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This thesis is presented for Degree of

Doctor of Psychology

of

Murdoch University

2017

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Declaration of Independent Work

I declare that this thesis is my own account of my research and contains as its main content work which has not previously been submitted for a degree at any tertiary education institution.

Bruce Campbell

Abstract

The purpose of this research project was to further the understanding of the construct of fear of emotion, its measurement, its relationship to anxiety disorders and depression, and how it changes over treatment and follow-up with CBT alone or combined with medication in a community mental health centre. Anxiety and depressive disorders cause considerable distress and impairment. Understanding the mechanisms that maintain these disorders is important to optimise the effectiveness of available treatments. Fear of emotion is postulated as a transdiagnostic construct that maintains anxiety and depression and is amenable to treatment. Two studies were carried out; in the first, data from 652 adult patients who had completed treatment were evaluated to determine the association between fear of emotion and symptoms of anxiety and depression, the change after CBT in those who were medicated and those who were not, and those who achieved clinically significant change. Fear of emotion was a weak to moderate predictor of depression and anxiety symptoms independent of medication status. Subscale scores explained a greater proportion of variance of symptoms than total scores, and suggested some specificity in differentiating between the symptoms and the fear of specific emotions. Fear of emotion decreased over treatment regardless of whether patients were medicated or not. There was a trend for a greater proportion of unmedicated patients to achieve clinically significant change. In the second study, 41 patients who completed treatment were assessed at six-month follow-up. Self-reported fear of emotion following CBT treatment predicted severity of anxiety and depression symptoms at follow-up, regardless of medication status. Enhancements to current treatments should continue to target fear of emotion.

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Acknowledgements

I would like to record my thanks to the patients and staff at the Centre for Clinical Interventions where the data for this project were sourced. I am grateful to the patients for the time and effort they put into completing the measures in addition to undertaking the hard work of therapy. The staff of the service have been supportive and encouraging. In particular, I would like to acknowledge Paula Nathan, the former director of the service, for her unwavering belief in my ability to complete this project.

Secondly, I wish to thank my supervisor for this project, Professor Peter Drummond, for his expertise and guidance. Peter was remarkably patient with my progress, critical in the most helpful way, supportive of my efforts and extremely proficient in reviewing drafts.

Lastly, and by no means least, I would like to thank Lynne, Isabelle and James who have supported my passion with their seemingly boundless energy, enthusiasm, patience and love.

Chapter 1 Overview

Anxiety and depression are the two most common psychological disorders affecting the Australian population (Australian Bureau of Statistics, 2008). From the 2007 National Survey of Mental Health and Wellbeing, 20.6% of Australians met criteria for an anxiety or affective disorder in the previous 12 months, and 45% met criteria for a mental disorder across their lifetime. Of those who reported a 12-month mental disorder, 14.4% had an anxiety disorder and 6.2% had an affective disorder.

Over the last 30 years, our understanding of the features of these disorders has changed markedly. In DSM-III (American Psychiatric Association, 1980) anxiety and depression were described in broad terms, and these divisions were gradually refined over subsequent revisions of the DSM to provide more specific differences between these disorders.

While our understanding of these disorders has improved, so too have the range of treatments to alleviate the distress and impaired functionality these problems cause. Research into the efficacy and effectiveness of these treatments has been intense in the previous decade resulting in the publication of expert consensus guidelines describing treatments that are considered to be empirically based or evidence supported (e.g. National Institute for Clinical Excellence, 2004).

Despite these increases in our understanding, no treatment has been shown to be completely effective for all people presenting with either an anxiety disorder or unipolar depression. This has led to research attempting to further unravel the mechanisms by which treatments work in order to gain a clearer understanding of how clinicians may better help sufferers improve their symptoms and functionality.

Current interest in psychotherapy research has refocussed on the role of emotion in the psychopathology and treatment of psychological disorders (Mennin & Farach, 2007). Recent theory and research have highlighted the role of altered emotional

reactivity and associated emotion regulation dysfunction in depressive and related psychological disorders (e.g., Barlow, Allen, & Choate, 2004; Gross & Munoz, 1995, Liverant, Brown, Barlow, & Roemer, 2008). One concept that has been proposed is fear of emotion where people are aware of and react to their emotional states in different ways, some more adaptive than others.

This dissertation will seek to define the fear of emotion construct and how to measure it. I will review the extant literature linking fear of emotion to psychological disorders, and present research which aims to further disentangle the mechanisms by which different effective treatments for anxiety and depression, namely CBT and combined CBT and pharmacotherapy, exert their therapeutic effect by investigating the changes in fear of emotion over the course of treatment.

A major feature of the current research is that it was carried out in the context of a community mental health clinic. This goes some way toward addressing criticisms of previous research by using data from a large number of patients, having few exclusion criteria, the presence of high comorbidity, and a broad range of experience among the treating clinicians involved in administering treatments in usual clinical settings.

Chapter 2 Introduction to Study 1

Defining Fear of Emotion

Fear of emotion can be considered as a construct that describes an individual's disposition to notice sensations associated with the experience of emotion and their fearful reactions to these sensations.

This concept is embedded in modern theories of emotion. In a well-articulated theory, Gross and Thomson (2007) describe a process of emotion regulation in response to the experience of emotions. As part of this process, they describe an 'emotional reactivity' which interacts with the experience of emotion, such that an individual needs to have a threshold sensitivity to notice an emotion, and the individual's response to that emotion is likely to have an effect on their further reactivity to that emotion. In an attempt to integrate a number of fields of psychology, Gross and Thomson (2007) proposed that this emotional reactivity can have a basis that is developmental, biological, neuropsychological, and cognitive. They also note that "little is known about how individuals interpret or reconstrue their physiological signs of emotional arousal" (p. 14).

In a clinical context, Barlow, Allen and Choate (2004) proposed that emotional regulation and dysregulation are at the core of emotional disorders. As such, individuals will attempt to modify positive and more typically negative emotions to alleviate this distress. Individuals who are fearful of their emotional responses are more likely to use maladaptive methods of regulating their emotions, leading to a vicious cycle of increased arousal.

The construct. From a cognitive perspective, the origin of this work dates back to the work described by Goldstein and Chambless (1978) investigating the aetiology of agoraphobia. In explaining the differences in panic symptoms experienced in the presence of situational cues and panic which appeared to occur spontaneously, they

described an anxiety about emotions through the construct of "fear of fear". This anxiety was proposed to arise through classical conditioning, where the physical sensations associated with an anxiety response (e.g. breathlessness, heart palpitations) were paired with an aversive event, such as intense anxiety.

This "fear of fear" was also seen to hold in patients with panic disorder (Chambless, Beck, Gracely, & Grisham, 2000) with an individual's catastrophic misinterpretation of physical symptoms being strongly related to the fear of anxiety related sensations. Individuals with panic disorder were seen to be sensitive to different aspects of the physical manifestation of the emotional experience and these symptoms could be further stratified to the location of the sensations such as cardiovascular, neurological, gastro-intestinal, and behavioural control.

This two-part sensitivity to emotion, that is, noticing the physical signs of emotion and then a secondary anxiety response to the signs, was further explored by Chambless and Graceley (1989). In this study, they argued that fear of the physical consequences of fear was a specific feature of anxiety states (agoraphobia and panic disorder in particular), while fear of the social or behavioural consequences of anxiety was a feature of anxious and depressed patients.

In parallel with the work of Chambless and colleagues (Chambless et al., 2000), Reiss and McNally (1985) investigated the psychopathology of panic disorder. They coined the term "anxiety sensitivity" to describe beliefs about anxiety and a tendency to fear symptoms of anxiety as a result of appraising these symptoms as having negative consequences on one's physical, cognitive or social wellbeing. A cognitive element was proposed to explain why some individuals will experience an emotion, while others will experience the same emotion and then go on to develop a fear of that emotion. This element involved the belief about the experience of that emotion being in some way harmful, which further elicited an anxiety response leading to panic (McNally, 1990).

Since then, considerable research has investigated the relationship between anxiety sensitivity and panic (Taylor, 1995).

Further work extended this concept to include an anxious response to the physical sensations associated with anxious arousal in all anxiety disorders, and anxiety about the experience of sadness in depressive disorders (Barlow, 1991; Reiss, 1991; Taylor & Rachman, 1991). Anxiety sensitivity is also described as a heritable characteristic that may predispose a person to later develop an anxiety disorder (Stein & Rapee, 1999).

In 1997, Williams, Chambless, and Ahrens proposed a generalised "fear of emotion", which they defined as apprehension about losing control over one's emotional experience or over one's reactions to those emotions. They expanded the concept of "fear of fear" to include fear of emotional reactions to the experience of depression, anxiety, anger, and positive emotion, based on the premise that those who feared anxiety would likely fear a range of other strong emotions.

This was supported in research settings where higher baseline anxiety about the experience of negative emotion (i.e. fear of emotion) may potentiate emotional responding and may be predictive of differential behavioural responses during laboratory emotion induction tasks (Brown, Smits, Powers, & Telch, 2003).

Increasingly, researchers suggest that anxiety or future-focused apprehension about the experience of emotion may be a shared component across psychological disorders (Barlow, Allen, & Choate, 2004; Mennin, Heimberg, Turk, & Fresco, 2005). Such a 'transdiagnostic' view is examined in the next section.

Fear of emotion can therefore be thought of as a term which indicates heightened awareness and fearful reactions to symptoms and appraisals of emotion. It can be considered to encompass the constructs of fear of fear and anxiety sensitivity. Fear of emotion, or anxiety about the experience of emotion, may be an important

individual difference among people with anxiety and depression that influences emotional reactivity and response to treatment.

Related constructs. The concept that an underlying individual difference in emotionality could be present across psychological disorders is not novel. Several constructs have been articulated and supported by research over the past 40 years. Perhaps the best known of these is negative affectivity proposed by Watson and Clark (1984). In their seminal work, negative affectivity was described as a "dimension of stable and pervasive individual differences in mood and self-concept" (p. 483), which has been seen to be associated with anxiety and depression when present in high levels (Clark & Watson, 1991).

While individuals who are high in fear of emotion are likely to be high in negative affectivity, the construct of fear of emotion differs in that it specifically relates to the response to the emotion, not merely the presence of the negative emotion. Indeed anxiety sensitivity, a construct related to fear of emotion, is differentiated from negative affectivity and trait anxiety in the psychopathology of panic and anxiety symptoms (Gregor & Zvolensky, 2008). While both are risk factors for anxiety and other psychopathology, differences remain such as the ability to predict future anxiety symptoms (Schmidt, Mitchell, & Richey, 2008).

Fear of emotion was also incorporated in Linehan's (1993a) influential work on the development of dialectical behaviour therapy (DBT) for treating borderline personality disorder. Linehan described the pervasive emotional dysregulation that occurs in an interaction between emotional vulnerability and ineffective emotion modulation strategies (1993b). Linehan further described emotional vulnerability as made up of three components: very high sensitivity to emotion stimuli, very intense response to emotional stimuli, and slow return to emotion baseline following arousal. There are few studies investigating this aspect of Linehan's model. The studies reported

used reactions to faces showing various emotional expressions (Wagner & Linehan, 1999); thus, in this line of research, fear of emotion is conceptualised as a reaction to other's emotion, as opposed to the reaction to one's own emotion as defined in this paper.

