarisation rather than to the CBT itself. Some of these difficulties might be explored further if a GAD waiting list control group were used as well, which were tested on both occasions, since this would highlight the potential effects of practice or task familiarisation.

**Pharmacological treatment of GAD.**

It has been estimated that between 55-95% of patients with an anxiety disorder who are referred to psychology departments for treatment are currently using some kind of anxiolytic medication (Otto, Pollack & Sabatino, 1996), with one of the most popular types being benzodiazepines (Bandelow, Sievert, Rothmeyer, Hajack, & Ruther, 1995). Such medication is thought to reduce the physiological symptoms of anxiety by altering central nervous system functioning, thereby helping to reduce subjective estimates of anxiety. It is proposed that if medication reduces the levels of arousal associated with highly anxious states, then it might also be associated with either a direct or indirect effect upon cognition.

**Benzodiazepine treatment for anxiety.**

Several studies have examined the effects of diazepam on attentional bias for words that are presented supraliminally. Golombok, Mathews, MacLeod & Lader (1990) examined the effect of an acute administration of diazepam 10 mg upon cognitive bias within a group of anxious gynaecological inpatients awaiting an operation. The group consisted of both high and low trait anxious individuals, all

of whom had currently high levels of state anxiety. The results suggest that diazepam did not alleviate anxious mood when compared to placebo, nor did it alter the cognitive bias. In a further examination of the relationship between medication and cognitive bias, Golombok, Stavrou, Bonn, Mogg, Critchlow & Rust (1991) found that a single dose of diazepam 10 mg successfully reduced the level of state anxiety within GAD patients but it did not affect the cognitive bias, as measured by the supraliminal modified Stroop task. The conclusion drawn from this study is that diazepam can be effective in reducing anxious mood state but the distorted cognitive processes associated with GAD patients remain intact. It is interesting to note that this study found that diazepam actually caused an increase in the level of cognitive impairment, since there was 'a very marked overall slowing' (p. 466) in the response speeds given by participants in the colour-naming task. Such slowing might have led to a cofounding effect upon the data gained from this study. However, given that the modified Stroop task contained both neutral and threatening stimuli words, any slowing of response times would have had a relative effect across both variables. Thus, even though overall response times were slower, the relative index of attentional bias was still present.

It is interesting to note that the Golombok et al. (1991) study did not find a difference in cognitive bias once the level of state anxiety had decreased. This finding is discrepant from the finding of Mathews et al. (1995) who report that the reduction in cognitive bias was associated with lower levels of state anxiety following anxiety management training. It is therefore presently unclear why such differences might have emerged, however Golombok et al. (1991) suggest that the effects of the diazepam might not have been sufficiently strong, since the decrease

in state anxiety just reached significance at the 10% level. It therefore remains possible that longer use of benzodiazepines, or a higher dose might be associated with a greater shift in bias.

In order to examine whether prolonged use of benzodiazepines affects cognitive bias, Stewart, Westra, Thompson, & Conrad (2000) examined 50 patients with a DSM-IV diagnosis of either panic disorder, social phobia, GAD or PTSD in order to establish whether the use of the medication altered their selective attention to threat cues. Since benzodiazepines are often taken on a 'when needed' basis (prn) (Otto et al., 1996), Stewart et al. (2000) examined whether differences existed in the selective attention of individuals who regularly took the medication on a prn-basis compared to those individuals who did not, when severity of anxiety symptoms were statistically controlled for. Stewart et al. report that when tested on a supraliminal version of the modified Stroop task, those individuals who regularly took the prn medication demonstrated a significantly greater attentional bias towards threat stimuli, compared to those who did not and this effect was still significant when the severity of baseline anxiety was partialled out. They argue that the use of prn medication might actually serve to heighten the individual's vigilance for threat, since detection of threat, along with a subsequent change in physiological symptoms, would be a cue for taking the medication. This finding suggests that the use of medication in this manner might serve to increase preferential attention towards threat cues and physiological sensations, which might, inadvertently, contribute to greater vigilance. Thus, Stewart et al., suggest that use of prn medication *per se* might inadvertently heightened vigilance for threat, which then leads to the maintenance of anxious mood.

The evidence outlined above suggests that neither acute administrations of diazepam to GAD patients (Golombok et al., 1991) or anxious non-clinical subjects (Golombok et al., 1990) is associated with a reduction in cognitive bias. Neither does there seem to be a reduction in such bias when more prolonged self-administrations of diazepam are used on a prn basis (Stewart et al., 2000). In fact, the latter style of medication use might actually serve to heighten vigilance for threat.

The treatment of anxiety disorders with benzodiazepines is gradually being replaced with the growing use of selective serotonin re-uptake inhibitors (SSRIs). Initially used for treatment of depression, SSRIs have been found to be effective in alleviating anxious mood in a range of anxiety disorders (Baldwin & Birtwistle, 2000, Baldwin 2001).

**Selective Serotonin Re-uptake Inhibitor (SSRI) treatment for anxiety.**

Although the exact neuropathological mechanisms involved in the drug treatment of anxiety disorders remains uncertain, numerous double-blind placebo-controlled trials have shown that SSRIs are efficacious in the short-term and long-term treatment of patients suffering from a range of anxiety disorders (e.g.: den Boer, Westenberg, Kamerbeek, Verhoeven & Kahn, 1987; Baldwin & Rudge, 1995; Hoehn-Saric, McLeod & Hipsley, 1993). Serotonin has been implicated as being an important neurotransmitter within the learning and memory process (McEntee & Crook, 1991) and it is also thought to play a significant part in mediating responses to aversive events (Deakin & Graeff, 1991). Consequently this

has raised the question to whether alterations to the level of serotonin could affect other areas of cognitive processing, particularly that of cognitive bias.

Weinstein & Nutt (1995) examined whether treatment with SSRIs influenced the appraisal of threat. Using a cohort of 15 clinically anxious patients (mainly panic disorder), 15 recovered-panic patients, 15 depressed patients, and 15 matched non-clinical controls, Weinstein & Nutt presented a range of sentences that were related to either physical anxiety, social anxiety or depression. Following the exposure of the sentence, a word probe were presented. The word probe was taken from a selection of anxiety, depression or neutral words, and these probes were presented to the participants in a manner that either semantically matched the preceding sentence, or not. For example, when an anxiety-related sentence was shown, this was followed by a word probe that was either anxiety-related, depression related, or positive. The participants were required to determine whether the probes matched the emotion of the preceding sentence or not. Weinstein & Nutt (1995) report that anxious individuals showed delayed processing of emotional words compared to depressed patients and recovered-panic patients. Following successful treatment with SSRI medication, however, this bias was no longer detectable. Weinstein & Nutt (1995) report that there were no differences in cognitive bias between the recovered-panic group and the control group, which suggests that SSRI treatment is implicated in the removal of cognitive bias. They conclude that cognitive bias in anxiety is due to state anxiety rather than trait anxiety, which suggests that cognitive bias is secondary, rather than primary to anxious mood. Consequently, Weinstein & Nutt (1985) question whether cognitive bias has an aetiological role with anxious mood. However, similar changes in

cognitive bias following treatment have been attributed to an interaction between state and trait anxiety variables, with the latter having been conceptualised as a vulnerability factor for developing anxious mood, when under stressful conditions (MacLeod & Mathews, 1988; MacLeod & Rutherford, 1992; Mathews et al., 1995).

A second study examining whether changes in the level of serotonin alters cognitive bias has been explored by Andrews & Anderson (1998) by looking at whether acute changes in the level of serotonin within anxious and non-anxious individuals led to changes in anxious mood and levels of cognitive bias. Using the serotonin agonist D-fenfluramine (30 mg), the serotonin antagonist methysergide (2 mg) and a placebo, Andrews et al. (1998) examined whether this manipulation improved or worsened anxious mood, as would be expected. Since cognitive bias is thought to be strongly associated with current anxious mood, it was also hypothesised that a subsequent shift in bias might also occur. Using the modified Stroop paradigm (supraliminal version only), Andrews & Anderson (1998) report that neither the serotonin agonist or antagonist influenced anxious mood state in either the anxious nor the non-anxious group. Furthermore, neither substance was found to alter cognitive bias. It therefore appears that acute administrations of either a serotonin agonist or antagonist are not associated with a reduction in anxious mood. Since anxious mood was not alleviated, it perhaps is unsurprising to note that cognitive bias remained consistent. However, the limitations of this study include the fact that Andrews et al used a small sample size of eleven individuals with a range of anxiety disorders. It is therefore possible that the data gained from this small heterogeneous group of patients was not enough to yield significant results, with reference to changes in mood or cognitive bias. Secondly, Andrews &

Anderson (1998) report that they used only the supraliminal version of the modified Stroop, which is influenced by conscious processing. Consequently, it would be interesting to examine whether serotonin agonists/antagonists alter preconscious processing.

**Summary of pharmacological treatment studies on cognitive bias.**

The literature reviewed above presents mixed findings concerning the amelioration of cognitive bias by pharmacological treatment. The results from Golombok et al., (1990); and Golombok et al., (1991) have suggested that an acute administration of diazepam does not alter cognitive bias, although both of these studies used fairly small samples. However, the evidence from Stewart et al. (2000) suggest that diazepam taken on a prn basis might actually increase preferential attention towards threatening stimuli, which suggests that this treatment might further maintain anxious mood. The evidence from Weinstein & Nutt (1995) suggests that SSRIs are efficacious in reducing anxious mood, along with an elimination of cognitive bias. This finding appears to be consistent with the findings of Mathews et al. (1995) following the removal of cognitive bias after CBT, although the exact role of state and trait variables appear to be have been interpreted differently in these reports.

**Conclusions.**

This review has explored the literature on GAD and the extent to which cognitive bias is implicated in the aetiology and maintenance of GAD. The review has focused on four cognitive theories of information processing contained within

the literature, which have sought to elucidate the cognitive mechanisms associated with anxiety. There is now fairly extensive evidence to suggest that anxious individuals are characterised by an interpretive bias and also an attentional bias. The exact role of these biases is hypothesised to differ depending upon the cognitive model employed. For example, Williams et al (1988, 1997) propose that individuals with high levels of trait anxiety demonstrate a vigilance for threat when state anxiety is also high. On the other hand, low trait individuals become avoidant of threat. The cognitive-motivational model of anxiety proposed by Mogg & Bradley (1998) suggests that individuals with high trait anxiety are likely to evaluate stimuli as being threatening in the first instance. As soon as the stimulus has been appraised as threatening, attentional resources are directed towards the source of the threat, which then brings about vigilance. Low trait anxious individuals are characterised by attentional avoidance of minor threat.

Since cognitive models propose that cognitive biases are associated with the aetiology and maintenance of anxious mood, the removal of such biases is considered to be important if the effects of treatment are to be sustained. Evidence from Mogg et al., (1995) suggests that GAD patients no longer showed an attentional bias following treatment with CBT and similar results have been gained from a study of SSRI medication (e.g. Weinstein & Nutt, 1995). However, the literature also contains negative findings, concerning the effects of medication on cognitive bias (e.g. Golombok et al., 1990; 1991; Andrews & Anderson, 1998). Stewart et al. (2000) suggest that the use of prn benzodiazepines might in fact enhance the effects of attentional bias. It would therefore appear that these issues,

such as the relationship between treatment outcome and changes in cognitive bias, need to be investigated further.

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# Cognitive bias in Generalised Anxiety Disorder and its relationship with the effect of Selective Serotonin Re-uptake Inhibitor (SSRI) treatment.

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# Cognitive bias in Generalised Anxiety Disorder and its relationship with the effect of Selective Serotonin Re-uptake Inhibitor treatment.


## Abstract

This study of cognitive biases in Generalised Anxiety Disorder (GAD) contains two parts. Firstly, the longitudinal part examines whether the level of cognitive bias before drug treatment (as measured by the modified Stroop task, two visual probe tasks and the homophone task) can be used as an index to predict which individuals are most likely to respond to such treatment. The results suggest that cognitive bias before treatment does not reliably predict anxious mood following treatment, when the severity of pre-treatment anxious mood is controlled for. Selective Serotonin Re-uptake Inhibitor (SSRI) treatment was found to significantly reduce the level of interpretive bias, although it did not appear to reduce the level of attentional bias. The second part of the study, the cross-sectional part, examines whether there are differences in the level of cognitive bias shown between the GAD group and a matched control group. The results show that the GAD group demonstrated a significantly larger interpretive bias, but there was no evidence of differences in attentional bias.

The discussion considers potential explanations for the findings of this study, and how such findings relate to theoretical understanding and clinical implications.