Measuring fear of emotion. Measurement of fear of emotion is most widely described by the Anxiety Sensitivity Index (ASI: Reiss, Peterson, Gursky, & McNally, 1986). Initially developed by Reiss and colleagues to assess anxiety sensitivity, it consists of a higher order factor representing the anxiety sensitivity construct (Zinbarg, Barlow, & Brown, 1997) and a number of lower order factors. Psychometric research has identified a multidimensional factor structure which shows one, two, three and four factor solutions. There is considerable research suggesting that the taxonomy of anxiety sensitivity is made up of a higher order factor, along with three lower order factors (Zinbarg, Mohlman, & Hong, 1999). A large study reported by Rodriguez et al. (2004) determined that the best explanation was a three-factor model; the lower order factors being Physical Concerns, Mental Incapacitation, and Social Concerns, all loading onto a single higher order factor. This holds in both adult populations and adolescent populations (Dehon, Weems, Stickle, Costa, & Berman, 2005).

Studies also show the ASI sub-scales are suggestive of different responses to fear and anxiety (Taylor & Cox, 1998; Zvolensky & Forsyth, 2002). Zvolensky and Forsyth (2002) found that the anxiety sensitivity dimensions were only weakly associated with body vigilance and emotional avoidance which they suggested may have been due to the sample consisting of adults without existing psychopathology.

There have been criticisms of the ASI (summarised in Blais et al., 2001), which were largely addressed in the Anxiety Sensitivity Index – Revised (ASI-R; Taylor & Cox, 1998). However, despite overcoming some problems, others emerged in the psychometric evaluation of the ASI-R (Deacon, Abramowitz, Woods, & Tolin, 2003).

Indeed, further psychometric research has led to a suggestion that a more reliable and valid 21-item version has a greater clinical utility (Armstrong, Khawaja, & Oei, 2006). Ultimately, Taylor et al. (2007) developed the ASI-3, an 18-item measure showing a consistent factor structure comprising three correlated factors (physical, social and cognitive concerns) loading on to a higher order anxiety sensitivity factor. Subsequent validation studies have proposed a bi-factor structure, as opposed to a hierarchical structure, as more accurate representation of the relationship between the subscales and the total scale (Ebesutani, McLeish, Luberto, Young & Mack, 2014). This bi-factor structure has also been shown in a clinical sample (Rifkin, Beard, Hsu, Garner, & Bjorgvinsson, 2015).

There has been much discussion in the literature about the nature of anxiety sensitivity and its measurement. This debate focuses on whether the construct is considered as taxonomic (i.e. individuals can be classified as having 'normal' or 'pathological' anxiety sensitivity) or dimensional (i.e. individuals have varying degrees of anxiety sensitivity along a continuum). A range of advanced mathematical techniques have been used in an attempt to understand the structure of anxiety sensitivity.

However, disparate findings are still reported with some researchers claiming that anxiety sensitivity has a dimensional latent structure (Asmundson, Weeks, Carleton, Thibodeau, & Fetzner, 2011), while others propose a taxonic structure (where people are considered to fall into either a 'normal' taxon or 'pathological' taxon), with each taxon exhibiting a multidimensional factor structure (Bernstein, Stickle, Zvolensky, Taylor, Abramowitz, & Stewart, 2010).

Results of studies that have employed the ASI implicate fear of emotion in the aetiology and maintenance of a number of psychological disorders (see next section).

Collectively, these results suggest that targeting fear of emotion may be critical for the

prevention and treatment of many psychological disorders (Smits, Berry, Tart, & Powers, 2008).

A similar scale, the Affective Control Scale (ACS), was developed by Williams, Chambless, and Ahrens (1997) specifically to measure the fear of emotion. Their work was an extension on the cognitive model of agoraphobia (Chambless, Caputo, Bright & Gallagher, 1984), and they proposed two measures to assess features of the model. They created the Agoraphobic Cognitions Questionnaire (ACQ) and Body Sensations Questionnaire (BSQ). The ACQ was designed to assess the frequency of thoughts of physical or behavioural consequences of anxiety symptoms whilst the BSQ was designed to assess the fear of anxiety-related sensations. Chambless et al. (1984) proposed that fear of the physical consequences of fear was a specific feature of anxiety states (agoraphobia and panic disorder in particular), while fear of the social or behavioural consequences of anxiety was a feature both of anxious and depressed patients.

Summary. Fear of emotion is a construct describing an individual's heightened awareness of and reactions to symptoms of emotion. It originated in the development of cognitive models of agoraphobia and panic and has been extended to other anxiety and affective disorders. Similar constructs assessing an underlying emotional reactivity have been derived, each with their distinct differences. Two scales have been developed to assess fear of emotion. One, the ASI, originally focused on anxiety, but with broader applicability, and the other, the ACS, was developed specifically for assessing a broader fear of emotion. It is expected that these two measures will be highly correlated as they are based on similar underlying theoretical principles, and the subscales are attempting to assess similar domains.

Relationship of fear of emotion to psychopathology

As stated in the previous section, fear of emotion has been shown to be associated with psychopathology of mood and anxiety disorders. Different approaches have been used to investigate this relationship; on one level suggesting that fear of emotion is related to specific psychopathology, while at another level suggesting it to be a feature common across a range of psychological disorders.

Fear of emotion and anxiety. Fear of emotion was a construct first developed to further understand the aetiology and psychopathology of panic disorder and agoraphobia. As such, the greatest amount of research investigating the fear of emotion and psychological disorders has been reported on subjects experiencing panic disorder. The presence of this factor has been investigated over a broad array of anxiety disorders, with the exception of specific phobia, and has been shown to be a risk factor across anxiety disorders (Zvolensky, Schmidt, Bernstein, & Keough, 2006); a summary of this is presented in the following sections.

Panic and Agoraphobia. Panic disorder is an anxiety disorder which features intense brief periods of anxiety symptoms (panic attacks) occurring on repeated occasions. These attacks are experienced as being unexpected or uncued, and the sufferer has ongoing concerns about experiencing further attacks, or about the consequences of having the attacks. These concerns relate to the anxious symptoms; for example, a person experiencing heart palpitations may believe they are having a heart attack (APA, 2013).

Fear of emotion may be involved in the aetiology and maintenance of panic disorder in three ways. Firstly, Goldstein and Chambless (1978) used the phrase "fear of fear" and proposed that panic disorder arose from a fearful response conditioned to a panic attack. Thus, in this model, fear of emotion results from experiencing the aversive physical sensations of a panic attack. A second model proposed by Clark (1986) sees

the panic response arising from a catastrophic misinterpretation of symptoms of anxiety which, in turn, heightens the fear and subsequent symptoms of anxiety. A third explanation was offered by McNally (1989) who proposed a broader fear of emotion construct, where the individual holds a dispositional tendency to respond fearfully to symptoms of anxiety. In this instance, the anxiety response may arise from any cue perceived as threatening, not just the experience of panic. Each of these models involves a fearful response to an intense emotional experience.

Fear of emotion has been shown to be elevated in panic disorder patients presenting for treatment and in healthy individuals participating in research trials (see Table 1).

Table 1
Summary and Features of Studies Investigating the Association of Fear of Emotion and Panic Disorder (PD)

Study	Population	Controls	Measures	Findings	Comments
Clinical samp	les		Mean (Standard Deviation)		
Brown & Barlow, 1995	63 patients with panic disorder		ASI 33.49 (10.91)	Fear of emotion scores were significantly higher than previously published norms.	Authors reported a follow-up study of patients treated for panic disorder. The score on the fear of emotion measure was reported at various time points, including pretreatment
Brown, Smits, Powers, & Telch, 2003	192 outpatients meeting criteria for panic disorder with agoraphobia undergoing a hyperventilation challenge		ASI 35.09 (12.01)	Fear of emotion scores were significantly higher than previously published norms.	Physical Concerns subscale of ASI predicted scores of subjective units of fear.
Cucchi et al., 2012	139 outpatients diagnosed with PD with Agoraphobia presenting for treatment at a university hospital	157 healthy controls	ASI PD with Agoraphobia 30.22 (10.39) Controls 16.83 (9.39)	Patients diagnosed with PD with Agoraphobia had significantly higher scores than healthy controls.	Twenty five patients had a comorbid psychological disorder
Kampfe et al., 2012	369 patients with PD with agoraphobia		ASI 31.31 (11.57)	ASI scores were similar to those reported in other samples of patients with PD with agoraphobia	Over 90% of the sample had a comorbid disorder

Study	Population	Controls	Measures	Findings	Comments
Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004	206 individuals, a subset of an ongoing longitudinal study, who met diagnostic criteria for an anxiety disorder. Of these, 103 met criteria for PD with Agoraphobia		Mean (Standard Deviation) ASI 44.61 (11.77)	Using regression analyses, scores on ASI were significantly associated with a diagnosis of PD with Agoraphobia.	Individuals included in the study were not assigned a primary diagnosis and all had a co-morbid anxiety disorder and/or MDD. On analysis of the ASI subscales, individuals with PD scored higher on the Physical Concerns subscale.
Sandin, Sanchez- Aribas, Chorot, & Valiente, 2015	168 individuals seeking treatment with a diagnosis of PD with or without agoraphobia at a Spanish health centre		ASI-3 31.71 (15.79)	Fear of emotion predicted PD severity.	
Smits, Powers, Cho, & Telch, 2004	130 people referred to a specialist anxiety disorders treatment centre with a diagnosis of PD with agoraphobia. 90 were allocated to a treatment condition and 40 to a waitlist control condition	Waitlist control	ASI Treatment group 37.32 (11.04) Waitlist control group 33.13 (10.78)	In both groups fear of emotion scores were significantly higher than previously published norms.	
Taylor et al., 1992)	151 patients from a group of 313 presenting for treatment at a university hospital	No control	ASI 36.6 (12.3)	Across a range of anxiety disorders, fear of emotion was seen to be elevated in patients with PD	Scores for patients with PD were significantly greater than those with other anxiety disorders (except PTSD)

Study	Population	Controls	Measures	Findings	Comments
Taylor, Koch, Woody, & McLean, 1996	135 people meeting diagnostic criteria for PD and MDD recruited for the study; 52 PD without MDD, 37 PD with MDD		Mean (Standard Deviation) ASI PD without MDD 31.4 (9.6) PD with MDD 40.3 (11.3)	People with PD, with and without MDD, scored higher on fear of emotion compared with previously published norms.	The study also investigated the subscales of the ASI and showed that Social Concerns and Physical Concerns were more strongly associated with PD than Mental Concerns
Taylor, et al., 2007	390 patients seeking treatment at two anxiety disorder clinics of which 143 were diagnosed with PD	4720 undergraduate students	ASI-3 Physical Concerns 11.3 (6.7) Mental Concerns 9.0 (6.4) Social Concerns 12.3 (5.8)	The patients with PD scored significantly higher on the three subscales compared with controls. Patients with PD scored significantly higher on Physical Concerns subscale than patients with other anxiety disorders.	
Telch, et al., 1993	67 patients meeting criteria for panic disorder. Patients were randomly assigned to a treatment or delayed treatment condition		ASI Treatment 33.74 (11.15) Delayed treatment 34.46 (11.33)	In both groups fear of emotion scores were significantly higher than previously published norms.	