*Key words*: Interpretive bias; attentional bias; cognition; predictor; SSRI medication; treatment outcome

## Introduction

There is now considerable evidence to suggest that individuals with Generalised Anxiety Disorder (GAD) selectively interpret ambiguous stimuli in a threatening manner (e.g. Eysenck, MacLeod & Mathews, 1987; Mathews, Richards & Eysenck, 1989), and also allocate visual processing resources towards threat stimuli, for stimuli presented within conscious awareness (e.g.: MacLeod, Mathews & Tata, 1986; Mathews & MacLeod, 1985; Mogg, Mathews & Eysenck, 1992; Bradley, Mogg, White, Groom, de Bono, 1999) and outside of conscious awareness (e.g. Mogg, Bradley & Williams, 1993; Bradley, Mogg, Millar, & White, 1995). Taken together, these findings are compatible with the proposal that biases in information processing play an important role in the aetiology and maintenance of anxiety disorders respectively, since anxious individuals are more likely to detect minor potential 'threats' from the environment, which in turn could intensify their anxious mood (Mathews, 1990). Such findings are also consistent with a schema model of anxiety (Beck, Emery & Greenberger, 1986), which suggests that clinically anxious individuals have overactive danger schemata, which cause them to filter information in a negative or threatening manner; such filtering is thought to contribute to further heightening of anxious mood.

Attentional biases for threat have been identified within a range of other anxiety disorders, such as social phobia (Mattia, Heimberg & Hope, 1993), spider phobia (Watts, McKenna, Sharrock & Trezise, 1986), obsessive compulsive disorder (Foa & McNally, 1986) and post-traumatic stress disorder

(PTSD) (Kaspi, McNally, Amir, 1995). Taken together, these findings provide evidence of a cognitive bias across a range of anxiety disorders and such findings are consistent with cognitive formulations of anxiety (e.g.: Beck et al., 1986).

The model outlined by Beck et al. (1986) predicts biases for threat-related information in all aspects of information processing. However, these biases are particularly apparent in attentional and interpretive processes in GAD, but not in explicit memory (e.g. review by Williams, Watts, MacLeod and Mathews, 1988, 1997). These findings led to the development of more recent information processing models of anxiety, such as the Integrative Model proposed by Williams, Watts, MacLeod & Mathews (1988, 1997) and the Cognitive-motivational model proposed by Mogg & Bradley (1998). Williams et al. (1988, 1997) suggest that anxiety-prone individuals become vigilant for threat, when state anxiety is high (a measure of situational anxious mood). Consequently, this model suggests that high trait anxious individuals are at risk of developing pathological levels of anxiety when exposed to stressful situations. On the other hand, individuals with low trait anxiety are thought to become avoidant when faced with threatening stimuli. Williams et al. (1988, 1997) propose that there are particular cognitive mechanisms which determine whether the individual attends to threat, or not. It is suggested that the Affective Decision Mechanism (ADM) is responsible for determining the threat-value of the incoming stimuli at a preconscious stage. If the individual is currently in a highly anxious state, the ADM is more likely to give a high threat value (i.e. high threat 'tag') to new incoming stimuli. Stimuli that are assigned such tags are identified by the Resource Allocation Mechanism (RAM), which controls attention. According to Williams et al. (1988, 1997) high levels of trait anxiety influence the direction of

attention towards threat stimuli. Thus, an overactive RAM causes increased attention to be allocated to threatening information. Williams et al. therefore propose that these attentional mechanisms are faulty within anxious individuals and this causes biases in the direction of attention towards threat stimuli. Williams et al., suggest that high levels of trait anxiety can be conceptualised as a latent vulnerability factor for developing pathological anxiety when state anxiety is also high.

In contrast, Mogg & Bradley (1998) suggest that vulnerability to anxiety arises as a result of dysfunction with the Valence Evaluation System (VES). It is proposed that anxiety-prone individuals have a VES that evaluates relatively harmless stimuli as threatening. This misinterpretation consequently causes the Goal Engagement System (GES) to respond in a manner that is consistent with there being actual threat. As a result, once the stimulus has been evaluated as threatening, the GES interrupts attention and the individual's attentional resources are directed towards the source of the threat. However, low levels of threat are not brought to the individual's attention. According to both Williams et al. (1997) and Mogg and Bradley (1998), cognitive biases operate at a very early stage of processing, so may be found even when the stimuli are presented subliminally to restrict awareness (e.g. with very brief masked presentations).

Since cognitive formulations of anxiety suggest that cognitive biases in selective attentional and interpretive processes are implicated in the development and the maintenance of clinical anxiety, it is of interest to consider briefly the effect of treatment on cognitive biases. Mathews, Mogg, Kentish & Eysenck (1995) examined whether seven sessions of cognitive-behaviour therapy (CBT) was effective in altering attentional bias in a group of GAD patients. They found

that the treatment was associated with a significant reduction of the level of attentional bias shown towards threat-related stimuli shown under supraliminal conditions (i.e. when the stimuli were available to awareness). Mogg, Bradley, Millar & White (1995a) found that, after a course of CBT, GAD patients no longer showed evidence of attentional biases for threat stimuli either under supraliminal, or subliminal conditions. These effects were still evident at two and twenty months following the end of treatment. Similar results indicating the effectiveness of psychological treatment for removing the cognitive bias in other anxiety disorders have been reported in disorders such as social phobia (Mattia, Heimberg & Hope, 1993), spider phobia (Watts, McKenna, Sharrock & Trezise, 1986), and obsessive compulsive disorder (Foa & McNally, 1986). These results combine to suggest that psychological treatment is efficacious in removing cognitive bias.

Several studies within the literature have examined whether medication reduces the level of cognitive bias. In order to assess the relationship between drug treatment and cognitive bias, Golombok, Mathews, MacLeod & Lader (1990) examined whether an acute administration of diazepam 10 mg to a group of anxious gynaecological inpatients awaiting an operation altered cognitive bias for operation-related threat words. The results suggest that diazepam did not alleviate anxious mood when compared to placebo, nor did it alter the cognitive bias. In a further study, Golombok, Stavrou, Bonn, Mogg, Critchlow & Rust (1991) found that a single dose of diazepam 10 mg successfully reduced the level of state anxiety within GAD patients but it did not affect the cognitive bias, as measured by the supraliminal modified Stroop task. The conclusion drawn from this study is that diazepam can be effective in reducing anxious mood state but

the distorted cognitive processes associated with GAD patients remain intact. Moreover, Stewart, Westra, Thompson, & Conrad (2000) report that clinically anxious individuals (with a diagnosis of either panic disorder, social phobia, GAD or PTSD) who used high levels of benzodiazepines on a 'when needed' (prn) basis, demonstrated a higher degree of attentional bias, compared with non-using anxious individuals, even when the severity of the anxiety was controlled. Stewart et al. (2000) suggest that the use of prn medication might serve to enhance the amount of attention that the individual directs towards detecting changes in environmental stimuli or physiological symptoms, as this would be a cue for taking the medication.

Treatment studies have also examined the effect of Selective Serotonin Re-uptake Inhibitors (SSRI) medication on cognitive bias. Andrews & Anderson (1998) examined whether acute changes in the level of serotonin (by using a serotonin agonist and a serotonin antagonist) were accompanied by either a reduction or intensification of anxious mood, and whether this affected attentional bias. Andrews & Anderson (1998) report that neither type of drug altered anxious mood, nor was there a difference in the level of attentional bias. Consequently, they suggest that changes in serotonin levels do not alter the level of attentional bias. A further study of the function of SSRIs on cognitive processing in the literature is reported by Weinstein & Nutt (1995). They report that anxious individuals demonstrated attentional biases for all type of stimuli (physical threat, social threat, or positive), compared to a depressed group, successfully treated anxious group, and a control group. These results suggest that SSRI treatment is associated with the removal of cognitive bias.

In summary, the studies that have examined the effect of treatment on cognitive bias have revealed rather mixed findings. The evidence from Mogg et al. (1995a) and Mathews et al. (1995) suggest that treatment with CBT is associated with a removal of cognitive bias. However, studies that have examined the effects of medication on cognitive bias have been less consistent, since these results have drawn a range of conclusions that have suggested that medication either makes no difference to levels of cognitive bias (e.g. Golombok et al. 1991; Andrews & Anderson, 1998), that the use of prn medication exacerbates levels of bias (Stewart et al., 2000), or that medication removes attentional bias (Weinstein & Nutt, 1995). It therefore appears that further research in this area is needed.

**Longitudinal component of the study.**

Although SSRIs are considered to be effective in treating anxiety disorders (e.g. den Boer, Westenberg, Kamerbeek, Verhoeven & Kahn, 1987; Baldwin, 2001; Baldwin & Rudge, 1995) and are associated with a reduction in cognitive bias (Weinstein & Nutt, 1995), there are a *substantial minority* of individuals who show no response to SSRI treatment and of those who do respond, the magnitude of improvement is often disappointing (Baldwin & Birtwistle, 2000). They also note that it is difficult to predict which anxious individuals will respond well to an SSRI and those who will not. It is therefore of some interest to identify whether there are particular features that distinguish individuals who respond to SSRI medication from those who do not.

As noted earlier, cognitive models of anxiety propose that anxious individuals have faulty information processing mechanisms which leads them to

interpret ambiguous stimuli in a threatening manner, as well as causing them to selectively allocate attention towards threatening stimuli. Moreover, anxious mood is thought to be intensified or prolonged as a result of greater levels of cognitive bias (Mathews, 1990) and it would therefore follow that the individuals who demonstrate the most marked cognitive bias before treatment may be those who experience the greatest levels of anxiety. Furthermore, if the strength of the bias is a primary indicator of intensity of anxious mood, then high levels of bias at the pre-treatment stage may be associated with poor treatment outcome. The question arises, therefore, whether the degree of cognitive bias at assessment is associated with the degree to which the patient responds to the medication.

The main aims of the study are to examine the effect of SSRI medication on anxiety levels and to see whether the severity of the cognitive bias before treatment (Time 1) predicts a higher level of anxiety at the end of treatment (Time 2). Given that treatment outcome is likely to depend to some extent on the severity of the anxiety problems, the study will also examine whether the cognitive bias measures before treatment predict anxiety symptoms at the end of treatment, even when initial severity of anxiety is taken into account. These aims led to the following hypotheses:

Hypothesis 1: SSRI medication will reduce the symptoms of anxiety from Time 1 to Time 2.

Hypothesis 2: There is a positive relationship between the degree of cognitive bias measured at Time 1 and level of anxious mood at Time 2.

Hypothesis 3. The above relationship will be apparent after controlling the level of anxiety before treatment (Time 1).

An additional aim of the study was to examine the effect of SSRI treatment on cognitive bias measures.

Hypothesis 4. Attentional and interpretive bias will significantly reduce from Time 1 to Time 2, following treatment with SSRI medication.

**Cross-sectional component of the study.**

Although the primary interest of the study was to examine the predictive relationship between the cognitive bias measures before treatment and anxiety levels after treatment, supplementary data were also collected from a normal control group. The aim of including this group was to check that the GAD group was showing significant cognitive biases for threat at the pre-treatment stage (i.e. replicating previous findings with the modified Stroop task, visual probe task and homophone task) as this information would be helpful in interpreting the results from the longitudinal part of the study. For example, a failure to find the predicted difference between the GAD and control group on particular cognitive bias measures would cast doubt on the sensitivity of that measure as an index of anxiety vulnerability, and its usefulness as a predictive measure in treatment. If cognitive bias was not found to be predictive of treatment outcome, then the data from the cross-sectional part of the study would be used to compare cognitive bias patterns between the two groups. Thus, the reason for conducting the cross-sectional part of the study after the longitudinal part was to examine whether the

measures of cognitive bias were sensitive in distinguishing levels of bias between the GAD and the matched control group. This cross-sectional part of the study led to the hypothesis that, prior to treatment, the GAD group will show significantly greater cognitive biases in the attentional and interpretive tasks in comparison with non-clinical matched control group.

Hypothesis 5. Individuals with GAD will demonstrate a cognitive bias for threatening stimuli, when compared to a control group.

In order to investigate the hypotheses, the investigation used three tasks that measured the allocation of attention and one measure of interpretive bias. The attentional bias tasks were the modified Stroop task (with both subliminal and supraliminal stimulus presentation conditions), a visual probe task using angry, happy and neutral faces as stimuli, and a visual probe task using physical health-threat and neutral scenes as stimuli. Thus, these tasks examined attentional biases for a wide range of negative stimuli, including social-threat related, physical-threat related words and pictures. Levels of interpretive bias were measured by using the homophone task, which indicated whether the individual interpreted ambiguous stimuli in a neutral or threatening manner. These tasks are described below in more detail.

# Method

## Design and overview

The investigation used two designs, namely a longitudinal design and a cross-sectional. The primary part of the study, the longitudinal component, used

a within-subjects design that examined the effect of treatment on mood and cognitive bias within GAD. The cross-sectional part of the study used a between-subjects design that compared the responses of GAD patients against that of a non-clinical control group. For both parts of the study, both GAD patients and non-clinical controls completed three attentional tasks and one interpretive bias task. The tasks were completed by all participants at the baseline period. The same tasks were then administered four weeks later at the follow-up appointment. The tasks were: the modified Stroop task and associated awareness check, the face pictures visual probe task, the health threat pictures visual probe task and the homophone task. All participants also completed a range of self report measures, relating to anxious and depressed mood.

The longitudinal section of the investigation required the GAD patients to complete the same attentional tasks and the homophone task at Time 1 (pre-treatment) and at Time 2 (post-treatment assessment). There was a period of four weeks between the commencement of SSRI medication and post-treatment assessment, since it is at this stage that SSRIs demonstrate a statistically significant effect in reducing anxious mood, when compared to placebo (Baldwin & Birtwistle, 2000).