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Zinbarg, Barlow, & Brown 1997	432 patients seeking treatment at an anxiety disorder clinic of which 156 were diagnosed with PD	32 non- patients recruited for the study	ASI Physical Concerns 18.65 (6.94) Mental Concerns 6.66 (4.53) Social Concerns 7.96 (2.79)	The patients with PD scored significantly higher on the three subscales compared with controls. Patients with PD scored significantly higher on Physical Concerns subscale than patients with other anxiety disorders.	Authors reported on a study determining the factor structure of ASI; the findings indicated a higher order factor and three lower order factors – Physical Concerns, Mental Concerns, Social Concerns
Student sampl	les				
Berg, Shapiro, Chambless, & Ahrens, 1998	116 undergraduate psychology students who had no reported history of panic attacks or panic- like symptoms		ACS 2.97 (.90)	Fear of emotion predicted higher scores on anxiety measures after induction of panic-like symptoms	Fear of emotion remained a significant predictor of panic-like symptoms after controlling for state and trait anxiety
Cox, Endler, Norton, & Swinson, 1991	265 college students		ASI 22.48 (10.12)	The students with high levels of fear of emotion reported more panic attacks in the preceding 12 months compared with those reporting low levels of fear of emotion	
Deacon & Valentiner, 2001	1071 undergraduate psychology students		ASI Non-panickers 17.8 9.6) Panickers 24.2 (9.5)	Nonclinical panickers scored significantly higher than did nonpanickers on ASI-total scores, and on two subscales (Physical Concerns, Mental Concerns)	

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Schmidt, Lerew, & Jackson, 1997	1401 young adults commencing military training		ASI 4.0 (2.9)	Fear of emotion predicted spontaneous PAs while controlling for panic history and trait anxiety	Low levels of fear of emotion at start of training – may be due to physically healthy young males enrolling, and that individuals with elevated levels of fear of emotion may choose not to enlist in the military
Schmidt, Lerew, & Jackson, 1999	1296 young adults commencing military training		ASI 17.9 (8.2)	Fear of emotion predicted spontaneous panic attacks while controlling for panic history and trait anxiety.	Fear of emotion at start of training was similar to previous published norms. Analysis using subscales of ASI found Mental Concerns predicted panic attacks
Williams, Chambless, & Ahrens, 1997	105 undergraduate psychology students who had no reported history of panic attacks or panic like symptoms		ACS	Fear of emotion predicted fear of panic-like sensations. Fear of emotion scale was modified to remove fear of anxiety items, and fear of emotion still predicted panic.	Baseline anxiety was not measured.
Zvolensky, Eifert, & Lejuez, 2001	96 university students from a larger screening sample. Participants were selected to be one SD above (n=48) and below (n=48) the mean of the sample		ASI Females 37.7 (2.4) Males 35.8 (3.5)	Individuals with high fear of emotion scores reported panic inducing challenges as more aversive than those with low fear of emotion scores.	The authors also investigated the role of having control over the panic-inducing challenges. Those with elevated fear of emotion scores reported higher anxiety when under the belief that the challenge could not be controlled.

PD = panic disorder; PTSD = posttraumatic stress disorder; MDD = major depressive disorder

In patients presenting for treatment, scores on measures of fear of emotions were shown to be elevated in comparison to previous published norms. In a group of patients with various anxiety disorders, Taylor, Koch and McNally (1992) reported those with panic disorder had a mean ASI score of 36.6 (SD=12.3) compared with normal individuals with a mean score of 17.8 (SD=8.8) (Peterson & Reiss, 1987). Similarly, Cucchi et al. (2012) found patients with panic disorder with agoraphobia reported ASI scores of 30.22 (SD=10.39) compared with scores of 16.83 (SD=9.39) in healthy controls. The scores in patients with panic disorder were higher than those for all other anxiety disorders, except for those with posttraumatic stress disorder. Similar elevations were reported in those with a diagnosis of panic disorder alone (Telch, et al., 1993, Smits, Powers, Cho, & Telch, 2004, Brown & Barlow, 1995), in those with panic disorder with or without comorbid major depressive disorder (Taylor, Koch, Woody, & McLean, 1996), and in those with panic disorder with comorbid major depressive disorder or other anxiety disorders (Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004, Kampfe, et al., 2012). Fear of emotion scores also predicted panic disorder severity (Sandin, Sanchez-Aribas, Chorot, & Valiente, 2015).

Lower order factors of the ASI. Zinbarg, Barlow and Brown (1997) found that patients with panic disorder scored significantly higher on the three subscales of Physical Concerns, Mental Concerns and Social Concerns compared with normal controls. Compared with other anxiety disorders, patients with panic disorder scored significantly higher on the subscale of Physical Concerns, which is to be expected given how the patients' perception of negative physical consequences of anxiety are featured in the models of psychopathology. This pattern of findings was also strongly supported in the study developing and validating the ASI-3 (Taylor, et al., 2007). Scores on the Physical Concerns subscale were also found to predict subjective fear in patients with

panic disorder undergoing an interoceptive challenge (Brown, Smits, Powers, & Telch, 2003).

The association between fear of emotion and symptoms of panic has also been found in non-clinical populations. These studies typically use students who self-report panic attack history and may be subject to various stressors which have been shown to induce panic-like symptoms.

In students reporting panic history retrospectively, those with elevated scores on the fear of emotion measure reported more panic attacks (Cox, Endler, Norton, & Swinson, 1991). Similarly, Deacon and Valentiner (2001) found that students reporting a history of panic attacks scored higher on the fear of emotion measure when compared with students reporting no history of panic. These researchers also analysed the subscales of the ASI and noted that scores on the Physical Concerns and Mental Concerns subscales were also significantly higher in the students reporting panic attacks. Fear of emotion is strongly related to panic-inducing challenges in laboratory-based tests, with those with higher anxiety sensitivity scores reporting high levels of emotional arousal, displeasure and concerns about control in response to those challenges (Zvolensky, Eifert, & Lejuez, 2001).

Fear of emotion is also predictive of panic attacks as shown in two studies by Schmidt, Lerew and Jackson (1997, 1999). Young adults with higher scores on the fear of emotion measure were more likely to have panic attacks during military training than those with lower scores. When examining the subscales of the ASI, Mental Concerns was the strongest predictor of the occurrence of spontaneous panic attacks.

In the few studies reported using the ACS as the measure of fear of emotion, elevated levels of fear of emotion were predictive of panic in subjects with no previous panic history (Williams, Chambless, & Ahrens, 1997; Berg, Shapiro, Chambless, & Ahrens, 1998).

In summary, models of the psychopathology of panic disorder describe aspects of fearful responding to anxiety symptoms. Fear of emotion is elevated in patients with a diagnosis of panic disorder. These patients report the highest scores on measures of fear of emotion compared with those with other anxiety disorders (except those with posttraumatic stress disorder). Investigation of the subscales of the ASI shows that Physical Concerns and Mental Concerns subscale scores are consistently elevated in patients with panic disorder. Fear of emotion is also associated with panic attacks in individuals without a formal diagnosis of panic disorder. This is seen in those individuals reporting a history of panic attacks, and in those who develop panic attacks or panic-like symptoms in induction challenges.

Social Anxiety Disorder. Social anxiety disorder is a psychological disorder characterised by excessive fear in perceived or actual social situations. The elevated levels of anxiety are associated with fears of being negatively evaluated or judged by others in social or performance situations, often with an individual's belief that their symptoms of anxiety are obvious to others resulting in further negative appraisal (APA, 2013).

Fear of emotion is likely to be a feature of social anxiety disorder given the external scrutiny those with the disorder are expecting. In social situations, the person with social anxiety disorder will begin to experience some anxiety as they reflect on the critical appraisals they expect to receive. Once this anxiety is felt, the person is likely to become increasingly concerned that this heightened anxiety will be visible to others, and lead to further negative evaluation. An example of this process may be illustrated thus: A person becomes anxious in the situation of having to give a speech to a group of strangers. This person may think, "They won't like what I have to say, they will realise I don't know what I'm talking about, and will request that I never present to them again!" As this person becomes more anxious they will likely experience increased

physiological symptoms of anxiety such as trembling, dryness in their mouth, increased sweating and a rapid rate of breathing. As they experience this anxious response, they are likely to become anxious about this response, which may also feed into their experience of social anxiety, "I'm becoming so anxious and everyone will see how anxious I am. They will see me sweating and shaking. They will probably think I am weak and stupid".

Fear of emotion has been shown to be associated with social anxiety disorder in patients presenting for treatment and in non-clinical individuals using a range of measures (see Table 2). The association between fear of emotion and social anxiety disorder is to be expected since one of the three lower-order factors of the Anxiety Sensitivity Index has been conceptualised as Social Concerns. This factor is derived from items on the measure asking, for example, "It is important to me not to appear nervous" and "Other people notice when I feel shaky".

In patients with a diagnosis of social anxiety disorder and presenting for treatment, measures of fear of emotion are elevated compared with normal controls (Collimore & Asmundson, 2014). In a study comparing anxiety sensitivity across the anxiety disorders, Taylor, Koch and McNally (1992) reported a mean score of 24.9 (SD=12.3), significantly different from normal controls of 17.8 (SD=8.8) (Peterson & Reiss, 1987).

Other studies have compared fear of emotion across anxiety disorders. In patients presenting with generalised social anxiety disorder, scores on a measure of fear of emotion (ASI) were elevated (mean=26.02, SD=10.04) compared with norms, although less than those with panic disorder (Hazen, Walker, & Stein, 1995). Similarly, across diagnostic groups consisting of social anxiety disorder alone, panic disorder alone, comorbid social anxiety and panic, and comorbid social anxiety and depression/dysthymia, fear of emotion was elevated as measured by the ASI compared

to previously published norms (Ball, Otto, Uccello, & Rosenbaum, 1995). In a study evaluating the development of a revised fear of emotion measure (ASI-3), patients with social anxiety disorder reported higher scores on the measure compared with undergraduate student controls (Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012).

Table 2
Summary and Features of Studies Investigating the Association of Fear of Emotion and Social Anxiety Disorder (SAD).

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Clinical samples	S				
Ball, Otto, Pollack, Uccello, and Rosenbaum (1995)	102 patients who presented for treatment of panic disorder or social anxiety disorder; 50 met criteria for SAD, and 14 met criteria for SAD and PD		ASI 28.0 (10.9)	Fear of emotion was elevated in individuals with SAD to a similar extent to those with PD	Also investigated co-morbidity with panic disorder and with depression
Collimore & Asmundson, 2014	37 individuals with social anxiety disorder recruited for the study	28 non-clinical controls	ASI-3 30.46 (12.06) Non-clinical 5.32 (4.11)	Individuals with social anxiety disorder reported significantly higher scores than non-clinical controls.	
Hazen, Walker and Stein (1995)	47 individuals recruited through an anxiety disorder research unit at a hospital with a diagnosis of SAD.	Comparison group was 47 individuals with a diagnosis of panic disorder	ASI SAD 26.02 (10.04) PD 33.79 (8.92)	Both groups were elevated on ASI in comparison to norms. Some differentiation between responses on items relating to physical concerns or social concerns.	Subjects could not have comorbid anxiety disorder, except GAD. Participants were all unmedicated when measures were taken.
Rector, Szacun- Shimizu, & Leybman, 2007	50 patients with primary diagnosis of SAD presenting for treatment at an anxiety disorders clinic			Those with SAD had a significantly higher score on Social Concerns subscale compared with those with a primary diagnosis of PD or GAD.	Data were analysed by primary diagnosis, while controlling for anxiety and depression severity. 65.6% of participants were stable on medications.