**Participants.**

After gaining ethical approval for the study (see Appendix 2) the GAD patients were primarily recruited through a press release via the University's External Relations Department (see Appendix 3). A small number were also recruited from the consultant psychiatrist's mood disorder clinic.

All of the participants underwent a structured diagnostic interview, using The Mini International Neuropsychiatric Interview (MINI; Sheehan, Lecrubier & Sheehan, 1997) with either a consultant psychiatrist, trainee clinical psychologist or a doctoral student. The MINI is a short structured interview that can be used to identify anxiety disorders and depression based upon the DSM-IV criteria. Those participants who met the DSM- IV diagnostic criteria for GAD entered the study and their level of anxiety and depression was assessed using the Hamilton Anxiety Scales for Anxiety and Depression scales (HAM-A; Hamilton, 1959; HAM-D; Hamilton, 1960). This measure was used to evaluate clinical improvement from baseline to outcome.

After an extensive health check, all of the individuals who entered the study were given a prescription of Paroxetine or Citalopram, 20mg. Those individuals who were already taking an anxiolytic and were still experiencing high levels of anxiety (indicating that the treatment was not being effective), were switched onto Paroxetine or Citalopram. A one-week wash-out period was used between the ending of one drug and the beginning of the second. These participants completed the computer and listening tasks at the end of the wash-out period.

A total of 52 people were interviewed, of whom 32 met the DSM-IV criteria for GAD. Those who had co-morbid severe depressive episode, psychosis, current substance abuse, or having previously been treated with CBT were excluded from the study. Of the 32 individuals who were identified as having GAD, 12 individuals declined to take part in the study. Twenty individuals with GAD entered the study and completed the pre-treatment tasks (Time 1), sixteen of these individuals returned for their post-treatment

assessment (Time 2). The participants were aged between 23-63 years of age, of whom 6 were males and 14 were females.

The non-clinical control group were matched as closely as possible for age, gender, and years of education to the GAD patients. The control group comprised of NHS and local authority education employees.

## MATERIALS

### DIAGNOSTIC MATERIALS.

*The Mini International Neuropsychiatric Interview*, Version 5.0, DSM-IV (MINI; Sheehan, Lecrubier & Sheehan, 1997). The MINI is a structured clinical interview that generates DSM-IV diagnoses. It is considered to have excellent psychometric properties and good inter-rater reliability.

*Hamilton Anxiety Scale & Hamilton Depression Scale* (HAM-A, HAM-D; Hamilton, 1959, 1960). The Hamilton scales are a semi-structured interview, mood rating scheme. The HAM-A is a 24 item scale that evaluates 'somatic' and 'psychic' factors of anxiety. The HAM-D is a 17 item scale that evaluates depression and comorbid anxiety symptoms. Both scales use a five point rating system, where 0= absent and 4= severe. The psychometric properties of both scales are well documented (Hamilton, 1960) and both are routinely used by the National Institute of Mental Health in evaluating treatment outcome.

*A Clinical Global Impression* (CGI) is a seven point scale on which the level of improvement is rated. This is scale requires the clinician to rate how much

improvement the patient has shown, and this ranges from 'very much improved' to 'very much worse'.

SELF-REPORT MEASURES.

The following self-report measures were also used:

*Spielberger State-Trait Anxiety Inventory (STAI)* (Spielberger, Gorsuch, Lushene, Vagg & Jacobs, 1983).

The STAI is a 40 item scale that distinguishes between the concepts of anxiety as a transitory emotional state and individual differences in anxiety-proneness as a relatively stable personality trait. The test-retest correlations for the trait anxiety scale are reported to be 0.73 to 0.83. The state anxiety scale coefficients were relatively lower and contain more variance (ranging from 0.16 to 0.62) and this is because a valid measure of state anxiety should reflect the influence of unique situational factors that exist at the time of testing.

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

A 21 item inventory measures specific items addressed in the DSM-IV Depression criteria. Beck et al. (1996) report that the BDI-II has high internal consistency, high content validity, validity in differentiating between depressed and non-depressed subjects, and sensitivity to change.

*Penn State Worry Questionnaire (PSWQ)* (Meyer, Miller, Metzger & Borkovec (1990). The PSWQ focuses on the defining feature of GAD: worrisome thoughts. Since uncontrollable worry in a central feature of GAD, individuals

with this disorder have been found to score even higher than other groups of anxiety disorder patients on the PSWQ (Molina & Borkovec, 1994), and this measure is reported to have good psychometric properties. It is able to successfully discriminate between samples that met all, some, or none of the DSM-III-R criteria for GAD. As a result, it is thought that the PSWQ taps an independent construct within persons who have GAD (Meyer et al., 1990).

*The Marlowe-Crowne Social Desirability Scale (MCS)*; (Crowne & Marlowe, 1960) was included because social desirability may be a confounding variable affecting self-report measures of anxiety (Fox, 1993; Weinberger, Schwartz & Davidson, 1979).

*Mogg, Bradley, Williams & Mathews Worry questionnaire (1993)* This visual analogue scale is used as a measure to rate the degree of worry concerning social, physical and depressed thoughts.

## MATERIALS FOR MEASURING COGNITIVE BIAS.

### Homophone task.

The stimuli were those from the Mathews et al (1989) homophone task. The task contained 14 critical homophones that had either a threatening or neutral interpretation (such as die/dye, weak/week, pain/pane). The task also contained 14 neutral words, which were presented in random order with the homophones. In addition to the 28 words, a further 10 words were presented at the beginning of the tape as practice trials (See Appendix 4). All of the words

were recorded onto an audio cassette to ensure standardised presentation for all participants (see Procedure).

**The Modified Stroop task.**

The task used 100 stimuli words (See Appendix 5) used previously by Bradley et al (1995) which comprised of 20 social threat-related words (e.g.: embarrassed, unpopular) 20 physical threat-related words (e.g.: assaulted, collapse), 20 depression-related words (e.g.: despair, hopeless) and 40 categorised household neutral words (e.g.: carpet, mattress). The words had previously been matched for word length and frequency using Carroll, Davies and Richman's (1971) norms. The task was run using a Compaq laptop computer, which had a 15 inch colour monitor to it. A microphone was attached to the MEL Response box (Micro Experimental Laboratory; Schneider, 1988; Mogg & Bradley, 1995c), which itself was attached to the computer.

**Presence /Absence awareness task.**

The presence/ absence awareness task, taken from Bradley et al. (1995), was used to examine whether any of the participants were able to detect the stimuli shown at the 14 ms SOA duration. The task comprised a total of thirty-two words, with eight words each being drawn from the four word categories described above (social threat, physical threat, depression related, and categorised neutral).

**Emotional faces visual probe task.**

Attentional bias for social threat cues (i.e. angry faces) was measured using the Bradley, Mogg, Falla & Hamilton (1998) emotional faces visual probe task. The stimuli were photographs of facial expressions, which had previously been chosen from 600 photos on the basis of the ratings of four judges on three, six point scales relating to how happy or threatening the faces were (1= not at all to 6= extremely). The mean threat and happy ratings were 4.7 and 1.0 respectively for threat faces, 1.0 and 5.1 for happy faces, and 1.2 and 1.1 for neutral faces (Bradley et al., 1998).

The task contained a total of 128 pairs of pictures, comprised of 32 happy male faces, 32 happy female faces, 32 threat male faces, and 32 threat female faces. Each pair of photos depicted an individual with an emotional expression (happy or threatening) and a matched neutral expression of the same individual. Each photograph was presented in 16-bit monochrome and measured 50mm x 70mm, and was set on a white background, with a border of 7mm and a gap of 70mm between the inner edges of the pictures. A laptop computer, a 15 inch colour monitor and a MEL Response box was used to run this task.

**Health-threat visual probe task.**

This task included 16 threat pictures, each paired with a matched neutral picture. The 16 threat pictures included various health-related scenes, such as a person in a wheelchair, an ambulance, and a collapsed person. The pictures had previously been compiled and rated by a previous researcher (Lees, 2001).

The picture pairs had been matched, as far as possible, for content, complexity and shading. Each photograph was presented in 16-bit monochrome

and measured 74 x 103 mm, and was set on a white background with a border of 9 mm and a gap of 70 mm between the inner edges of each picture. The pictures had previously been rated by Lees (2001) who asked four independent judges to rate on a 7 point scale ( 0= not at all, 6= extremely) how much each picture was related to health /illness. The mean rating for the health threat pictures was 4.78 (sd=1.11) and the mean rating for the neutral pictures was 0.05 (sd=0.39). A paired samples t-test showed that there was a significant difference between the ratings given for the health threat and neutral pictures (p=<0.001).

This task used the same computer equipment as described within the previous tasks.

## PROCEDURE

After reading the Information sheet (see Appendix 6 for GAD patients, Appendix 8 for controls) summarising the study, each participant provided consent to take part in the research (see Appendix 7 for GAD patients, Appendix 8 for controls). Each participant underwent a comprehensive clinical interview and health check and each was free of anxiolytic medication at the time of completing the computer and listening tasks.

Each participant was tested on two separate occasions, four weeks apart. The testing took place within a consulting room within a hospital outpatients department. The room was quiet and free of distractions and the lighting was controlled (but neither very light or dark). Each task was presented in the same order for each participant, with the Homophone task beginning presented first, followed by the modified Stroop task, the awareness check, the faces task and the physical-threat picture task. The homophone task was presented first to prevent

any priming from the other threat-related words or stimuli materials contained within the following tasks.

**Homophone task procedure.**

The stimuli words were recorded onto an audio cassette and this was played to each participant. Each word was presented one at a time, with each followed by an 8 second gap to allow the participant enough time to write the word down. The participants were instructed to write down their answers on a pre-printed response sheet.

**Modified Stroop task procedure.**

The participant sat approximately 60cm in front of the computer screen. The main task contained 100 stimuli words, which were presented once within the suprathreshold condition and once within the subthreshold condition. The trials were presented in a new fully randomised order for each participant. Each trial began with a fixation box (15 mm high x 30 mm wide) for 500 ms in the centre of the screen. This was immediately followed by the stimulus word presented in white, uppercase letters (approx. 4 mm high; average length 18 mm). Each word was accompanied by a flash of colour (either red, blue, green) that was displayed for a single refresh rate on the colour monitor. In order to restrict the participants' visual awareness to the area of the screen that contained the stimulus word, two rectangular pieces of black cardboard (measuring 35 mm x 100 mm) were attached to the monitor. Before the main task began, 16 practice trials were presented in the same manner as described above, although the words were all categorised neutral words (e.g.: bathroom, carpet).

In the supraliminal condition, the stimulus word remained on the screen until the participant made a vocal response. In the subliminal condition each trial began as described before, however a mask was presented 14 ms after the onset of the of the stimulus word (14 ms stimulus onset asynchrony: 14 ms SOA). The mask consisted of a random string of letters (e.g.: YKWPO) The background flash of colour (either red, blue or green) accompanied the stimulus word, as described previously. The mask remained on the screen until the participant made a vocal response.

The computer recorded the latency between the onset of the stimulus word and background colour and the vocal response. Each trial ended with the experimenter manually recording the participants' colour-naming responses on the computer and each trial began with the participant being instructed to 'Press the Start key'.

**Awareness check.**

In each trial of the awareness check, a stimulus word, or a blank space was presented for a period of 14 ms, using similar presentations conditions to those used in the modified Stroop task (i.e. each was presented on a background flash of colour for 14 ms). After 14 ms, a random string of letters replaced either the original stimuli word, or the blank space. The awareness check consisted of thirty trials, of which fifteen trials contained a stimulus word and fifteen trials did not contain a stimulus word. The participants were asked to determine whether or not a word preceded the mask of white random letters. They were asked to respond by either pressing the 'yes' or 'no' button on the MEL response box, depending on whether they had perceived a word or not.

**Emotional faces visual probe task procedure.**

Each participant sat in front of the computer screen as described previously. The task was similar to that used by Bradley, Mogg & Miller (2000).Each trial commenced with a small white fixation cross, measuring 4mm, which appeared in the centre of the screen for 500 ms. This fixation point was then replaced by a pair of photographs for a duration of 500 ms. After the picture pair had been displayed for 500 ms, they were replaced by a probe (arrow) which measured 5mm. This probe appeared in the middle of the space that had previously been occupied by one of the previous pictures. The arrow either pointed up or down, and the participant was requested to press a corresponding button on a response box as quickly as possible. Each participant used their left index finger to respond to an arrow pointing upwards and their right index finger to respond to an arrow pointing downwards.

On half of the trials the emotional face appeared on the left (and the neutral face on the right) and on the other half, the emotional face appeared on the right (and the neutral face on the left) (face location: left/right). On each trial the probe (an arrow) either replaced the critical stimuli face or the neutral face (probe congruous/ incongruous) and on half of these trials the arrow was pointing upwards and on the other half it was pointing downwards (arrow up/ down). Thus, in order to achieve counterbalancing of face location (left/right), probe location (congruous/ incongruous) and probe type (up arrow/ down arrow) a 2x2x2 combination was used for each picture type. Thus all possible combinations were presented with an equal number of trials in each condition.