Study	Population	Controls	Measures	Findings	Comments
Rodriguez et al., 2004	206 individuals with a diagnosis of an anxiety disorder recruited from psychiatric treatment clinics. 60 patients presented who met criteria for SAD.		Mean (Standard Deviation) ASI 45.45 (10.87)	Those with a diagnosis of PD, SAD, and MDD had elevated ASI. Significant associations were found between those with SAD and lower order domains of Mental Incapacitation, and Social Concerns.	86 of the 206 were tested 1 year later, and scores were found to remain stable. Therefore, fear of emotion appears not to change without some kind of intervention
Taylor et al., 1992).	23 patients from a group of 313 presenting for treatment at a university hospital	No control	ASI 24.9 (12.3)	Across a range of anxiety disorders, fear of emotion was seen to be elevated in patients with SAD	
Taylor, et al., 2007	390 patients seeking treatment at two anxiety disorder clinics of which 38 were diagnosed with SAD	4720 undergraduate students	ASI-3 Physical Concerns 6.2 (4.5) Mental Concerns 7.9 (6.1) Social Concerns 17.3 (4.8)	The patients with SAD scored significantly higher on the Social Concerns subscale compared with those with other anxiety disorders and controls.	
Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012	73 patients with SAD from a group of 506 patients with anxiety disorders presenting for treatment	315 undergraduate students	ASI-3 30.4 (13.05) Undergraduate s 13.83 (10.79)	Patients with SAD reported significantly higher scores than undergraduates.	Scores were elevated on all three subscales, and a stronger association was seen on the Social Concerns subscale

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Zinbarg et al., 1997	432 patients presenting for treatment to an outpatient clinic; 71 met criteria for SAD.	32 non-patients	ASI ASI- social concerns 9.51 (3.21)	Main finding was that ASI is hierarchical identifying a unitary anxiety sensitivity construct, with clinically useful subscales identifying predominant feared consequences for different diagnostic groups. In this case, those with a diagnosis of SAD had elevated concerns at the social consequences of their anxiety experience compared with those diagnosed with GAD, OCD, simple phobia and non-clinical controls.	
Student samples Keough, Riccardi, Timpano, Mitchell, & Schmidt, 2010)	418 university students completed measures for course credit.		ASI 15.92 (10.35)	ASI strongly correlated with Social Interaction Anxiety Scale, but participants were in the range of community norms for both ASI and social anxiety measure.	Non-clinical sample.
Norton, Cox, Hewitt and McLeod (1997)	95 undergraduate university students completed measures for course credit.		ASI 17.7 (10.9)	ASI predicted scores on two measures of social anxiety; on the Social Phobia Scale measuring social performance anxiety, ASI was a significantly strong predictor.	Fear of emotion in normal range, as were the two measures of social anxiety. Non-clinical sample.

Study	Population	Controls	Measures	Findings	Comments
Spokas, Luterek & Heimberg, 2009	95 undergraduate students reporting low, mild-moderate or high levels of social anxiety		Mean (Standard Deviation) ACS Ang 3.73 (.86) Pos 3.17 (.83) Anx 3.84 (1.04) Dep 3.44 (1.43)	Students reporting high levels of social anxiety symptoms had significantly higher fear of emotion scores than those reporting mild-moderate or low levels of social anxiety symptoms.	Non-clinical sample
Turk, Heimberg, Luterek, Mennin, & Fresco, 2005	766 undergraduate students participated in study for course credit. Of these 105 reported elevated scores on SIAS (>= 34).	550 of the students who did not meet study criteria for GAD or SAD	ACS Ang 3.82 (.75) Pos 3.34 (.69) Anx 3.68 (.64) Dep 3.55 (.94)	This with GAD or SAD did not differ on fear of emotion measures, except for fear of depression (GAD>SAD). Those with SAD scored higher than controls on each subscale measure.	Non-clinical sample

SAD = social anxiety disorder; GAD = generalized anxiety disorder; PD = panic disorder; OCD = obsessive-compulsive disorder; MDD = major depressive disorder

Elevations in scores of fear of emotion in individuals with social anxiety disorder were found in studies investigating the convergent validity of the lower order factors of the ASI. Higher scores on the Social Concerns subscale were found in those with a diagnosis of social anxiety disorder seeking treatment (Taylor, et al., 2007; Zinbarg, et al., 1997), and those meeting criteria for social anxiety disorder in the presence of a co-morbid anxiety disorder or depression (Rector, Szacun-Shimizu, & Leybman, 2007). A diagnosis of social anxiety disorder was also predictive of scores on the Social Concerns subscale in a cross-sectional study (Rodriguez et al., 2004).

Scores on measures of fear of emotion are also associated with measures of symptoms of social anxiety in individuals who have not been formally diagnosed with social anxiety disorder (i.e. non-clinical samples). In a sample of university students, scores on a measure assessing fears related to social interactions were strongly associated with scores on a measure of fear of emotion (ASI) (Keough, Riccardi, Timpano, Mitchell, & Schmidt, 2010). Fear of emotion scores also predicted aspects of social performance anxiety in a non-clinical sample (Norton, Cox, Hewitt, & McLeod, 1997). In both these studies, however, the symptom scores and the fear of emotion scores were in the normal range. In a non-clinical sample where symptoms of social anxiety were in the clinical range, participants reported more fear of anger, fear of depression, and fear of positive emotions compared with controls (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005), and compared with individuals reporting low or mild-moderate symptoms of social anxiety (Spokas, Luterek & Heimberg, 2009).

In summary, fear of emotion is expected to be elevated in individuals diagnosed with social anxiety disorder, as those with the disorder are convinced that visible signs of emotion (such as anxiety) will be evaluated negatively by others. Research supports this assertion, with elevated scores on measures of fear of emotion being found in patients who have been diagnosed with social anxiety disorder only or in those with

social anxiety disorder co-morbid with other anxiety disorders or depression. The association between fear of emotion and symptoms of social anxiety has also been found to be strong in non-clinical populations.

Generalized Anxiety Disorder. The key feature of generalized anxiety disorder is the presence of excessive levels of worry, and an individual's sense that this worry is uncontrollable or dangerous. The worries that an individual reports are typically across many domains (e.g. relationships, finances, work), are present for at least six months, and are associated with symptoms such as tension, fatigue and irritability (APA, 2013).

Several explanatory models of generalized anxiety disorder have implicated a role for fear of emotion. Borkovec (1994) proposed that the act of worrying serves an avoidant function, in that an individual can intellectually concern themselves with a problem and therefore avoid potential heightened troubling emotions and cognitions associated with the consequences of that problem, should it occur. Another line of investigation has suggested that the worry itself becomes aversive (Wells, 2002) as individuals experience it as potentially dangerous and uncontrollable. This fearful response leads individuals to use unhelpful and impairing strategies to manage the worry, such as substance use or physical and/or cognitive avoidance. Mennin, Turk, Heimberg and Fresco (2002) have proposed further development of these models to explicitly include fear of emotion. They propose that it is an individual's fear of emotion, their ability to control that emotion, and the consequences (behavioural, interpersonal) of not controlling the emotion that lead an individual to fear the internal experience. Given this fear, an individual will use a cognitive strategy, in this case worry, to process the experience leading to a lesser expression of emotion in the short term.

Numerous studies have been reported investigating the relationship between generalized anxiety disorder and fear of emotion (see Table 3).

Table 3
Summary and Features of Studies Investigating the Association of Fear of Emotion and Generalized Anxiety Disorder (GAD).

Study	Population	Controls	Measures	Findings	Comments
			Mean		
			(Standard		
~ .	_		Deviation)		
Clinical samp	bles				
Lee, Orsillo,	33 patients with a primary	33 non-	ACS	Patients with GAD reported significantly	Discriminant function analysis also
Roemer, &	diagnosis of GAD presenting for	anxious	GAD	higher scores on measures of fear of emotion	showed that ACS-dep and ACS-
Allen, 2010	treatment at an anxiety disorders	controls	ACS-ang 3.72	compared with non-anxious controls.	anx were the strongest predictors
	clinic	recruited	(1.01)		of GAD-status
		through	ACS-pos 3.11		
		community	(.88)		
		advertisement	ACS-anx 4.70		
			(.83)		
			ACS-dep 3.81		
			(1.02)		
			Non-GAD		
			ACS-ang 2.49		
			(.66)		
			ACS-pos 2.25 (.64)		
			ACS-anx 2.13		
			(.65)		
			ACS-dep 1.94		
			(.76)		

Study	Population	Controls	Measures	Findings	Comments
•	•		Mean (Standard Deviation)		
Rector, Szacun- Shimizu, & Leybman, 2007	28 patients with primary diagnosis of GAD presenting for treatment at an anxiety disorders clinic			Those with GAD had a significantly higher score on Mental Concerns subscale compared with those with a primary diagnosis of SAD. These scores were not significantly different than those for patients with a primary diagnosis of PD.	Those patients with a primary diagnosis of GAD were also diagnosed with PD/A 10.7%, SAD 35.7%, specific phobia 7.1%, PTSD 3.6%, and 42.9% were experiencing a depressive episode. Data were analysed by primary diagnosis, while controlling for anxiety and depression severity. 65.6% of participants were stable on medications.
Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004	206 individuals with a diagnosis of an anxiety disorder recruited from psychiatric treatment clinics of which 69 patients met criteria for GAD.		ASI 44.23 (11.71) ASI-physical 22.63 (7.07) ASI-mental 9.99 (4.03) ASI-social 12.35 (3.14)	Individuals with a diagnosis of GAD had elevated ASI scores similar to those with depression and other anxiety disorders. A diagnosis of GAD was also significantly associated with the Mental Concerns subscale and the Social Concerns subscale.	86 of the 206 were tested 1 year later, and scores were found to remain stable. Therefore, fear of emotion appears not to change without some kind of intervention. Patients were not assigned a primary diagnosis, and most presented with at least one comorbid anxiety disorder or MDD.
Roemer & Orsillo, 2007	16 clients diagnosed with GAD presenting to a specialist anxiety treatment clinic		ACS-tot 3.97 (0.62)	Compared with previously published data, score on ACS was elevated.	

Study	Population	Controls	Measures	Findings	Comments
Stapinski, Abbott, & Rapee, 2010	126 participants who met criteria for GAD presenting for treatment at an outpatient clinic.	79 non- anxious controls recruited through community advertisement	Mean (Standard Deviation) ACS GAD ACS-tot 3.90 (.76) Non-anxious ACS-tot 2.56 (.70)	Fear of emotion scores were significantly higher for GAD participants than for non-anxious controls, while controlling for depressive symptoms.	Fear of emotion predicted GAD status over and above previously established predictors of GAD e.g. intolerance of uncertainty, metacognitive appraisal
Taylor, Koch, & McNally, 1992.	17 patients from a group of 313 presenting for treatment at a university hospital	No control	ASI 26.2 (10.9)	Across a range of anxiety disorders, fear of emotion was seen to be elevated in patients with GAD	
Taylor, et al., 2007	390 patients seeking treatment at two anxiety disorder clinics of which 30 were diagnosed with GAD	4720 undergraduate students	ASI-3 Physical Concerns 8.1 (5.3) Mental Concerns 8.9 (7.4) Social Concerns 10.5 (7.0)	The patients with GAD scored significantly higher on the three subscales compared with controls. Patients with GAD and PD scored significantly higher on Mental Concerns subscale than patients with other anxiety disorders and controls.	
Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012	53 patients with GAD from a group of 506 patients with anxiety disorders presenting for treatment	315 undergraduate students	ASI-3 29.91 (16.0) Undergraduate s 13.83 (10.79)	Patients with GAD reported significantly higher scores than undergraduates.	Scores were elevated on all three subscales compared with the undergraduate group.