The order of presentation of the trials was randomised for each participant. Twelve practice trials were presented at the beginning of the task, followed by two buffers trials, and the main 64 main trials. Half the trials showed threat-neutral faces and half showed happy-neutral face pairs.

**Health-threat visual probe task procedure.**

The participant was seated in front of the computer as described before. Each trial commenced with a small white fixation cross, measuring 4mm, which appeared in the centre of the screen for 500 ms. The fixation point disappeared and was followed by a pair of photographs for 500 ms. After the picture pair had been exposed for 500ms it was replaced by an arrow (probe), measuring 5mm which appeared in the middle of the space that had previously been occupied by one of the pictures. The arrow was either pointing up or down and the participant was requested to press the relevant button on a response box as quickly as possible. Each participant used their left index finger to press a button that corresponded to an arrow pointing upwards and their right index finger to respond to an arrow pointing downwards.

The main task consisted of 64 trials of health-threat related pictures and neutral pairs, along with 32 filler trials of neutral-neutral pictures. These trials were presented in a randomised order for each participant. Each task began with 12 practice trials, followed by 2 buffer trials. All of the main task picture pairs were shown four times so that critical picture appeared twice on the left and twice on the right, with the probe appearing twice in place of the critical picture (congruent) and twice in place of the neutral picture (incongruent).

DRUG ADMINISTRATION.

The GAD patients were given a four week prescription of SSRI medication, (Paroxetine or Citalopram 20mg/day) once the diagnostic interview, health check and the baseline cognitive bias tasks had been completed. These two SSRIs were chosen because they are licensed for treatment of GAD and approximately 80% were treated with Paroxetine. Those individuals prescribed Citalopram were those who had been unsuccessfully treated with Paroxetine on a previous occasion. The consultant psychiatrist informed the patient's GP of the assessment and subsequent treatment.

POST-TREATMENT ASSESSMENT.

The longitudinal section of the study involved the GAD patients returning for a post-treatment appointment four weeks after commencement of SSRI medication, where the Hamilton Anxiety and Depression scales were re-administered, along with the complete battery of cognitive bias tasks, and the self-report measures described above.

**Preparation of data from cognitive bias tasks.**

Firstly, all trials with errors were discarded from the analysis. The remaining correct reaction time (RT) data was inspected using box and whisker plots, which gave a visual representation of the RT data for each task. All RTs that were greater than 2000 ms and more than 3 standard deviations above each participants' mean, as well as those RTs that were less than 200 ms, were identified as outliers and were excluded from the analyses (Mogg, Bradley, Williams & Mathews, 1993).

Bias scores for the modified Stroop task were calculated by subtracting the mean colour-naming latency for a neutral word (categorised neutral words) from the mean colour-naming latency for negative words (averaged across social threat, physical threat and depressed words[1]). A positive value indicates that the individual has taken longer to colour-name negative words than neutral words, which would indicate an attentional bias for negative stimuli. Bias scores were calculated for each individual for the subliminal and supraliminal exposure conditions.

For the visual probe tasks (the emotional faces task and the health-threat pictures task) the attentional bias score was calculated from trials that contained a threat and neutral picture. The bias score was obtained by subtracting the time taken to respond to probes that appeared in place of a threatening picture from the time taken to respond to probes that appeared in place of the matched neutral picture. A positive score indicates that the individual was faster to respond to probes that replaced threatening pictures, which would show vigilance for threat. A negative score indicates that the individual responded faster to probes that replaced neutral pictures, which would suggest an avoidance of threat.

With reference to the homophone task, any answers that were not spelt consistently with either of the homophone version were excluded. Of those words that were correctly spelt, the total number of threat-related spellings were recorded, along with the total number of neutral spellings. The interpretive bias was gained by dividing the number of threat-related spellings by the total number

[1] Preliminary analyses using a related T-test indicated that there were no significant differences between each individuals' level of bias shown for physical threat, social threat and depressed words. Therefore an averaged bias score from all three word types was used to reduce the amount of analyses needed.

of correctly spelt homophones and multiplying by 100. This gave a percentage of threat interpretations.

All the bias scores were examined for normal distributions using a one sample Kolomgorov-Smirnov test. The results from this test suggest that all of the scores were normally distributed, when tested at the 2-tailed significance level, for the GAD group and for the control group

## Results

Longitudinal study.

***Question 1. Does SSRI treatment have an effect on mood?***

A Paired T-test compared the scores between Time 1 and Time 2 for each of the following measures: the HAM-A, HAM-D, Spielberger Anxiety Inventory: State subscale (STAIS), Spielberger Anxiety Inventory: Trait subscale (STAIT), the BDI, the PSWQ, and the level of social/physical/depressed worry. (See Table 1 for means).

Insert Table 1 about here.

The results suggest that anxious and depressed mood did change significantly from Time 1 to Time 2, according to the HAM-A, HAM-D, STAIS, PSWQ and the social worries scale. The scores from the BDI indicated a non-significant trend towards improvement. There was no significant difference in the level of STAIT between Time 1 and Time 2.

***Question 2. Does the cognitive bias measured before treatment predict mood after treatment?***

The second set of analyses examined whether cognitive bias at Time 1 predicted anxious mood or depressed mood at Time 2, using Pearson's Correlation Coefficient. The results (contained in Table 2) indicate that, in general, the bias scores from the attentional tasks at Time 1 (i.e. modified Stroop[2] and visual probe tasks) were not significantly correlated with anxious or depressed mood at Time 2, as measured by the HAM-A, HAM-D, STAI, BDI, PSWQ, nor the social/physical/depressed worry measures. There was one exception, which was a significant negative correlation between levels of attentional bias on the Health-threat pictures visual probe task and the self report measure of levels of physical-health worry ($r=-0.61$, $p<0.05$). This suggests that individuals with greater levels of avoidance of health threat pictures before treatment had higher levels of physical-health worries after treatment.

In addition, the Interpretive bias at Time 1, measured by the homophone task, was significantly associated with mood at Time 2. Specifically, there is a significant relationship between the level of interpretive bias at Time 1 and anxious mood at Time 2 (HAM-A, $p<0.05$); and also depressed mood at Time 2 (HAM-D, $p<0.001$; BDI, $p<0.05$). There were no other significant results.

Insert Table 2 about here.

[2] The result from the presence/absence task showed that GAD patients performed no better than chance (50%) when deciding if a stimuli word was present or not, at Time 1 (mean percentage of trials correct = 48.7%, $t(17) = 1.07$, ns) or at Time 2 (mean = 50.7%, $t(14) = 0.29$, ns).

***Question 3. Do pre-treatment cognitive bias measures predict post-treatment mood after controlling symptom severity before treatment?***

In order to further examine the significant relationships identified in response to Question 2 (cited above), some further analyses were conducted to examine whether the relationship between cognitive bias at Time 1 and mood at Time 2 was confounded by the level of anxious or depressed mood at Time 1. First, to check whether mood at Time 1 is significantly associated with mood at Time 2, a series of correlations were used. The results contained in Table 3 suggests that all of the mood measurements at Time 1 are significantly correlated with their respective measurements at Time 2, along with some correlations between the different measures. These results indeed show that, as expected, people with higher anxiety and depression scores before treatment tended to also have higher scores after treatment (See Table 3). This confirms the need to control the effect of pre-treatment mood in further analyses.

Insert Table 3 about here.

A series of partial correlation coefficient tests were used in order to examine whether the relationships between the cognitive bias measures at Time 1 and mood at Time 2 (reported earlier) remained significant after controlling the effect of mood at Time 1. The results from the partial correlation suggest that, when HAM-A at Time 1 is controlled for, the correlation between interpretive bias Time 1 and HAM-A Time 2 does not reach significance ($r=0.28$, ($df=13$) $p=0.31$). When the influence of HAM-D Time 1 is controlled, the interpretive bias at Time 1 still significantly predicts the HAM-D score at Time 2 ($r=0.56$

(df=13) $p<0.05$). When the effect of the BDI at Time 1 is controlled, the relationship between interpretive bias at Time 1 and BDI at Time 2, is no longer significant ($r=0.45$ (df=13) $p=0.09$).

With reference to the relationship between attentional avoidance of health-threat pictures at Time 1 and greater physical-health worries at Time 2, the result of the partial correlation suggests that when the level of physical-health worry Time 1 is controlled, the avoidance of health–threat pictures at Time 1 does significantly predict the level of physical-health worry at Time 2 ($r=-0.63$ (df 13) $p<0.01$).

As can be seen in Table 3, the level of physical-health worry at Time 2 was also significantly predicted by the level of Trait anxiety at Time 1(as measured by the STAIT). Thus, a further partial correlation was conducted to examine whether the relationship between attentional avoidance of health threat-related stimuli at Time 1 and measures of physical-health worry at Time 2 was confounded by STAIT at Time 1. The results suggest that the relationship between attentional avoidance of health-threat pictures at Time 1 and the level of physical-health worry at Time 2 remains significant, after controlling the effect of trait anxiety at Time 1 ($r=-0.61$ (df=13), $p<0.02$)

***Question 4. Does SSRI treatment affect cognitive bias?***

Additional analyses examined whether there was a significant change in cognitive bias in GAD patients from Time 1 to Time 2 (see Table 4 for means). The results from the Paired Samples T- test suggest that there was no significant change in attentional bias between Time 1 and Time 2, as measured by the Supraliminal Stroop, the Subliminal Stroop, Emotional faces task, nor the

Health-threat pictures task (see Table 4). These results suggest that attentional bias does not change from Time 1 to Time 2.

However, the table also shows that the level of interpretive bias, as measured by the Homophone task does significantly change from Time 1 to Time 2. A subsequent Pearson's Correlation suggests that levels of Interpretive bias reduce as Level of Clinical Improvement (as measured by the GCI) increases ($r=-0.63$ $(df=14)$ $p<0.05$). The level of improvement was further indicated by the finding that the reduction in interpretive bias following treatment was comparable to that of the non-anxious control group (the data for the control group is presented in the following section).

Insert Table 4 about here.

**Cross sectional study.**

A control group was matched as closely as possible to the GAD group for gender, years of age and years of education. An Independent t-test confirmed that the two groups were suitably matched for age and education (see Table 5).

Insert Table 5 about here.

Next, the GAD group and control group were compared on their level of anxious and depressed mood. Using an Independent T-test, the results suggest that the two groups differed significantly in levels of anxious and depressed mood, as expected. The two groups did not differ significantly on measures of

social desirability, as measured by the Marlow-Crowne Social Desirability Scale (see Table 6).

Insert Table 6 about here.

An Independent samples T-test was also used to examine whether there was a significant difference in cognitive bias between GAD patients and the control group. and the results suggest that there was no significant difference in attentional biases for threat stimuli, as measured by the supraliminal modified Stroop, the subliminal modified Stroop, the emotional faces visual probe task, nor the health-related threat visual probe task (See Table 7). There was a significant difference, however, in the level of interpretive bias, as measured by the Homophone task, which suggests that GAD patients gave significantly more threatening interpretations of the homophones than the control group.

Insert Table 7 & Figure 1 about here.

## Discussion

*Longitudinal study results.*

The results from the longitudinal study suggest that, firstly, SSRI medication significantly reduces anxious and depressed mood, from Time 1 to Time 2, as measured by the HAM-A, HAM-D, STAIS, PSWQ, and the anxious thoughts ratings. The results from the BDI indicate that there was a non-significant reduction in the level of self-reported depressed mood across this time. The results also suggest that levels of trait anxiety, as measured by the

STAIT remained relatively unchanged from Time 1 to Time 2, and this is consistent with the notion of trait anxiety being a relatively enduring individual characteristic in vulnerability towards anxious mood (Spielberger et al., 1983). These results support the first hypothesis, which predicted that SSRI medication would reduce the symptoms of anxiety. The finding that the medication reduced levels of anxiety from Time 1 to Time 2 is consistent with previous findings (e.g. Baldwin & Birtwistle, 2000, Hoehn-Saric, MacLeod & Hipsley, 1993).

In response to the second question addressed by this study, i.e., whether pre-treatment cognitive bias (at Time 1) predicts anxious mood after treatment (at Time 2), the results suggest that patients with greater levels of interpretive bias at Time 1 had higher levels of anxious mood at Time 2, as measured by the HAM-A. The interpretive bias at Time 1 also predicted more depressed mood (HAM-D and BDI) at Time 2. There was also evidence that patients with the greater avoidance of health-threat related pictures at Time 1 reported more health-related worry at Time 2. However, it is important to remember that the small sample size used in this study may have affected the level of variance and subsequently, the number of significant correlations. Thus, the small sample size might not have been large enough to detect the effect size.

The partial correlations showed that, when the effect of anxious mood at Time 1 is controlled for, interpretive bias does not predict anxious mood (as measured by HAM-A) at Time 2. When the effect of depressed mood at Time 1 was controlled for, the level of interpretive bias significantly predicted depressed mood at Time 2, when measured by the HAM-D, but not when measured by the BDI. Therefore, the findings for depressed mood are equivocal. Thus, the main conclusion drawn from this study is that cognitive bias at Time 1 does not predict

anxious mood at Time 2 independently of the effect of pre-treatment anxiety levels. These findings suggest that the severity of cognitive bias at the pre-treatment stage is not associated with response to treatment, nor does it seem to play a useful role in predicting outcome for anxious mood.

It was also examined whether SSRI medication significantly altered the level of cognitive bias. The results suggest that SSRI treatment reduced the level of interpretive bias from Time 1 to Time 2. This finding suggests that the medication might have a role in altering how the individual evaluates potentially threatening stimuli. One possible explanation of the findings is that the interpretative bias is an interactive function of both state and trait variables. Thus, when levels of state anxiety reduce in anxiety-prone individuals, then the level of interpretative bias also decreases. This perspective would also seem consistent with the findings reported by MacLeod (1990) who found that a group of non-clinical high trait anxious individuals were more vulnerable to developing an interpretive bias when levels of state anxiety were high, in comparison with a low trait anxiety group who showed no such trend. It therefore appears that the reduction in the level of interpretive bias gained in the present study, following treatment with SSRI medication, may be due to a reduction in levels of state anxiety. However, the present results are also consistent with the suggestion that the interpretive bias is solely determined by current anxiety levels (rather than an interaction effect of state and trait anxiety). Indeed, this was proposed by Weinstein and Nutt (1995), whose results also suggested that a cognitive bias for threat information was reduced by SSRI treatment in anxious patients.

Although the level of interpretive bias changed from Time 1 to Time 2, the results from the attentional bias tasks suggest that there was no change from Time 1 to Time 2, as measured by the subliminal Stroop, the supraliminal Stroop, the emotional faces visual probe task and the health-threat pictures visual probe task. These findings are inconsistent with previous treatment studies that have suggested that attentional bias (for example, measured by the modified Stroop) is present before treatment, but is no longer detectable following treatment with CBT (Mathews et al., 1995) or SSRI medication (Weinstein & Nutt, 1995). However, the results regarding treatment effects on cognitive bias are inconsistent, since other studies have reported that medication does not remove the attentional bias (Golombok et al., 1990; Golombok et al., 1991; Andrews & Anderson, 1998), or that medication is associated with an increase in the level of attentional bias (Stewart et al., 2000). These discrepant findings make it difficult to draw any firm conclusions concerning the effectiveness of medication in altering cognitive bias. A noteworthy point is that a large proportion of the studies reported above suggest that anxious mood did *not* decrease following treatment. Thus, if the cognitive bias is largely influenced by state anxiety, it becomes improbable that cognitive bias will decrease if anxious mood is still high. As a result, these studies do not help to clarify the potential relationship between state anxiety and cognitive biases. Subsequently, it would appear that this area would benefit from further research. A further reason why this study failed to find a difference in attentional bias between Time 1 and Time 2 might be due to the small sample size (n=16). The study originally aimed to recruit n=30 GAD patients, which, based on comparable studies from Mathews et al., (1995); Mogg et al., (1995a), would have been sufficient to detect a change in

cognitive bias following treatment. The reason that this study did not contain the desired amount of GAD patients was due to recruitment and retention difficulties.

*Cross sectional study results.*

The results revealed that the GAD group were significantly more anxious and depressed than the control group, as expected. It was also found that the GAD group had significantly higher levels of interpretive bias, and this is consistent with previous studies of clinically anxious individuals (Mathews, et al., 1989) and also with non-clinical high trait anxious individuals (Mogg, Bradley, Miller, Pott, Glenwright & Kentish, 1994, study 2).

However, there were no significant differences in attentional bias between the GAD group and the control group. These results are somewhat surprising, given the previous evidence that suggests that individuals with GAD preferentially allocate attentional resources towards the processing of threat stimuli, when compared to non-anxious individuals. For example, research suggests that individuals with GAD show attentional bias favouring negative information when tested on the modified supra- and sub-liminal Stroop task (Mathews & MacLeod, 1985; Mogg, Mathews & Weinman, 1989; Mogg, et al., 1993; Mathews & Klug, 1993, Bradley et al., 1995); visual probe tasks using threat-related words (MacLeod et al., 1986; Mogg, Mathews & Eysenck, 1992, Mogg et al., 1995b) and the visual probe task using emotional faces (Bradley, et al., 1999; Mogg, Millar & Bradley, 2000). The findings from the present study are therefore clearly inconsistent with such previous findings.

Since the results from the present study found no significant differences in attentional bias between the GAD group and control group, this questions whether the tasks used were sensitive measures of attentional bias in the first instance. If not, they are unlikely to have any value in predicting levels of anxious mood at Time 2. Thus, the null findings from the attentional tasks within the longitudinal component of the study, which suggest that the attentional bias does not predict subsequent anxious mood, might be explained by a lack of task sensitivity.

One possible reason for this study finding null results on the attentional tasks is that the testing conditions were slightly different to those normally achieved within a laboratory. Principally, it appears that the lighting conditions were different to those used in many of the previous studies, since many of the laboratory based studies were completed in semi-dark conditions. On the other hand, the present study was conducted in ambient lighting conditions which were subdued but not dark (i.e. the curtains were drawn within a consulting room and the background lighting was sufficient to allow reading a book). A recent study (the results of which only became available after data collection on the present study) suggests that the lighting conditions are important in determining the sensitivity of the attentional tasks (Mogg & Bradley, 2002). They report that high trait anxious individuals demonstrated an attentional bias for angry faces on the visual probe task (similar to the one used here) when tested in the dark condition, but that no such bias was identified when bright ambient light conditions were used. It is possible that darker conditions not only help focus the person's attention on the task, but also enhance the subjective emotional impact of the stimuli (similar to how horror films are often experienced as more anxiety-

provoking if watched in the dark). These findings give a potential reason why the present study did not find the predicted pattern of results from the attentional tasks.

With regard to the attentional bias for physical-health threat scenes, the lack of difference between the GAD and control group on this measure makes it difficult to interpret the correlation which suggests that avoidance of physical threat pictures at pre-treatment predicted greater physical worries after treatment, even after controlling initial worry or trait anxiety levels. On one hand, this correlation seems compatible with the suggestion that high levels of cognitive avoidance strategies may interfere with normal 'emotional processing' and may thereby maintain anxiety problems (e.g. Rachman, 1980), which would result in poor treatment outcome. On the other hand, the correlational results need to be viewed with caution because there was no evidence from the present study that GAD is associated with a higher level of cognitive avoidance strategies. Moreover, since this correlational result was not specifically predicted, it is possible that it reflects a Type 1 error, arising from the large number of analyses carried out in the study. This potential relationship would therefore benefit from further investigation within further studies.

The principal question posed by this study has investigated whether cognitive bias can be used as an index to predict which individuals are most likely to respond to SSRI medication. This question has not previously been investigated within the literature and this study has provided a first step in answering the question. However, it is important to note that this study does have some limitations, since it used a small sample size and it was conducted in non-

laboratory conditions. The small sample size means that the analyses were limited to correlational tests which do not identify causal relationships, unlike regressional analyses. Thus, if this study were to be replicated, a larger sample size would have to be used, which would allow for regressional analyses to test for predictive relationships. If, in subsequent studies, a predictive relationship between attentional bias and treatment outcome were to be revealed then this means that individual patient profiles could be used to identify those most likely to respond to SSRI medication. Thus, ultimately, the merits of using measures of attentional bias could mean that individuals could be accurately matched to the type of treatment that is most likely to work for them. It is clear, however, that further research is needed in this area before any conclusions can be drawn.

In conclusion, anxious patients who have a stronger bias to interpret ambiguous information in a threatening manner before treatment are more likely to have higher anxiety levels at the end of drug treatment. The results suggest that this relationship may be mediated by current anxious mood, as it was no longer significant when the effect of pre-treatment anxiety was controlled. SSRI treatment significantly reduced both anxious mood and the interpretive bias. The results seem consistent with the view that the interpretative bias is a function of current anxiety levels and so may not be an enduring vulnerability factor for clinical anxiety. The attentional tasks provided very few significant results, which may be partly due to testing under non-laboratory conditions.

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Table 1. Paired t-test comparing mood measures from Time 1 (pre-treatment) to Time 2 (post-treatment) in patients with GAD.

| | Time 1 | | Time 2 | | |
|---|---|---|---|---|---|
| | Mean | sd | Mean | sd | t |
| Hamilton Anxiety Scale | 19.95 | 6.30 | 14.87 | 7.32 | 3.84** |
| Hamilton Depression Scale | 16.16 | 5.60 | 12.75 | 7.25 | 3.66** |
| Speilberger State anxiety scale | 53.68 | 11.06 | 46.75 | 13.25 | 4.43** |
| Speilberger Trait anxiety scale | 59.32 | 8.78 | 58.06 | 10.38 | 0.86 |
| Beck Depression Inventory | 20.26 | 8.82 | 17.81 | 9.26 | 1.82 |
| PSWQ | 67.47 | 9.26 | 63.43 | 12.28 | 2.10* |
| Social worry scale | 5.68 | 2.16 | 4.81 | 2.34 | 2.09* |
| Physical worry scale | 6.21 | 1.87 | 5.56 | 2.16 | 2.71* |
| Depressed worry scale | 5.53 | 2.14 | 4.81 | 2.07 | 1.59 |

**= $p<0.01$, Two Tailed significance

*=p<0.05, Two Tailed significance

Table 2. Pearson Coefficient Correlation r-values showing relationship between Cognitive bias at Time 1 and Mood at Time 2.

| | HAM-A T2 | HAM-D T2 | STAIS T2 | STAIT T2 | BDI T2 | PSWQ T2 | Social worry T2 | Physical worry T2 | Depressed worry T2 |
|---|---|---|---|---|---|---|---|---|---|
| Sup Stroop T1 | -0.21 | -.021 | -0.29 | -0.12 | -0.15 | 0.18 | 0.12 | 0.14 | -0.32 |
| Sub Stroop T1 | -0.21 | 0.01 | -0.36 | -0.20 | -0.22 | 0.06 | 0.01 | 0.06 | -0.43 |
| Faces T1 | 0.14 | 0.29 | 0.29 | 0.11 | 0.23 | -0.40 | -0.22 | -0.02 | 0.29 |
| Health T1 | -0.42 | -0.31 | -0.14 | -0.28 | -0.21 | -0.23 | -0.18 | -0.61* | -0.20 |
| Homophone T1 | 0.52* | 0.64** | 0.37 | 0.33 | 0.57* | 0.12 | 0.12 | 0.31 | 0.18 |

** Correlation significant at p<0.01 (2 Tailed).
* Correlation is significant at p<0.05 (2 Tailed).
n=16, except for Faces task, where n=15.
Note:
Sup Stroop T1= Supraliminal Modified Stroop, Time 1; Sub Stroop, T1= Subliminal modified Stroop, Time 1; Faces T1= Emotional Faces visual probe task, Time 1; Health T1= Health-related threat visual probe task, Time 1; Homophone T1= Homophone task, Time 1.
HAM-A, T2= Hamilton Anxiety Scale, HAM-D, T2= Hamilton Depression Scale, Time 2; STAIS T2= Spielberger State scale, Time 2; STAIT T2 =Spielberger Trait scale, Time 2; BDI, T2= Beck Depression Inventory, Time 2; PSWQ T2= Penn State Worry Questionnaire, Time 2; Social Worry, T2= Social worry scale, time 2; Physical worry T2= Physical worry scale, Time 2; Depressed worry, T2= Worry about depressed thoughts, Time 2.

Table 3. Does mood at Time 1 (T1) predict mood at Time 2 (T2) in patients with GAD?

| | HAM-A T2 | HAM-D T2 | STAIS T2 | STAIT T2 | PSWQ T2 | BDI T2 | Social Worry T2 | Physical Worry T2 | Depressed Worry T2 |
|---|---|---|---|---|---|---|---|---|---|
| HAM-A T1 | 0.70* | 0.80** | 0.38 | 0.37 | 0.22 | 0.60* | 0.09 | 0.26 | 0.44 |
| HAM-D T1 | 0.60 ** | 0.80** | 0.22 | 0.29 | 0.12 | 0.69** | 0.10 | 0.01 | 0.31 |
| STAIS T1 | 0.51* | 0.32 | 0.86** | 0.71** | 0.28 | 0.62* | 0.35 | 0.29 | 0.65** |
| STAIT T1 | 0.49 | 0.50* | 0.45 | 0.83** | 0.66** | 0.70** | 0.69 ** | 0.58** | 0.72** |
| PSWQ T1 | 0..40 | 0.47 | 0.19 | 0.72** | 0.74** | 0.64** | 0.65 ** | 0.35 | 0.41 |
| BDI T1 | 0.43 | 0.52* | 0.35 | 0.64** | 0.36 | 0.75** | 0.46 | 0.09 | 0.53* |
| Social Worry T1 | 0.41 | 0.23 | 0.38 | 0.66** | 0.46 | 0.60* | 0.91** | 0.49 | 0.34 |
| Physical Worry T1 | 0.23 | 0.10 | 0.37 | 0.42 | 0.43 | 0.30 | 0.40 | 0.77** | 0.59* |
| Depressed Worry T1 | 0.42 | 0.39 | 0.51* | 0.73** | 0.61* | 0.38 | 0.41 | 0.33 | 0.69** |

**=p<0.01 level (2-Tailed)
*= p<0.05 (2-Tailed)
n=16
Note: HAM-A, = Hamilton Anxiety Scale, HAM-D, = Hamilton Depression Scale,; STAIS = Spielberger State scale, STAIT =Spielberger Trait scale; BDI = Beck Depression Inventory; PSWQ = Penn State Worry Questionnaire; Social Worry= Social worry scale; Physical worry = Physical worry scale; Depressed worry = Worry about depressed thoughts.