Study	Population	Controls	Measures	Findings	Comments
Zinbarg, Barlow, & Brown, 1997	432 patients presenting for treatment to an outpatient clinic; 44 met criteria for GAD	32 non-patients	Mean (Standard Deviation) ASI ASI-physical 12.87 (7.40) ASI-mental 6.36 (3.85) ASI-social 8.79 (2.74)	Patients with a diagnosis of GAD had elevated scores on the ASI subscales compared with those with no psychological disorder	
Student samp Floyd, Garfield, & LaSota, 2005	342 undergraduate students completed measures for course credit.		ASI 24.59 (5.43)	Score on ASI was a significant predictor of score on worry measure, after controlling for distress.	ASI score was significantly correlated with score on measure of worry (r=.41. p<.05)
Mennin, Heimberg, Turk, & Fresco, 2005	Study 1 – undergraduate sample 538 undergraduate students participated in study for course credit. Of these 47 met criteria for GAD based on self-report diagnostic questionnaire.	491 of the students who did not meet study criteria for GAD	ACS Ang 3.95 (.83) Pos 3.50 (.65) Anx 4.17 (.91) Dep 4.12 (1.00)	Students with self-reported GAD diagnosis reported higher scores on fear of emotion measure compared with controls.	Limitations included use of self- report for GAD diagnosis and no assessment of comorbidity

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
	Study 2 – clinical sample 42 people presenting for treatment at an anxiety disorders clinic	55 individuals without a diagnosis of an anxiety or mood disorder	ACS Ang 4.08 (1.11) Pos 3.27 (.98) Anx 4.52 (.96) Dep 4.11 (1.22)	People diagnosed with GAD reported higher scores on fear of emotion measure compared with controls.	
Roemer, Salters, Raffa, & Orsillo, 2005	Part 1 Undergraduate sample 240 female undergraduate university students		ACS ACS-tot 3.41 (0.72) ACS-ang 3.55 (0.90) ACS-pos 3.18 (0.76) ACS-dep 3.47 (1.22) ACS-anx 3.53 (0.81)	Fear of anxiety was associated with self-reported GAD severity, and fear of depression associated with worry	

Study	Population	Controls	Measures	Findings	Comments
	Part 2 Clinical sample 19 clients diagnosed with GAD presenting to a specialist anxiety treatment clinic		Mean (Standard Deviation) ACS-tot 3.71 (0.64) ACS-ang 3.49 (0.88) ACS-pos 2.96 (0.78) ACS-dep 3.91 (0.87) ACS-anx 4.47 (0.84)	Fear of depression and anxiety was associated with those individuals meeting diagnostic criteria for GAD.	In comparing the clinical sample with the undergraduate sample, the clinical sample scored higher on fear of anxiety and total ACS scores, and showed a tendency to score higher on the fear of depression subscale.
Tull, Stipelman, Salters- Pedneault, & Gratz, 2009.	320 undergraduate students completed measures for course credit. Of these, 99 had a possible diagnosis of GAD based on the Generalized Anxiety Disorder Questionnaire-IV		ASI ASI-physical 11.65 (6.51) ASI-mental 3.63 (2.92) ASI-social 8.15 (2.90)	Compared with students without a self-reported diagnosis of GAD, those with GAD scored higher on each of the subscales of the ASI	
Turk, Heimberg, Luterek, Mennin, & Fresco, 2005	766 undergraduate students participated in study for course credit. Of these 68 met criteria for GAD based on self-report diagnostic questionnaire.	550 of the students who did not meet study criteria for GAD or SAD	ACS Ang 3.80 (.98) Pos 3.23 (.87) Anx 3.86 (.76) Dep 3.87 (1.07)	Students with GAD reported higher scores on fear of emotions, on each of the four subscales, compared with controls. Those with GAD or SAD did not differ on fear of emotion measures, except for fear of depression (GAD>SAD).	Non-clinical sample

GAD = generalized anxiety disorder; MDD = major depressive disorder; PD = panic disorder; ACS-tot= ACS total score; ACS-ang = ACS anger subscale; ACS-pos = ACS positive emotions subscale; ACS-dep = ACS depression subscale; ACS-anx = ACS anxiety subscale

The studies have reported on the investigations of the relationship in individuals diagnosed with generalized anxiety disorder and seeking treatment, in individuals who self-report symptoms consistent with a diagnosis of generalized anxiety disorder, and in individuals reporting elevated levels of worry, the central feature of generalized anxiety disorder. Fear of emotion scales contain items which are consistent with the proposed models of generalized anxiety disorder, such that elevated scores on these measures would be expected. Items from the Anxiety Sensitivity Index include "When I cannot keep my mind on a task, I worry that I might be going crazy", and "It scares me when I am nervous". Similarly, items from the ACS include "When I get nervous, I think that I am going to go crazy", and four items which use the word "worry" in the question; e.g., "I worry about losing self-control when I am on cloud nine".

As has been shown with other anxiety disorders, patients who have been diagnosed with generalized anxiety disorder reported higher scores on measures of fear of emotion than people without an anxiety disorder. In the study reported by Taylor, Koch, and McNally (1992) investigating fear of emotions scores (as measured by ASI) across anxiety disorders, patients diagnosed with generalized anxiety disorder had elevated scores compared with previously published norms. In this study, only 17 patients met criteria for a generalized anxiety disorder diagnosis. In a larger study (Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004), fear of emotion scores were elevated in 69 patients diagnosed with generalized anxiety disorder. These scores were very much higher than those reported by Taylor et al. (1992) which may be due to the patients presenting with comorbid disorders. Patients diagnosed with generalized anxiety disorder also had higher scores on the Mental Concerns and Social Concerns subscales of the ASI. Elevations on each of the three subscales of the ASI were reported by individuals diagnosed with generalized anxiety disorder compared with those with no psychological disorder (Zinbarg, Barlow, & Brown, 1997; Taylor, et al., 2007;

Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012). Elevations on the Mental Concerns subscale were also reported in a study comparing patients with a diagnosis of generalized anxiety disorder to those with social anxiety disorder (Rector, Szacun-Shimizu, & Leybman, 2007).

More studies have been published using the Affective Control Scale (ACS) as the measure of fear of emotion for people meeting criteria for generalized anxiety disorder than any other anxiety disorder. Total scores on the ACS were found to be elevated in patients diagnosed with generalized anxiety disorder presenting for treatment compared with previously published norms (Roemer & Orsillo, 2007). Two more recent studies also reported elevations in ACS scores compared with non-anxious controls recruited for the respective studies both on total scores (Stapinski, Abbott, & Rapee, 2010) and on ACS subscale scores (Lee, Orsillo, Roemer, & Allen, 2010).

The association between fear of emotion and generalized anxiety disorder was also shown in two studies where a diagnosis of presumed generalized anxiety disorder was made on the basis of a validated self-report diagnostic questionnaire (Roemer, Salters, Raffa, & Orsillo, 2005; Mennin, Heimberg, Turk, & Fresco, 2005). In the first study, fear of anxiety was associated with severity of generalized anxiety disorder symptoms. A secondary study measured fear of emotion in 19 patients diagnosed with generalized anxiety disorder presenting to an anxiety disorders treatment clinic. These patients scored higher on the fear of emotion measure than did the undergraduate sample. In the second study, a similar study design was used with the addition of each study including a control group. Both the student group completing the self-report diagnostic measure and the clinical group diagnosed with generalized anxiety disorder scored higher on measures of fear of emotion than their respective control groups.

Individuals without a formal diagnosis of generalized anxiety disorder also have elevated scores on measures of fear of emotion. In a large study of undergraduate

students, those who completed a self-report questionnaire indicating a likely diagnosis of generalized anxiety disorder scored higher on fear of emotion (as measured by ACS) than those students who did not self-report generalized anxiety disorder symptoms (Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). Similarly, in another study with fear of emotion assessed by the ASI, students with self-reported generalized anxiety disorder symptoms scored higher on each of the subscales of the ASI (Tull, Stipelman, Salters-Pedneault, & Gratz, 2009). Worry, the key cognitive feature of generalized anxiety disorder, has also been found to be associated with elevated scores on fear of emotions (Floyd, Garfield, & LaSota, 2005).

In summary, as with other anxiety disorders, fear of emotion is elevated in individuals presenting for treatment diagnosed with generalized anxiety disorder, with or without co-morbid anxiety and depressive disorders. This heightened fear of emotion is also seen in student populations self-reporting generalized anxiety disorder.

Individuals reporting high levels of worry, the key defining feature of generalized anxiety disorder, are also found to have elevated scores on a measure of fear of emotion. These findings were confirmed in a meta-analysis (Naragon-Gainey, 2010) which showed a strong association between fear of emotion and generalized anxiety disorder. The fear of emotion factor was as strong as for those with a diagnosis of panic disorder or posttraumatic stress disorder. In consideration of the lower order factors of the ASI, generalized anxiety disorder was related to all three dimensions with the Mental Concerns and Social Concerns subscales being particularly strong. This association was maintained even after controlling for the effects of shared symptoms of other anxiety and depressive disorders.

Posttraumatic Stress Disorder. Posttraumatic stress disorder is defined by several criteria. The primary criterion is that an individual has been exposed to at least one event where there was the threat of death, injury or assault, and the individual

responded with intense fear, helplessness or horror. Following this experience or experiences, the individual is troubled by memories of the events which are often accompanied by physical sensations of anxiety. The individual avoids cues associated with the events and is constantly on alert for threat (APA, 1994).

Fear of emotion has been proposed as associated with posttraumatic stress disorder in two ways. Firstly, people who are generally more fearful of emotions may experience a traumatic event with increased anxiety as they not only are distressed by the event but also by the high levels of their emotions associated with the experience. This may lead to an increased sensitivity to trauma such that a posttraumatic reaction may arise from a relatively low-level stressor (Marshall, Miles, & Stewart, 2010). Secondly, fear of emotion may be increased in response to a traumatic event along with posttraumatic stress disorder symptoms that arise. An individual may fear the anxiety symptoms that arise during exposure to the traumatic stressor and, through conditioning principles, associate anxiety symptoms with the consequences of exposure to the stressor. As such, a person with posttraumatic stress disorder experiencing a flashback of the traumatic event will begin to feel anxious and to experience symptoms associated with anxiety (Taylor, 2003). At this point, the individual's fear of that emotional response heightens the anxiety. For example, once a person is aware of the anxiety symptoms they may think "Oh no, not again ... this can't be good for me ... I will probably have a heart attack or go crazy." As such, they not only experience the anxiety as unpleasant but also escalate the anxiety response through fear of the consequences of being anxious in a similar way to an individual experiencing panic symptoms (see panic section).