Table 4. Mean bias scores from Time 1 and Time 2 in patients with GAD.

| | Time 1 | | Time 2 | | |
|---|---|---|---|---|---|
| | Mean | sd | Mean | sd | t |
| Supraliminal Stroop | 3.5 | (18.83) | -1.47 | (22.80) | 0.41 |
| Subliminal Stroop | 0.46 | (16.92) | -1.60 | (19.40) | 0.26 |
| Emotional faces task | 7.68 | (30.04) | -3.29 | (24.68) | 0.95 |
| Health-threat pictures task | -1.33 | (28.73) | -6.00 | (24.90) | 0.42 |
| Homophone task | 82.20 | (8.93) | 73.90 | (13.61) | 2.81** |

Note: **=p<0.01 (Two Tailed).
Df = 15, except for the emotional faces task where df =14.

Table 5. Demographic information for GAD and control group.

| | GAD | | Control | | | |
|---|---|---|---|---|---|---|
| | Mean | (sd) | Mean | (sd) | t(35) | p |
| Age | 43.70 | (11.12) | 39.76 | (8.61) | 1.19 | 0.24 |
| Years spent in education | 13.10 | (2.38) | 12.76 | (1.95) | 0.46 | 0.65 |
| Gender (m:f) | 6:14 | | 6:14 | | | |

df=35
Significance set at Two Tailed.

Table 6.

Characteristics of GAD patients and non-anxious controls

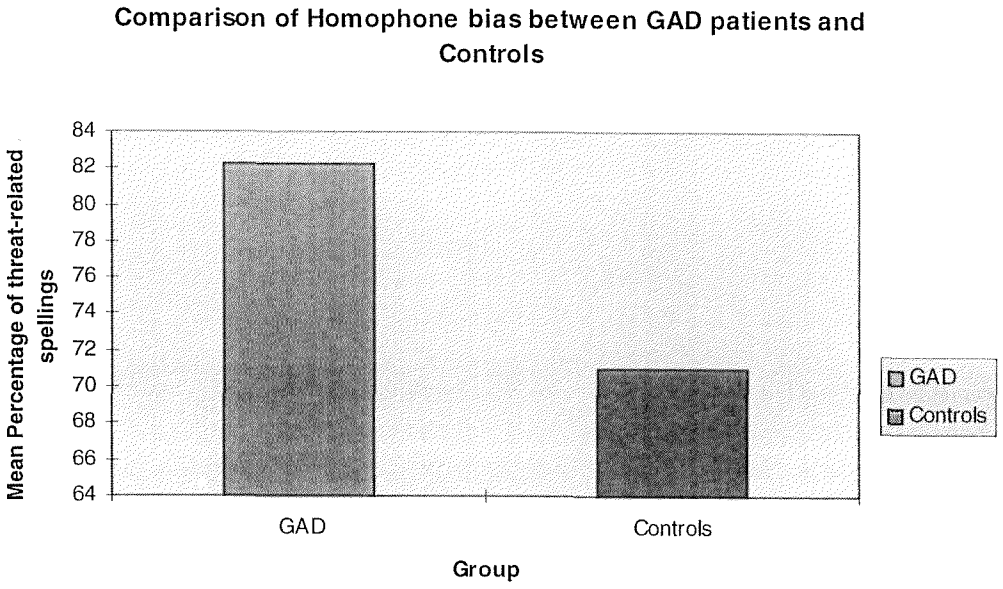
| | GAD | Controls | t (36) |
|---|---|---|---|
| | Mean (sd) | Mean (sd) | |
| BDI | 20.26 (8.82) | 2.74 (0.90) | 8.12** |
| STAIS | 53.68 (11.06) | 24.59 (3.00) | 10.48** |
| STAIT | 59.31 (8.78) | 32.82 (4.51) | 11.17** |
| PSWQ. | 67.47 (9.26) | 33.29 (3.91) | 14.11** |
| Marlowe-Crowne scale | 5.42 (1.74) | 5.82 (1.70) | 0.70 |
| Social worry | 5.68 (2.16) | 1.41 (0.79) | 7.69** |
| Physical worry | 6.21 (1.87) | 1.11 (0.93) | 10.14 ** |
| Depressed-related worry | 5.52 (2.14) | 1.29 (1.31) | 7.04** |

Table 7.

Characteristics of GAD patients and Controls for cognitive bias measures.

| | GAD Mean (sd) | Controls Mean (sd) | t (36) | p |
|---|---|---|---|---|
| Supraliminal Stroop | 3.51 (18.83) | 5.16 (19.67) | 0.26 | 0.80 |
| Subliminal Stroop | 0.46 (16.92) | 0.76 (15.82) | 0.05 | 0.96 |
| Emotional faces task (threat) | 7.68 (30.04) | 14.12 (30.08) | 0.63 | 0.53 |
| Health-threat pictures | -1.33 (28.73) | 2.86 (22.60) | 0.49 | 0.63 |
| Homophone bias | 82.20 (8.93) | 70.94 (5.74) | 4.46 | <0.00 |

Figure 1.


Comparison of Homophone bias between GAD patients and Controls
Mean Percentage of threat-related spellings
84
82
80
78
76
74
72
70
68
66
64
GAD
Controls
Group
GAD
Controls

# APPENDICES

# Appendix 1. Behaviour Research & Therapy: Notes to contributors.

BEHAVIOUR RESEARCH AND THERAPY
incorporating BEHAVIORAL ASSESSMENT

# *Information for Contributors*

*Submission of Papers*

Authors are requested to submit their original manuscript and figures with two copies. Manuscripts for the regular section should be sent to Dr S. Rachman, Department of Psychology, University of British Columbia, Vancouver, British Columbia, Canada, V6T 1Z4. Manuscripts for the *Behavioral Assessment* Section should be sent to Dr S. Taylor, Department of Psychiatry, 2255 Wesbrook mall, Vancouver, British Columbia, Canada, V6T 2A1.

Submission of a paper implies that it has not been published previously, that it is not under consideration for publication elsewhere, 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.

*Manuscript Preparation*

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Gray, J.A. (1976). The behavioral inhibition system: a possible substratum for anxiety. In M. P. Feldman & A. Broadhurst. *Theoretical and experimental bases of the behaviour therapies* (pp. 3–41). London: Wiley.

Taber, I.I., McCormick, R.A., Russo, A.M., Adkins, B.J., & Ramirez, L.F. (1987). Follow-up of pathological gamblers after treatment. *American Journal of Psychiatry, 144*, 757–761.

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# Appendix 2: University of Southampton, Psychology sub-committee, Southampton & S.W. Hants, Joint Ethics Committee approval letter for research.

University of Southampton

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28 August 2001

Paul Brodrick
Trainee Clinical Psychologist
University of Southampton
Highfield, Southampton
SO17 1BJ

Dear Paul,

**Re: Submission No. PSY/10/01**

Following the conditional approval and in response to your letter dated 16th August 2001, I am pleased to confirm **full approval** having received the required amendments.

This committee is fully compliant with the International Committee on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials involving the participation of human subjects as they relate to the responsibilities, composition, function, operations and records of an Independent Ethics Committee/Independent Review Board. To this end it undertakes to adhere as far as is consistent with its Constitution, to the relevant clauses of the ICH Harmonised Tripartite Guideline for Good Clinical Practice, adopted by the Commission of the European Union on 17 January 1997.

Yours sincerely,

P G Coleman

Professor Peter Coleman
Chairman
Psychology Sub-Committee, Southampton & S.W. Hants, Joint Ethics Committee

cc Janet Turner

# Appendix 3: Press release.

Ref: 01/150 7 November 2001

# Anxiety sufferers wanted for Southampton study

A research team at the University of Southampton is seeking people with high levels of anxiety or worry for a new study into Generalised Anxiety Disorder (GAD). Around 1 in 20 of us will experience GAD at some point in our lives. People with GAD worry excessively about anything from health to money or relationships, and this has a dramatic effect on their day to day living. The new research aims to give a better understanding of the disorder and of how treatment may reduce anxiety symptoms.

Sufferers may not even know that they have GAD, but people with the disorder are likely to experience uncontrollable worry or anxiety. Symptoms include the following: restlessness or feeling keyed up or on edge; becoming tired easily; difficulty concentrating or mind going blank; irritability; muscle tensions; and sleep disturbance. If you have GAD you have probably experienced three or more of these symptoms daily over a period of six months or more, and may find your life is significantly affected.

If you are aged between 18 and 65 and think you may have GAD, you are invited to take part in the study, which will involve you completing a short listening task and a series of short computer tasks. If you show severe anxiety symptoms you will be offered treatment, after discussions involving your GP. The same tasks would then be done again four weeks after starting treatment.

Dr David Baldwin at the University's School of Medicine is leading the research: GAD sufferers can be severely disabled by their anxiety, but the disorder is treatable. The results from this research may help us predict which patients will

:espond best to different types of treatment. Treatment can then be targeted more
effectively in the future.'

All the volunteers for the study will receive a comprehensive assessment and be able to discuss their concerns with someone who is new to them,' he added, 'and with treatment, the majority of those taking part should see some improvement in their symptoms.'

If you would like to take part in the study, or would like further information, please contact Paul Brodrick on 023 8082 5531 or David Baldwin on 023 8082 5533.

**Notes for editors:**

Volunteers for the study should live in the Southampton area, to allow their participation in tasks for the study on at least two different occasions.

The University of Southampton is a leading UK teaching and research institution with a global reputation for leading-edge research and scholarship. The University has 20,000 students and over 4,500 staff and plays an important role in the City of Southampton. Its annual turnover is in the region of £200 million.

For the latest news from the University visit SotONLINE, the University of Southampton's daily electronic news service: www.today.soton.ac.uk

**For further information:**

Dr David Baldwin, Department of Mental Health, School of Medicine, University of Southampton
(tel: 023 8082 5533)
Paul Brodrick, Department of Mental Health, School of Medicine, University of Southampton
(tel: 023 8082 5531)
Sue Nottingham, Press Officer, University of Southampton (tel: 023 8059 4993)

# Appendix 4: Homophone stimulus words.

**Homophone stimulus words.**

| Practice | Neutral | Homophone |
|---|---|---|
| Pencil | Month | Die/Dye |
| Shoe | Blanket | Slay/Sleigh |
| Telephone | Survey | Foul/Fowl |
| Plant | Deed | Moan/Mown |
| Fabric | Mobile | Groan/Grown |
| Coffee | Signet | Liar/Lyre |
| Salt | Flannel | Bore/Boar |
| Window | Rake | Pain/Pane |
| Bird | Regard | Weak/Week |
| Caravan | Poodle | Skull/Scull |
| | Avenue | Tease/Teas |
| | Playmate | Bury/Berry |
| | Spade | Guilt/Gilt |
| | Clog | Flu/Flew |

# Appendix 5: Modified Stroop stimulus words.

| Social threat. | Physical threat | Depression | Categorised neutral |
|---|---|---|---|
| DESPISED | ASSAULTED | BLAME | BATHROOM |
| DISGRACE | BLEEDING | DESOLATE | CARPET |
| EMBARRASSED | CANCER | DESPAIR | CUTLERY |
| HOTILE | COLLAPSE | DESPERATE | DOMSTIC |
| INSULTED | EMERGENCY | DISCOURAGED | DOORBELL |
| OFFENDED | FATAL | DISMAL | DUSTER |
| RIDICULE | HARMED | DRAINED | GARAGE |
| SHY | INFECTIOUS | DREAD | GROCERIES |
| SILLY | INJURED | GLOOM | INDOOR |
| UNFRIENDLY | KILLER | GRIEF | LINEOLEUM |
| BLUNDER | ACCIDENT | HELPLESS | MATTRESS |
| FOOLISH | COFFIN | HOPELESS | PROPERTIES |
| HATED | CORONARY | MELANCHOLY | RADIATOR |
| IDIOTIC | FUNERAL | MISERY | SCRUBBED |
| INTIMIDATED | HAZRAD | MOURNFUL | SEATED |
| SCORN | INCURABLE | PITIFUL | SHELF |
| SNEER | LETHAL | SADNESS | STAIRCASE |
| TIMID | MUTILATED | SORROW | TAPESTRY |
| UNPOPULAR | UNWELL | TORMENTED | TEACUP |
| USELESS | | WRETCHED | TOASTER |
| | | | BUCKET |
| | | | BUNGALOW |
| | | | CLEANING |
| | | | COOKING |
| | | | CUPBOARD |
| | | | DRAWER |
| | | | FLANNEL |
| | | | FLOORBOARDS |
| | | | HEATER |
| | | | MANTELPIECE |
| | | | MATCHBOX |
| | | | POLISHED |
| | | | RECIPE |
| | | | SPONGE |
| | | | SPOON |
| | | | TABLECLOTH |
| | | | TENANT |
| | | | TOOTHBRUCH |
| | | | TORCH |
| | | | VENTILATED |

# Appendix 6: Participant information sheet for GAD patients.

University of Southampton

Department of Psychology

*Doctoral Programme in Clinical Psychology*

*University of Southampton*
*Highfield*
*Southampton*
*SO17 1BJ*
*United Kingdom*

Telephone 023 8082 5531

November 2001.