Fear of emotion has been investigated in relation to its association with posttraumatic stress disorder and posttraumatic stress disorder symptoms in individuals seeking treatment and in healthy individuals screened for symptoms. A summary of

studies is shown in Table 4. Given the very high levels of anxiety symptoms reported by individuals with posttraumatic stress disorder, it would be expected that fear of emotion would be elevated in such individuals. The lower order factors of Physical Concerns and Mental Concerns are associated with individuals experiencing posttraumatic stress disorder symptoms (Naragon-Gainey, 2010). This is also to be expected as the items on those subscales relate directly to catastrophic fears of anxiety sensations.

Fear of emotion is elevated in individuals receiving treatment for posttraumatic stress disorder compared with normal controls. In patients presenting for treatment at a specialised posttraumatic stress disorder treatment centre (Schoorl, Van Mil-Klinkenberg & Van Der Does, 2015), scores on the ASI were elevated at pre-treatment (M=37.1, SD=12.0) compared with previously reported normal controls (mean=19.0, SD = 9.1; Peterson & Reiss, 1992). Similar elevations (M=38.4, SD=7.7) were observed in individuals recruited to a treatment trial who met diagnostic criteria for posttraumatic stress disorder (Wald & Taylor, 2007). In this treatment trial, fear of emotion decreased during the treatment as did patients' reported symptoms of posttraumatic stress disorder.

In a related study, Wald and Taylor (2008) found that fear of emotion correlated significantly with posttraumatic stress disorder symptom severity in patients with a diagnosis of posttraumatic stress disorder. Prior to treatment patients reported a mean score on a fear of emotion measure (ASI-R) of 77.0 (SD=18.0), which was significantly elevated compared with previously published norms (mean=25.7, SD=19.6; Deacon, Abramowitz, Woods, & Tolin, 2003).

Similarly, Lang, Kennedy and Stein (2002) reported on a study investigating the relationship between posttraumatic stress disorder and fear of emotion in women. This study benefited from including a control group. ASI scores were elevated in women diagnosed with posttraumatic stress disorder (mean=30.7, SD=16.5) compared with those in the control group (mean=7.1, SD=6.8).

Fear of emotion was seen to predict posttraumatic stress disorder symptoms in patients seeking treatment following motor vehicle accidents (Federoff, Taylor, Asmundson, & Koch, 2000). Following treatment, the reduction in fear of emotion also predicted reduction in posttraumatic stress disorder symptoms.

The relationship between fear of emotion and posttraumatic stress disorder has also been investigated using the ACS as a measure of fear of emotion. In a group of military veterans with chronic posttraumatic stress disorder, fear of emotion was a predictor of change scores over treatment (Price, Monson, Callahan, & Rodriguez, 2006). Further, fear of emotion predicted change scores in other aspects of posttraumatic stress disorder psychopathology (re-experiencing, avoidance/numbing) and symptoms of comorbid depression. In a similar population, treatment outcome was predicted by fear of anger as a subscale of fear of emotion from a range of potential mediators including social support, problematic relationships, and therapeutic alliance (Forbes et al., 2008).

The relationship between fear of emotion and posttraumatic stress disorder symptoms has also been investigated using other study designs. In a longitudinal study, fear of emotion predicted posttraumatic stress disorder symptom severity at six and 12-month time points even after adjusting for initial posttraumatic distress (Marshall, Miles, & Stewart, 2010). Keogh, Ayers, and Francis (2002) used a prospective design to investigate the association between fear of emotion and the development of posttraumatic stress disorder symptoms following childbirth. The mean of the pre-natal fear of emotion scores were in the normal range, but still predicted posttraumatic stress disorder symptoms.

Table 4
Summary and Features of Studies Investigating the Association of Fear of Emotion and Posttraumatic Stress Disorder (PTSD).

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Clinical samp	les				
Federoff, Taylor, Asmundson, & Koch, 2000	81 patients seeking treatment, 48 for pain as a result of motor vehicle accident (of which 8 met criteria for PTSD), and 33 for MVA-induced PTSD.	No control	ASI	Fear of emotion predicted PTSD symptoms.	For those completing treatment, reduction in fear of emotion predicted a reduction in PTSD symptoms. 20% of patients were taking medication.
Forbes, Parslow, Creamer, Allen, McHugh & Hopwood, 2008	103 male combat veterans attending a specialist treatment program		ACS ACS-tot: 4.40 (.69) ACS- ang: 4.96 (.75) ACS-pos: 3.93 (.83) ACS-anx: 4.38 (.54) ACS-dep: 4.64 (.65)	Anger was a predictor of treatment outcome. Fear of emotion (anger) accounted for variance between anger and outcome.	The study investigated the influence of anger on PTSD treatment outcome and potential mediating factors. Patients also reported comorbidities of major depressive, substance use, and anxiety disorders.

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Lang, Kennedy, & Stein, 2002.	72 women in three groups: 30 had no history of exposure to trauma 23 had experienced intimate partner violence, but did not have PTSD		ASI 7.1 (6.8) 17.0 (11.6)	Scores of fear of emotion were elevated in those with PTSD. Scores in those with PTSD were elevated on all subscales, with two subscales (physical, mental) being significantly higher than in those who had been exposed to trauma.	Unusually low scores in non-trauma group
	19 had experienced intimate partner violence and had PTSD		30.7 (16.5)	Fear of emotion scores were significantly associated with PTSD symptom severity.	
Marshall, Miles, & Stewart, 2010	677 patients who had experienced traumatic physical injuries (such as motor vehicle accident, knife wounds, gunshot wounds)		ASI Baseline 17.8 (12.9) 6-months 17.1 (13.8) 12-months 16.3 (14.0)	Fear of emotion predicted PTSD symptom severity even after adjusting for initial post-traumatic distress. This relationship was also reciprocal, with PTSD symptom severity also predicting fear of emotion.	Longitudinal study.
Price, Monson, Callahan, & Rodriguez, 2006	81 males with military related PTSD seeking treatment at a specialist PTSD treatment centre of which 40 completed pre/post measures		ACS	Fear of emotion was associated with PTSD symptoms. Changes in fear of emotion during treatment predicted change in PTSD symptoms.	Bivariate correlations between ACS and PTSD symptoms: ACS-tot: .38 ACS-ang: .25 ACS-pos: .26 ACS-anx: .24 ACS-dep: .31
Schoorl, Van Mil- Klinkenberg & Van Der Does, 2015,	101 outpatients diagnosed with PTSD attending a specialised treatment centre		ASI 37.1 (12.0)	Fear of emotion scores were significantly higher than previously published norms.	

Study	Population	Controls	Measures	Findings	Comments
Wald & Taylor, 2007	7 patients recruited to participate in treatment trial with diagnosis of PTSD		Mean (Standard Deviation) ASI 38.4 (7.7)	Fear of emotion was associated with PTSD symptoms. Score of fear of emotion and PTSD symptoms both reduced over treatment.	
Wald and Taylor, 2008	23 patients recruited from a study investigating the efficacy of interventions for PTSD.	No control	ASI-R 77.0 (18.0)	Patients with a diagnosis of PTSD showed elevations on a measure of fear of emotion (compared with previously published norms). Fear of emotion scores were significantly positively correlated with PTSD symptom scores.	11 patients were medicated. 13 patients met criteria for depression or another anxiety disorder.
Screened sam	ples				
Asmundson and Stapleton, 2008	138 police officers who screened positive to PTSD.		ASI 24.6 (12.5) (for those with probable PTSD) 13.7 (10.4) (for those without PTSD)	Officers with probable PTSD scored significantly higher on fear of emotion (total score and on subscales) compared with those who did not have PTSD. The somatic subscale of the ASI was a significant predictor of PTSD symptoms.	44 officers had 'probable' PTSD, 134 did not have PTSD. Co-morbid psychopathology was not assessed. PTSD was not diagnosed with structured clinical interview.
Bernstein, Zvolensky, Feldner, Lewis, Fauber, Leen- Feldner, & Vujanovic, 2005	331 young adults recruited through a university and its local community. 254 self-reported at least one traumatic event.		ASI – see comments	Fear of emotion and number of traumatic events predicted higher scores of PTSD symptoms, controlling for negative affectivity.	Authors used a "taxon" scale consisting of 8 items from the ASI. Values are therefore not reported in this table

Study	Population	Controls	Measures	Findings	Comments
Feldner, Lewis, Leen- Feldner, Schnurr, & Zvolensky, 2006	61 students who had experienced a traumatic event		Mean (Standard Deviation) ASI 20.15 (11.25)	Fear of emotion moderated the effect of frequency of traumatic event exposure on PTSD symptoms. In those high in fear of emotion, greater trauma frequency was associated with high levels of PTSD symptoms.	Other studies have shown that fear of emotion is related to PTSD symptoms; this study looks at other risk factors (frequency of traumatic event exposure).
Keogh, Ayers, & Francis, 2002	40 women at 36 weeks gestation		ASI 17.98 (7.00)	Prenatal AS was associated with PTSD symptoms following childbirth, controlling for post-natal psychiatric state.	Prospective study. In this study, PTSD symptoms were measured two weeks post- partum, which would suggest a diagnosis of Acute Stress Disorder, rather than PTSD.

PTSD = Posttraumatic Stress Disorder; MVA = motor vehicle accident; ACS-tot= ACS total score; ACS-ang = ACS anger subscale; ACS-pos = ACS positive emotions subscale; ACS-dep = ACS depression subscale; ACS-anx = ACS anxiety subscale

Individuals reporting posttraumatic stress disorder symptoms have also been found to report higher scores of fear of emotion, even in the absence of a diagnosis of posttraumatic stress disorder. Police officers with elevated posttraumatic stress disorder symptom scores scored highly on a measure of fear of emotion compared with police officers with low scores on the posttraumatic stress disorder symptom measure (Asmundson & Stapleton, 2008). Scores on two of the subscale domains, Physical Concerns and Mental Concerns, were also elevated in those with higher scores of posttraumatic stress disorder symptoms.

The association between fear of emotion and posttraumatic stress disorder symptoms was also assessed in a community sample. Bernstein et al. (2005) found that the association existed, independent of negative affect. Further, they suggested that individuals with high fear of emotion may be more at risk of developing posttraumatic stress disorder symptoms as a result of a traumatic event than those with lower levels of fear of emotion.

Fear of emotion has also been shown to moderate the effect of traumatic event frequency on posttraumatic stress disorder symptoms (Feldner, Lewis, Leen-Feldner, Schnurr, & Zvolensky, 2006). This moderating effect was over and above other variables potentially related to the development of posttraumatic stress disorder symptoms, such as negative affectivity, gender, alcohol use and socio-economic status.

In summary, fear of emotion has been shown to be associated with posttraumatic stress disorder symptoms, and shown to be elevated in individuals meeting diagnostic criteria for posttraumatic stress disorder. In those with posttraumatic stress disorder, scores on measures of fear of emotion are significantly elevated compared with normal controls. The association between fear of emotion and posttraumatic stress disorder symptoms has been shown in cross-sectional studies, and in longitudinal and

prospective studies. The relationship has been found in a range of non-clinical populations, and across measures of fear of emotion.

Obsessive-Compulsive Disorder. The core feature of obsessive-compulsive disorder is the presence of repetitive obsessions or compulsions. Obsessions are cognitions that occur repeatedly and which the individual finds anxiety-provoking due to their content that is seen as "ego-dystonic" (inconsistent with a person's values, not welcome, and uncontrollable). Compulsions are driven behaviours performed repeatedly in response to the obsession to prevent a feared outcome. In order to meet criteria for the disorder, the obsession or compulsion needs to be excessive, distressing and impairing, indicated by taking up more than one hour per day or interfering with a person's regular functioning (APA, 2013).

The process by which fear of emotion may be involved in the maintenance of obsessive-compulsive disorder has perhaps best been explained by Freeston, Rheaume and Ladouceur (1996). They proposed a mechanism where a person with obsessive-compulsive disorder holds beliefs that the anxiety in response to obsessive thoughts is unacceptable and/or dangerous. Given the unpleasant nature of the physical sensation of anxiety, people with obsessive-compulsive disorder may interpret these symptoms as further evidence of the dangerousness of anxiety, thus strengthening their beliefs. This may also lead to patients undergoing treatment to not engage in therapy with an exposure component, due to the risk of heightened anxiety. For example, a person may have an intrusive thought that they may yell out an inappropriate comment in public. They may then be concerned that if they have the thought they will become anxious, and if anxiety escalates they may lose control. Should they lose control, they are then likely to yell out the inappropriate comment, resulting in people noticing them, which may further exacerbate the anxiety. The person may believe that the continuing

escalation of the anxiety may lead them to completely lose control or to go crazy and be hospitalised.

Consistent with the proposed mechanism of the association between fear of emotion and obsessive-compulsive disorder, scores on fear of emotion measures have been found to be elevated in patents diagnosed with obsessive-compulsive disorder who present for treatment (see Table 5).

In the important early work of Taylor, Koch and McNally (1992), patients with obsessive-compulsive disorder within an anxiety disorders sample were found to have elevated scores on fear of emotion, as measured by ASI. The scores of these patients were higher than previously published norms, but lower than those presenting with panic disorder. Similar results were found in a smaller study comparing patients with obsessive-compulsive disorder to those with panic disorder (Zeitlin & McNeilly, 1993). This pattern of results was also confirmed in a review by Robinson and Freeston (2014). The results of a study investigating potential differences in patients with proposed subtypes of obsessive-compulsive disorder showed fear of emotion scores were elevated across all subtypes (Calamari et al., 2004).

Patients with obsessive-compulsive disorder also show elevated scores of fear of emotion compared with control groups who do not meet criteria for an anxiety disorder, and who are randomly chosen (Zinbarg, Barlow, & Brown, 1997) or matched (Amir, Cobb, & Morrison, 2008).

Table 5
Summary and Features of Studies Investigating the Association of Fear of Emotion and Obsessive-Compulsive Disorder.

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Clinical samp	les		,		
Amir, Cobb, and Morrison, 2008	19 individuals presenting for assessment at an anxiety disorders treatment clinic and meeting criteria for a diagnosis of OCD	19 community volunteers who did not meet criteria for any anxiety disorder	ASI 31.3 (11.19)	Scores on measure of fear of emotion were significantly higher for those with OCD compared with non-anxious controls	
Calamari et al., 2004	220 patients presenting for treatment at two specialist OCD clinics. These were made up of two samples of 106 and 114 patients. All met criteria for DSM-IV diagnosis for OCD	No control	ASI 26.8 (13.7)	Fear of emotion score was elevated compared with previously published norms.	Patients were also stratified according to OCD subgroups; no differences were found between the means of fear of emotion scores between five proposed subgroups
Calamari, Rector, Woodard, Cohen, and Chik, 2008	280 patients diagnosed with OCD seeking treatment	No control	ASI 27.29 (13.15)	Scores on measure of fear of emotion were elevated compared with previously published norms	Results also showed stronger loadings for Mental Concerns and Physical Concerns compared with earlier studies.
Deacon & Abramowitz, 2006	232 patients seeking treatment for a primary anxiety disorder. 92 met criteria for a diagnosis of OCD	453 undergraduate students	ASI-R	Fear of emotion score was significantly higher in the patients with OCD compared with the undergraduate sample	Scores on fear of emotion measure were moderately correlated with scores on measure of OCD symptoms.

Study	Population	Controls	Measures	Findings	Comments
Raines, Oglesby, Capron & Schmidt, 2014	76 outpatients diagnosed with OCD attending a university clinic		Mean (Standard Deviation) ASI Total 27.37 (13.39) ASI-Physical Concerns 13.41 (8.21) ASI-Mental Concerns 5.53 (3.84) ASI-Social Concerns 8.36 (3.35)	Scores on total scale and each of the subscales were similar to other published clinical samples	
Taylor, Koch and McNally, 1992	67 patients from a group of 313 presenting for treatment at a university hospital	No control	ASI 25.4 (12.4)	Across six anxiety disorders, fear of emotion was seen to be elevated in patients with OCD, compared with previously published norms.	
Taylor, et al., 2007	390 patients seeking treatment at two anxiety disorder clinics of which 102 were diagnosed with OCD	4720 undergraduate students	ASI-3 Physical Concerns 8.3 (6.2) Mental Concerns 7.7 (6.0) Social Concerns 10.3 (6.7)	The patients with OCD scored significantly higher on the three subscales compared with controls.	

Study	Population	Controls	Measures	Findings	Comments
Zeitlin and McNally, 1993	31 patients with OCD and 27 with panic disorder who sought treatment at an anxiety disorders clinic	No control	Mean (Standard Deviation) ASI 27.94 (11.67)	Scores on measure of fear of emotion were elevated compared to previously published norms.	Patients with panic disorder scored higher on fear of emotion measure
Zinbarg, Barlow, and Brown, 1997	432 patients presenting for treatment to an outpatient clinic; 25 met criteria for OCD.	32 non-patients	ASI ASI-Physical Concerns 12.82 (8.15) ASI-Mental Concerns 6.38 (5.04) ASI-Social Concerns 7.85 (3.46)	Hierarchical structure of anxiety sensitivity shown in patients with anxiety disorders. Patients with a diagnosis of OCD had elevated scores relative to other anxiety disorders on Mental Concerns and Physical Concerns subscales.	
Student samp	les				
Stern, Nota, Heimberg, Holoway, & Coles, 2014	21 undergraduate students reporting symptoms of OCD at a level consistent with diagnosis of OCD	26 students reporting or very few symptoms of OCD	ACS ACS-ang: 3.72 (.79) ACS-pos: 3.53 (.62) ACS-anx: 3.61 (.74) ACS-dep: 3.58 (.89)	Fear of emotion scores were significantly higher in the high OCD symptom group compared with the no or low OCD symptom group.	

OCD = obsessive-compulsive disorder

Elevated fear of emotion scores were seen in patients with obsessive-compulsive disorder on the lower order factors of the ASI (Raines, Oglesby, Capron & Schmidt, 2014). Patients with obsessive-compulsive disorder had higher scores on the Mental Concerns subscale, similar to those with panic disorder (Zinbarg et al., 1997; Calamari et al 2008). Scores were also elevated on the Physical Concerns subscale (Zinbarg et al., 1997; Taylor, et al., 2007) and on the Social Concerns subscale (Taylor, et al., 2007; Calamari, Rector, Woodard, Cohen, & Chik, 2008).

Fear of emotion scores in patients with obsessive-compulsive disorder were also moderately correlated with scores on measures of obsessive-compulsive disorder symptoms (Calamari et al., 2008; Deacon & Abramowitz, 2006; Stern, Nota, Heimberg, Holaway, & Coles, 2014).

In summary, in theoretical models of obsessive-compulsive disorder, fear of emotion is described as a possible factor which can maintain and exacerbate the disorder. Individuals experiencing intrusive and distressing thoughts may become fearful of their anxious responses due to the perceived negative consequences of experiencing these anxiety symptoms. In studies measuring fear of emotion, patients diagnosed with obsessive-compulsive disorder have reported higher scores on fear of emotion measures compared with non-anxious controls, undergraduate students, and previously published norms. Measures of symptoms of obsessive-compulsive disorder are also moderately correlated with fear of emotion scores.

Hypochondriasis. Hypochondriasis is a psychological disorder characterised by a person's concerns about their health and possible disease. The person will frequently present for treatment or seek reassurance for their concerns, but their illness fears will persist in spite of this (APA, 1994). In diagnostic guidelines, hypochondriasis is grouped with somatic symptom disorders since the person's health concerns are disproportional to the symptoms that they are experiencing. Hypochondriasis is being

considered here as part of the investigation of fear of emotions and anxiety disorders as researchers consider hypochondriasis to be a severe form of health anxiety, and fear or anxiety is a necessary criterion for the diagnosis to be made (Abramowitz, Olatunji, & Deacon, 2007).

The proposed mechanism by which fear of emotion may be linked to hypochondriasis is closely related to the mechanism by which fear of emotion is linked to panic disorder. Individuals with hypochondriasis are likely to notice ambiguous physical symptoms and sensations and interpret these as threatening due to the possibility that the sensations may be associated with development or exacerbation of serious disease. This anxious response leads to further physical sensations which may be interpreted as a worsening of the feared disease or the development of further medical problems, leading the individual to engage in self-checking behaviours, reassurance seeking or presenting for medical assessment and treatment.

Despite the theoretical similarities with panic disorder, relatively few studies have investigated the association between fear of emotion and hypochondriasis/health anxiety. Individuals with hypochondriasis are likely to show elevated scores on measures of fear of emotion given the wording on the measures. For example, the ASI contains items which specifically link sensations of emotion to disease. Item 11 is "When my stomach is upset, I worry that I might be seriously ill" and Item 15 is "When I am nervous, I worry that I might be mentally ill" (Peterson and Reiss, 1992).

Table 6 shows the studies where fear of emotion scores have been reported for patients diagnosed with hypochondriasis presenting for treatment and for a study of depressed patients presenting for treatment who also had hypochondriacal concerns. In patients with a primary diagnosis of hypochondriasis, scores on measures of fear of emotion were elevated compared with previously published norms (Deacon & Abramowitz, 2008; Hedman, et al., 2010; Hedman, et al., 2011; Wheaton, Deacon,

McGrath, Berman, & Abramowitz, 2012). The measures of fear of emotion were the ASI, the ASI-R and the ASI-3. Further investigation of the subscales of these measures showed elevations of fear of cardiovascular and respiratory symptoms (ASI-R) and Mental, Physical and Social Concerns (ASI-3).

Elevated fear of emotion scores were also seen in a sample of patients with a primary diagnosis of depression (Otto, Demopulos, McLean, Pollack, & Fava, 1998). While 29% of these also met criteria for an anxiety disorder, none currently met criteria for panic disorder, nor had a history of panic disorder. Fear of emotion was the strongest predictor of hypochondriacal concerns in this group from potential predictors including measures of mood (depression, anxiety, anger) and somatic symptoms.

A literature search failed to find any published research investigating fear of emotion and hypochondriasis using the ACS as the fear of emotion measure.

The elevations in fear of emotion scores are not as great as those seen with the previously reviewed anxiety disorders. This may reflect that physiological symptoms associated with emotions can also be interpreted as symptoms of medical problems which hold a different fear. However, at this point the literature is relatively sparse and further measurement of fear of emotion in health anxiety is warranted.