Information sheet (for Study No. Psy 10/01).

Dear Participant,

**Study Title: The effects of anxiety-reducing medication on computer and listening tasks.**

You are being invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully and discuss it with your friends, relatives, and your GP if you wish. 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.

This study aims to investigate the effects of anxiety-reducing medication on concentration and thinking which may be measured from responses that people give on a series of short computer and listening tasks. The study will require you to complete several computer exercises, some listening tasks and some questionnaires before you start taking your new medication. These measures will also be repeated in around four weeks time when you come back for a check-up. It is thought that each appointment will take approximately one hour.

You have been asked to take part in this research because you are someone who experiences difficulties with anxiety. Other people who experience difficulties with anxiety have also been invited to take part. The information gained from the study may improve theoretical understanding of the processes involved in people who experience anxiety problems.

It is up to you to decide whether or not to take part. If you decide to take part you will be given this information sheet to keep and be asked to sign a consent form. If you decide to take part you are still free to withdraw at any time without giving a reason. This will not affect the standard of care you receive.

If you consent to take part in the research your medical records may be inspected by the clinicians involved in this study for the purposes of analysing the results. Your name, however, will not be disclosed outside of the hospital.

This research has been approved by the Southampton & South West Local Research Ethics Committee. However, if you feel that you need to contact the committee, you can do so confidentially by contacting: Chair of the Ethics Committee, Department of Psychology, University of Southampton, Highfield, Southampton, SO17 1BJ. Tel. 023 8059 3995.

You may want to keep this letter for any future questions that you may have.

If you have any further questions, please feel free to contact me. My email address is pmb@soton.ac.uk and my telephone number is at the top of the page.

Yours faithfully,

**Paul Brodrick.**
**Trainee Clinical Psychologist.**
**Supervised by Prof. Karin Mogg, Dr. David Baldwin & Prof. Brendan Bradley.**

# Appendix 7: Consent form for GAD patients.

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Telephone 023 8082 5531

Study Number: **Psy 10/01**
Patient Identification Number for this trial:

# CONSENT FORM

**Title of Project:** The role of cognitive bias in predicting response to SSRI medication.

**Name of Researcher:** Paul Brodrick

**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 understand that sections of any of my medical notes may be looked at by responsible individuals from regulatory authorities where it is relevant to my taking part in research. I give permission for these individuals to have access to my records. ☐
4. I agree to take part in the above study. ☐

_____ _____
Name of Patient Date
Signature

_____ _____
Name of Person taking consent Date
Signature
(if different from researcher)

_____ _____
Researcher Date
Signature

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

# Appendix 8: Information sheet and consent form for control participants.

University of Southampton

Department of Psychology

Doctoral Programme in Clinical Psychology

University of Southampton
Highfield
Southampton
SO17 1BJ
United Kingdom

Telephone +44 (0)23 8059 5321
Fax +44 (0)23 8059 2588
Email

Consent Form for Experimental and Interview Based Studies

**Cognitive bias and mood**
**Consent Form for Research Participants**

**Information sheet**

I am Paul Brodrick, a Trainee Clinical Psychologist from the University of Southampton. I am requesting your participation in a study that is examining the relationship between mood, personality, attention and concentration. This will involve completing a series of simple computer tasks and a listening task and the whole session will last for approximately one hour. You will also be asked to complete some short questionnaires relating to your mood. Personal information will not be released to or viewed by anyone other than researchers involved in this project and will be treated in the strictest of confidence. Results of this study will not include your name or any other identifying characteristics.

Your participation is voluntary and you may withdraw your participation at any time without giving a reason. If you have any questions please ask them now, or contact me, Paul Brodrick at 023 8059 5321 or pmb@soton.ac.uk

Signature Date
Name: Paul Brodrick

**Statement of Consent**

I _____ have read the above informed consent form.

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

**(Circle Yes or No)**

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

Signature Date
Name

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

# Appendix 9: Hamilton Anxiety Scale

# STRUCTURED INTERVIEW GUIDE FOR THE HAMILTON DEPRESSION AND ANXIETY SCALES (SIGH-AD)*

PT'S INITIALS: _____ PT'S ID: _____ TIME BEGAN SIGH-AD: _____

INTERVIEWER: _____ DATE: __ __ __ __ __ __

OVERVIEW: I'd like to ask you some questions about the past week. How have you been feeling since last (DAY OF WEEK)? IF OUTPATIENT: Have you been working? IF NOT: Why not?

What's your mood been like this past week (compared to when you feel OK)?

Have you been feeling down or depressed?

Sad? Hopeless? Helpless? Worthless?

In the last week, how often have you felt (OWN EQUIVALENT)? Every day? All day?

Have you been crying at all?

DEPRESSED MOOD (sadness, hopeless, helpless, worthless):

0 - absent
1 - indicated only on questioning
2 - spontaneously reported verbally
3 - communicated non-verbally, i.e., facial expression, posture, voice, tendency to weep
4 - VIRTUALLY ONLY those feeling states reported in spontaneous verbal and non-verbal communication

IF SCORED 1-4 ABOVE, ASK: How long have you been feeling this way?

NOTES:

* Janet B.W. Williams, DSW, Unit 60, 1051 Riverside Drive, New York, NY 10032

How have you been spending your time this past week (when not at work)?

Have you felt interested in doing (THOSE THINGS), or do you feel you have to push yourself to do them?

Have you stopped doing anything you used to do? (What about hobbies?) IF YES: Why?

About how many hours a day do you spend doing things that interest you?

Is there anything you look forward to?

IF WORKING (IN OR OUT OF THE HOME): Have you been able to get as much (work) done as you usually do?

WORK AND ACTIVITIES:

0 - no difficulty
1 - thoughts and feelings of incapacity, fatigue or weakness related to activities, work or hobbies
2 - loss of interest in activity, hobbies or work - by direct report of the patient or indirect in listlessness, indecision and vacillation (feels he has to push self to work or activities)
3 - decrease in actual time spent in activities or decrease in productivity. In hosp., pt. spends less than 3 hrs./day in activities (hospital job or hobbies) exclusive of ward chores
4 - stopped working bec. of present illness. In hospital, no activities except ward chores, or fails to perform ward chores unassisted

Now let's talk about your sleep. What were your usual hours of going to sleep and waking up, before this began?

When have you been falling asleep and waking up over the past week?

Have you had any trouble falling asleep at the beginning of the night? (Right after you go to bed, how long has it been taking you to fall asleep?)

How many nights this week have you had trouble falling asleep?

INSOMNIA EARLY (INITIAL INSOMNIA):

0 - no difficulty falling asleep
1 - complains of occasional difficulty falling asleep - i.e., more than 1/2 hour
2 - complains of nightly difficulty falling asleep

During the past week, have you been waking up in the middle of the night? IF YES: Do you get out of bed? What do you do? (Only go to the bathroom?)

When you get back in bed, are you able to fall right back asleep?

Have you felt your sleeping has been restless or disturbed some nights?

How many nights this week have you had that kind of trouble?

INSOMNIA MIDDLE:

0 - no difficulty
1 - complains of being restless and disturbed during the night
2 - waking during the night - any getting out of bed (except to void)

What time have you been waking up in the morning for the last time, this past week?

IF EARLY: Is that with an alarm clock, or do you just wake up yourself? What time do you usually wake up (that is, when you feel well)?

How many mornings this past week have you awakened early?

INSOMNIA LATE (TERMINAL INSOMNIA):

0 - no difficulty
1 - waking in early hours of morning but goes back to sleep
2 - unable to fall asleep again if gets out of bed

In the last week, have you had broken sleep or unsatisfying sleep and felt tired when you woke up? How about having bad dreams or nightmares? FOR EACH SX: How bad has that been? How often has this happened in the past week?

INSOMNIA (difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

This past week, have you been feeling better or worse at any particular time of day - morning or evening?
IF VARIATION: How much worse do you feel in the (MORNING OR EVENING)? IF UNSURE: A little bit worse or a lot worse?

NOTE: MOST OF THE INFORMATION NEEDED TO RATE THIS ITEM HAS ALREADY BEEN OBTAINED.

DEPRESSED MOOD (loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

In the last week, have you had trouble concentrating, or trouble remembering things? (How much?)

INTELLECTUAL (difficulty in concentrating, poor memory):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

Sometimes, along with depression or anxiety, people might lose interest in sex. This week, how has your interest in sex been? (I'm not asking about actual sexual activity, but about your interest in sex.)

Has there been any change in your interest in sex (from when you were feeling OK)?
IF NOT: Is this unusual for you, compared to when you feel well? (Is it a little less or a lot less?)

GENITAL SYMPTOMS (such as loss of libido, menstrual disturbances):

0 - absent
1 - mild
2 - severe

FOR WOMEN: When some women feel nervous or anxious, they are unable to have an orgasm, although they have had them in the past. Has that happened to you since you started feeling bad? IF YES: How bad has that been? (When did that start?)

Have you had your period in the last month or so? IF NOT: Do you know why not? IF YES: Has it been especially heavy?

In the past week, have you had to urinate frequently? Have you had the urge to?

FOR MEN: Sometimes when men feel nervous or anxious, they find they have trouble with premature ejaculation, or they have trouble keeping an erection. Has that happened to you since you started feeling bad? IF YES: How bad has that been? (When did that start?)

In the past week, have you had to urinate frequently? Have you had the urge to?

GENITOURINARY SYMPTOMS (frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

How has your appetite been this past week? (What about compared to your usual appetite?)

Have you had to force yourself to eat?

Have other people had to urge you to eat? (Have you skipped meals?)

SOMATIC SYMPTOMS GASTROINTESTINAL:

0 - none
1 - loss of appetite but eating without encouragement
2 - difficulty eating without urging

Have you lost any weight since this (DEPRESSION) began? IF YES: Did you lose any weight this last week? (Was it because of feeling depressed or down?) How much did you lose?

IF NOT SURE: Do you think your clothes are any looser on you?

AT FOLLOW-UP: Have you gained any of the weight back?

NOTE: RATE 1 TO 3 ONLY IF PATIENT LOST WEIGHT AND HAS NOT BEGUN TO GAIN IT BACK.

LOSS OF WEIGHT (Rate either A or B):

A. When rating by history:
0 - no weight loss
1 - probable weight loss due to current depression
2 - definite (according to patient) weight loss due to depression
3 - not assessed

B. On weekly ratings by ward staff, when actual weight changes are measured:
0 - less than 1 lb. loss in week
1 - more than 1 lb. loss in week
2 - more than 2 lb. loss in week
3 - not assessed

NOTE: AVOID CODING "3" IF POSSIBLE

How has your energy been this past week?

IF LOW ENERGY: Have you felt tired? (How much of the time? How bad has it been?)

This week, have you had any aches or pains? (What about backaches, headaches, or muscle aches?)

Have you felt any heaviness in your limbs, back, or head?

SOMATIC SYMPTOMS GENERAL:

0 - none
1 - heaviness in limbs, back, or head. Backaches, headaches, muscle aches. Loss of energy and fatiguability.
2 - any clear-cut symptoms

Have you been putting yourself down this past week, feeling you've done things wrong, or let others down?
IF YES: What have your thoughts been?

Why do you think you are feeling bad now?

Have you been feeling guilty about anything that you've done or not done? What about things that happened a long time ago?

Have you thought that you've brought (THIS DEPRESSION) on yourself in some way?

(Have you been hearing voices or seeing visions in the last week? IF YES: Tell me about them.)

FEELINGS OF GUILT:

0 - absent
1 - self-reproach, feels he has let people down
2 - ideas of guilt or rumination over past errors or sinful deeds
3 - present illness is a punishment. Delusions of guilt.
4 - hears accusatory or denunciatory voices and/or experiences threatening visual hallucinations

This past week, have you had thoughts that life is not worth living? IF YES: What about thinking you'd be better off dead? Have you had thoughts of hurting or killing yourself?

IF YES: What have you thought about? Have you actually done anything to hurt yourself?

SUICIDE:

0 - absent
1 - feels life is not worth living
2 - wishes he were dead or any thoughts of possible death to self
3 - suicidal ideas or gesture
4 - attempts at suicide

In the last week, how much have you been worrying?

How much have you been thinking about the worst that can happen, or been afraid of what's going to happen?

Have you been especially irritable this past week?

ANXIOUS MOOD (worries, anticipation of the worst, fearful anticipation, irritability):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

Have you been feeling especially tense this past week? IF YES: Is this more than is normal for you?

Have you been unusually argumentative or impatient?

Have you been worrying a lot about little things, things you don't ordinarily worry about?
IF YES: Like what, for example?

ANXIETY PSYCHIC:

0 - no difficulty
1 - subjective tension and irritability
2 - worrying about minor matters
3 - apprehensive attitude apparent in face or speech
4 - fears expressed without questioning

In the past week, how much have you felt tired?

How much have you been bothered by any of these things: being startled easily, crying easily, trembling, feeling restless, not being able to relax? FOR EACH SX: How bad has that been this past week?

TENSION (feelings of tension, fatiguability, startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

In the last week, have you been bothered by muscle twitching, stiffness, or sudden muscle jerks?

How about grinding your teeth, having an unsteady voice, or your muscles being tight?

IF YES: How bad has that been? (How much has it bothered you?)

SOMATIC (MUSCULAR) (pains and aches, twitching, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

In the past week, have you had ringing in your ears, blurred vision, hot or cold flashes, feelings of weakness, or pricking sensations? IF YES: How bad has that been? (How much has it bothered you?)

SOMATIC (SENSORY) (tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, pricking sensation):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

This past week, have you been afraid of the dark, of strangers, of being left alone, of animals, of traffic, or of crowds? IF YES: How afraid?

Do you have any other special fears?

NOTE: INCLUDE ANY IRRATIONAL ANXIETY ABOUT OBJECTS OR SITUATIONS.

FEARS (of dark, of strangers, of being left alone, of animals, of traffic, of crowds):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

In the last week, have you had trouble swallowing? Have you had stomach pain or fullness, nausea, vomiting, burning or rumbling in your stomach, or constipation?

FOR EACH SX: How bad has that been this past week?

GASTROINTESTINAL SYMPTOMS (difficulty in swallowing, wind, abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

In the past week, have you had any flushing in your face, or have you been pale? Have you felt lightheaded, or had any tension headaches, or felt the hair rise on your arms, the back of your neck, or your head?

FOR EACH SX: How bad has that been this past week?

AUTONOMIC SYMPTOMS (dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

In the past week, has your heart skipped or pounded? Have you had pain in your chest, throbbing blood vessels, or fainting feelings?

FOR EACH SX: How bad has that been this past week?

CARDIOVASCULAR SYMPTOMS (tachycardia, palpitations, pain in chest, throbbing vessels, fainting feelings):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

In the last week, have you had pressure or tightness in your chest, or choking feelings? What about shortness of breath?

FOR EACH SX: How bad has that been this past week?

RESPIRATORY SYMPTOMS (pressure or constriction in chest, choking feelings, sighing, dyspnea):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - very severe

Tell me if you've had any of the following physical symptoms in the past week. (READ LIST)

FOR EACH SX ACKNOWLEDGED AS PRESENT: How much has (THE SX) been bothering you this past week? (How bad has it gotten? How much of the time, or how often, have you had it?)

NOTE: DO NOT RATE SXS THAT ARE CLEARLY RELATED TO A DOCUMENTED PHYSICAL CONDITION.

ANXIETY SOMATIC (physiologic concomitants of anxiety, such as
GI - dry mouth, gas, indigestion, diarrhea, stomach cramps, belching
C-V - heart palpitations, headaches
Resp - hyperventilating, sighing
Urinary frequency
Sweating):

0 - not present
1 - mild
2 - moderate
3 - severe
4 - incapacitating

In the last week, how much have your thoughts been focused on your physical health or how your body is working (compared to your normal thinking)? (Have you worried a lot about being or becoming physically ill? Have you really been preoccupied with this?)

Do you complain much about how you feel physically?

Have you found yourself asking for help with things you could really do yourself?
IF YES: Like what, for example? How often has that happened?

HYPOCHONDRIASIS:

0 - not present
1 - self-absorption (bodily)
2 - preoccupation with health
3 - frequent complaints, requests for help, etc.
4 - hypochondriacal delusions

RATING BASED ON OBSERVATION DURING INTERVIEW

INSIGHT:

0 - acknowledges being depressed and ill OR not currently depressed
1 - acknowledges illness but attributes cause to bad food, overwork, virus, need for rest, etc.
2 - denies being ill at all

RATING BASED ON OBSERVATION DURING INTERVIEW

AGITATION:

0 - none
1 - fidgetiness
2 - playing with hands, hair, etc.
3 - moving about, can't sit still
4 - hand-wringing, nail biting, hair-pulling, biting of lips

RATING BASED ON OBSERVATION DURING INTERVIEW

BEHAVIOR AT INTERVIEW (fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, belching, brisk tendon jerks, dilated pupils, exophthalmos, etc.):

0 - absent
1 - mild
2 - moderate
3 - severe
4 - very severe

RATING BASED ON OBSERVATION DURING INTERVIEW

RETARDATION (slowness of thought and speech; impaired ability to concentrate; decreased motor activity):

0 - normal speech and thought
1 - slight retardation at interview
2 - obvious retardation at interview
3 - interview difficult
4 - complete stupor

# Appendix 10: Hamilton Depression Scale.

See Appendix 9

# Appendix 11: Spielberger State-Trait anxiety inventory (STAI)

MIND GARDEN
Palo Alto, California

# SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-1

Please provide the following information:

Name_____ Date_____ S_____

Age_____ Gender (*Circle*) M F T_____

**DIRECTIONS:**

number of statements which people have used to describe themselves are given below. ead each statement and then circle the appropriate value to the right of the statement to dicate how you feel *right now*, that is, *at this moment*. There are no right or wrong nswers. Do not spend too much time on any one statement but give the answer which eems to describe your present feelings best.

| | NOT AT ALL | SOMEWHAT | MODERATELY SO | VERY MUCH SO |
|---|---|---|---|---|
| 1. I feel calm | 1 | 2 | 3 | 4 |
| 2. I feel secure | 1 | 2 | 3 | 4 |
| 3. I am tense | 1 | 2 | 3 | 4 |
| 4. I feel strained | 1 | 2 | 3 | 4 |
| 5. I feel at ease | 1 | 2 | 3 | 4 |
| 6. I feel upset | 1 | 2 | 3 | 4 |
| 7. I am presently worrying over possible misfortunes | 1 | 2 | 3 | 4 |
| 8. I feel satisfied | 1 | 2 | 3 | 4 |
| 9. I feel frightened | 1 | 2 | 3 | 4 |
| 0. I feel comfortable | 1 | 2 | 3 | 4 |
| 1. I feel self-confident | 1 | 2 | 3 | 4 |
| 2. I feel nervous | 1 | 2 | 3 | 4 |
| 3. I am jittery | 1 | 2 | 3 | 4 |
| 4. I feel indecisive | 1 | 2 | 3 | 4 |
| 5. I am relaxed | 1 | 2 | 3 | 4 |
| 6. I feel content | 1 | 2 | 3 | 4 |
| 7. I am worried | 1 | 2 | 3 | 4 |
| 8. I feel confused | 1 | 2 | 3 | 4 |
| 9. I feel steady | 1 | 2 | 3 | 4 |
| 0. I feel pleasant | 1 | 2 | 3 | 4 |

# SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-2

Name_____ Date_____

## DIRECTIONS

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

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

# Appendix 12: Penn State Worry Questionnaire.

The Penn State Worry Questionnaire.

**Below are a list of statements relating to feelings of anxiety and worry. Please rate how accurately each statement applies to you, by using the following scale:**

| **Not typical of me** | | | | **Very typical of me** |
|---|---|---|---|---|
| **1** | **2** | **3** | **4** | **5** |

If I do not have enough time to do everything, I do not worry about it.

1 2 3 4 5

My worries overwhelm me

1 2 3 4 5

I do not tend to worry about things.

1 2 3 4 5

Many situations make me worry.

1 2 3 4 5

I know that I should not worry about things, but I just cannot help it.

1 2 3 4 5

When I am under pressure I worry a lot.

1 2 3 4 5

I am always worrying about something.

1 2 3 4 5

I find it easy to dismiss worrisome thoughts.

1 2 3 4 5

As soon as I finish one task, I start to worry about something else I have to do.

1 2 3 4 5

I never worry about anything.

1 2 3 4 5

When there is nothing more I can do about a concern, I do not worry about it any more.

1 2 3 4 5

I have been a worrier all my life.

1 2 3 4 5

I notice that I have been worrying about things.

1 2 3 4 5

Once I start worrying, I cannot stop.

1 2 3 4 5

I worry all the time.

1 2 3 4 5

I worry about projects until they are all done.

1 2 3 4 5

# Appendix 13: Beck Depression Inventory

# BDI-II

Date:

ame: _____ Marital Status: _____ Age: _____ Sex: _____

ccupation: _____ Education: _____

**nstructions:** This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and ien pick out the **one statement** in each group that best describes the way you have been feeling during the **past two veeks, including today**. Circle the number beside the statement you have picked. If several statements in the group eem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one tatement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

### 1. Sadness

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

### 2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

### 3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

### 4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

### 5. Guilty Feelings

0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

### 6. Punishment Feelings

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

### 7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

### 8. Self-Criticalness

0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

### 9. Suicidal Thoughts or Wishes

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

### 10. Crying

0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

_____ Subtotal Page 1

Continued on Back

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### 11. Agitation

0 I am no more restless or wound up than usual.

1 I feel more restless or wound up than usual.

2 I am so restless or agitated that it's hard to stay still.

3 I am so restless or agitated that I have to keep moving or doing something.

### 12. Loss of Interest

0 I have not lost interest in other people or activities.

1 I am less interested in other people or things than before.

2 I have lost most of my interest in other people or things.

3 It's hard to get interested in anything.

### 13. Indecisiveness

0 I make decisions about as well as ever.

1 I find it more difficult to make decisions than usual.

2 I have much greater difficulty in making decisions than I used to.

3 I have trouble making any decisions.

### 14. Worthlessness

0 I do not feel I am worthless.

1 I don't consider myself as worthwhile and useful as I used to.

2 I feel more worthless as compared to other people.

3 I feel utterly worthless.

### 5. Loss of Energy

0 I have as much energy as ever.

1 I have less energy than I used to have.

2 I don't have enough energy to do very much.

3 I don't have enough energy to do anything.

### 6. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.

1a I sleep somewhat more than usual.

1b I sleep somewhat less than usual.

2a I sleep a lot more than usual.

2b I sleep a lot less than usual.

3a I sleep most of the day.

3b I wake up 1–2 hours early and can't get back to sleep.

### 17. Irritability

0 I am no more irritable than usual.

1 I am more irritable than usual.

2 I am much more irritable than usual.

3 I am irritable all the time.

### 18. Changes in Appetite

0 I have not experienced any change in my appetite.

1a My appetite is somewhat less than usual.

1b My appetite is somewhat greater than usual.

2a My appetite is much less than before.

2b My appetite is much greater than usual.

3a I have no appetite at all.

3b I crave food all the time.

### 19. Concentration Difficulty

0 I can concentrate as well as ever.

1 I can't concentrate as well as usual.

2 It's hard to keep my mind on anything for very long.

3 I find I can't concentrate on anything.

### 20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.

1 I get more tired or fatigued more easily than usual.

2 I am too tired or fatigued to do a lot of the things I used to do.

3 I am too tired or fatigued to do most of the things I used to do.

### 21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.

1 I am less interested in sex than I used to be.

2 I am much less interested in sex now.

3 I have lost interest in sex completely.

8 9 10 11 12 A B C D E



_____ Subtotal Page 2

_____ Subtotal Page 1

_____ Total Score

# Appendix 14: Mogg & Bradley (1993) Worry scales.

# WORRY QUESTIONNAIRE

This questionnaire is concerned with some very common types of unpleasant thoughts that many people have.

(1.) Please circle one of the numbers on the scale below to indicate how much you have recently been troubled by **anxious thoughts about physical health concerns** e.g. about illness, death or accidents.

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|
| Not at all | | A little | | Moderately | | Very | | Extremely |

(2.) Please circle one of the numbers on the scale below to indicate how much you have recently been troubled by **anxious thoughts about social concerns** e.g. about what other people think of you, embarrassment, getting on with other people.

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|
| Not at all | | A little | | Moderately | | Very | | Extremely |

(3.) Please circle one of the numbers on the scale below to indicate how much you have recently been troubled by **sad or depressing thoughts** e.g. about loss of a loved one, failure at work or home, loss of a job, feeling hopeless about the future.

| 0 | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|
| Not at all | | A little | | Moderately | | Very | | Extremely |

# Appendix 15: Marlowe-Crowne Social Desirability scale.

PERSONAL REACTION INVENTORY

Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is True or False as it pertains to you personally, then circle that answer.

| | | | |
|---|---|---|---|
| (1) | I like to gossip at times. | TRUE | FALSE |
| (2) | There have been occasions when I took advantage of someone. | TRUE | FALSE |
| (3) | I am always willing to admit it when I make a mistake. | TRUE | FALSE |
| (4) | I always try to practice what I preach. | TRUE | FALSE |
| (5) | I sometimes try to get even with people rather than forgive and forget. | TRUE | FALSE |
| (6) | At times, I have really insisted on having things my own way. | TRUE | FALSE |
| (7) | There have been occasions when I felt like smashing things. | TRUE | FALSE |
| (8) | I never resent being asked to return a favour. | TRUE | FALSE |
| (9) | I have never been irritated when people expressed ideas very different from my own. | TRUE | FALSE |
| (10) | I have never deliberately said something that hurt someone's feelings. | TRUE | FALSE |