In summary, fear of emotion scores are likely to be elevated in patients meeting diagnostic criteria for hypochondriasis, given the attention these individuals give to somatic symptoms and the anxious interpretations they make about those symptoms.

The summarised research supports this assertion with elevated scores being seen on fear of emotion measures. Fear of emotion is also associated with hypochondriacal concerns in depressed patients without co-morbid panic disorder.

Table 6
Summary and Features of Studies Investigating the Association of Fear of Emotion and Hypochondriasis (HC).

Study	Population	Controls	Measures	Findings	Comments
Deacon and Abramowitz, 2008	23 patients from a group of 94 treatment seeking adults		Mean (Standard Deviation) ASI-R ASIresp 19.83 (9.58) ASIcardio 22.56 (12.42)	Scores on the fear of cardiovascular symptoms subscale of the ASI-R were significantly higher for patients with HC than those with PD or OCD.	
Hedman, et al., 2010	22 patients who completed treatment at a mental health outpatients clinic, and who met criteria for a diagnosis of hypochondriasis and disease phobia		ASI 27.88 (14.48)	Scores on fear of emotion measure were elevated compared with previously published norms	Patients included in this study may have had higher scores due to more severe presentation (patients met criteria for hypochondriasis and disease phobia)
Hedman, et al., 2011	81 treatment seeking patients diagnosed with hypochondriasis. These were split into two groups; one group (<i>n</i> =40) received an active treatment, the other group (<i>n</i> =41) acted as a control condition.		ASI Treatment condition 26.0 (12.1) Control condition 26.8 (11.0)	All patients with hypochondriasis showed elevated fear of emotion compared with previously published norms	
Otto, Demopulos, McLean, Pollack, & Fava, 1998	100 patients diagnosed with major depression. Of these 29 had a comorbid anxiety disorder (not hypochondriacal disorder)		ASI 25.6 (12.3)	Compared with previously published norms, scores were elevated on measure of fear of emotion	All patients were assessed for hypochondriacal concerns. Fear of emotion was found to be the strongest predictor of these.

Study	Population	Controls	Measures	Findings	Comments
Wheaton, Deacon, McGrath, Berman, & Abramowitz, 2012	29 patients with hypochondriasis from a group of 506 patients with anxiety disorders presenting for treatment		Mean (Standard Deviation) ASI-3 27.9 (17.39)	Compared with previously published norms, patients with hypochondriasis reported elevated scores on fear of emotion measure	Scores were elevated on all three subscales, and a stronger association was seen on the Physical Concerns subscale

Specific phobia. Specific phobia is characterised by a significant immediate fear reaction, often panic-like, to a particular object, activity, animal or situation. This reaction is predicable in that it nearly always occurs in the presence of the stimulus. The individual understands the reaction to be excessive or irrational and often believes it to be automatic or uncontrollable. To meet diagnostic criteria, the fear has to interfere with the person's functioning, either at work, socially or a home (APA, 2013).

Fear of emotion may serve as a predisposing factor in the development of specific phobia or might be a maintaining factor in its continuance. Individuals with elevated fear of emotion perceive experiencing anxiety as particularly aversive and anticipate harmful effects either physically, mentally or socially. This fear leads to hypervigilance towards anxiety symptoms which may lower the threshold for developing an anxiety disorder. Once an individual has experienced an aversive anxious response in the presence of a phobic stimulus, they become hypervigilant to both the feared stimulus and the physical sensations of the anxiety response, which serves to intensify the emotional reaction (Killgore et al., 2011).

Unlike the other anxiety disorders, the relationship between fear of emotion and specific phobia is not clear. Table 7 summarises studies on individuals meeting criteria for specific phobia where fear of emotion was measured using the ASI.

Fear of emotion scores were elevated compared to previously published norms in two studies. In the first study, patients with specific phobia presenting for treatment from among a large sample of treatment-seeking patients had scores in the range of other anxiety disorders, and these scores were significantly higher than scores in a community control group (Carleton, Abrams, Asmundson, Antony, & McCabe, 2009). In the second study, university students with specific phobia (animal subtype) reported elevated scores on the fear of emotion measure. However, there were no differences between the scores of those students with specific phobia and the healthy control group

(Lueken, et al., 2011). This was not surprising as there was considerable variation of scores in the small sample.

Fear of emotion scores were similar to community norms in five published studies of patients with specific phobias. In two of these (Britton, Gold, Deckersbach, & Rauch, 2009; Rosso et al., 2010), the patients recruited for the study meeting criteria for specific phobia recorded scores in the normal range, and these were significantly higher than matched control groups. However, individuals with specific phobia were also shown not to differ from healthy controls (Killgore et al., 2011). The remaining two studies did not employ control groups, but participants with specific phobia (animal subtype) scored in the range of previously published norms (De Quervain et al., 2011; Diemer et al., 2013).

There have been no published studies investigating fear of emotion and specific phobia using ACS as the measure.

In summary, the relationship between fear of emotion and specific phobia is not as clear as it is with other anxiety disorders. Individuals who meet criteria for specific phobia generally score higher on a measure of fear emotion compared with healthy controls, but these scores often fall within the range of previously published community norms. This considerable overlap between these two groups may be explained by the high lifetime prevalence of approximately 40% for fears, that is sub-clinical phobia, of specific objects or situations (Curtis, Magee, Eaton, Wittchen, & Kessler, 1998; Depla, ten Have, van Balkom, & de Graaf, 2008).

Table 7
Summary and Features of Studies Investigating the Association of Fear of Emotion and Specific Phobia.

Study	Population	Controls	Measures	Findings	Comments
Britton, Gold, Deckersbach, and Rauch, 2009	12 individuals recruited for study who met criteria for specific phobia (snake, spider, rodent)	12 matched controls	Mean (Standard Deviation) ASI Specific phobia 16.8 (9.0) Controls 8.8 (4.9)	Individuals with specific phobia scored significantly higher on fear of emotion measure compared with controls.	Individuals with specific phobia reported scores similar to previously published community norms.
Carleton, Abrams, Asmundson, Antony, & McCabe, 2009	14 individuals who met criteria for specific phobia from a sample of 418 individuals presenting for treatment of an anxiety or mood disorder	Community convenience sample of 102 individuals	ASI Specific phobia 25.07 (18.49) Controls 15.26 (12.59)	Individuals with specific phobia scored significantly higher on measure of fear of emotion compared with community controls.	Scores for those with specific phobia were elevated compared with previously published norms.
De Quervain et al., 2011	42 participants recruited for the study. Each met criteria for specific phobia (environmental). Participants were randomised to two groups; one receiving a placebo treatment and one an active treatment.		ASI Active treatment 16.2 (1.8) Placebo treatment 17.9 (1.8)	Individuals with specific phobia reported scores similar to previously published community norms.	The mean scores for the two groups were not significantly different.
Diemer et al., 2013	58 patients recruited for medication treatment trial using virtual reality exposure. Patients met criteria for specific phobia (spiders)		ASI 12.14 (7.07)	Participants' scores on measure of fear of emotion were similar to previously published norms	

Study	Population	Controls	Measures	Findings	Comments
Killgore et al., 2011	17 individuals meeting criteria for specific phobia (animal)	22 healthy controls	Mean (Standard Deviation) ASI Specific phobia 14.7 (8.0) Controls 11.2 (5.8)	There was no significant difference between scores on measure of fear of emotion between those with specific phobia and healthy controls.	Scores were similar to previously published community norms.
Lueken et al., 2011	24 university students who met criteria for specific phobia (snake or dental)	17 healthy controls (a selection of students scoring in the lowest quartile on screening measures for dental and snake phobia)	ASI Dental Phobia 22.1 (11.17) Spider 22.0 (7.36) Controls 14.9 (8.31)	Phobic subjects reported higher scores on fear of emotion measure compared with previously published norms, however these were not significantly different from healthy controls (p =.054).	
Rosso et al., 2010	19 individuals recruited for study who met criteria for specific phobia (small animal)	20 matched controls	ASI Specific phobia 15.37 (7.84) Controls 10.70 (6.04)	Scores on measure of fear of emotion were significantly higher for those with specific phobia compared with healthy controls.	Scores for those with specific phobia were similar to previously published community norms.

Fear of emotion and depression. Major depressive disorder is a mood disorder typified by the presence of depressed mood or loss of interest or pleasure in most activities over at least a two-week period. At least four other symptoms must be present for a clinical diagnosis to be made; these symptoms include weight or appetite changes, sleep changes, fatigue, worthlessness or guilt, indecisiveness or suicidal thinking (APA, 2013).

Despite fear of emotion being originally conceptualised with regard to panic disorder, and later all anxiety disorders, the construct has also been observed in individuals experiencing depression. While there is high comorbidity between anxiety and depression, the association between fear of emotion and depression exists outside of the presence of anxiety and anxiety symptoms.

Two mechanisms have been suggested to explain the role of fear of emotion in depression. The first mechanism explains fear of emotion as a vulnerability factor for developing depression. Individuals with a tendency to have distorted cognitions about symptoms of depression may be more prone to symptoms of depression. For example, concerns about the meaning of impaired concentration or decision-making ability may cue individuals to notice these symptoms, leading to an anxious response which is likely to result in exacerbation of these depressive symptoms (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995).

A second proposed mechanism is that the experience of depression is aversive, priming individuals to be fearful of experiencing future symptoms of depression. Given the catastrophic nature of the appraisal of these symptoms, individuals are prone to experience depressed mood based on the conclusion that they will be suffering irreversible mental incapacitation (Cox, Borger, & Enns, 1999). These concerns are evaluated in the fear of emotion instruments with items such as "When I cannot keep

my mind on a task, I worry that I might be going crazy", and "Depression is scary to me

– I am afraid that I could get depressed and never recover".

Fear of emotion scores have been found to be elevated in patents presenting for treatment with depressive disorders, with or without a comorbid anxiety disorder, and in those with elevated symptoms of depression. Fear of emotion scores are also associated with symptoms of depression, and can predict depression symptom severity (see Table 8).

In patients diagnosed with major depressive disorder, fear of emotion scores are consistently elevated when compared with control groups, consisting of those with no history of panic attacks (Blais et al., 2001) or mood or anxiety disorders (Otto, Pollack, Fava, Uccello, & Rosenbaum, 1995). Elevated fear of emotion scores are also seen in depressed patients when comparing with previously published norms (Taylor, Koch, Woody, & McLean, 1996; Cox, Enns, Freeman, & Walker, 2001), and in a sample of older adults (Bravo & Silverman, 2001).

Given the high comorbidity between depression and anxiety disorders, studies have compared fear of emotion in patients with depression and anxiety to those with depression only. While these studies found that all groups had elevated scores of fear of emotion compared with previously published norms, one study showed a significant difference between those with depression and anxiety and those with depression only (Cox, Enns, & Taylor, 2001), while another study found no difference between groups (Otto et al., 1995).

Fear of emotion scores as measured by the Mental Concerns subscale of the ASI were associated with depression symptom scores in a group presenting with either panic disorder, social anxiety disorder or generalized anxiety disorder. The subscale scores also discriminated between the patients experiencing a current depressive episode, and those who were not currently depressed (Rector, Szacun-Shimizu, & Leybman, 2007).

Table 8
Summary and Features of Studies Investigating the Association of Fear of Emotion and Depression.

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation)		
Clinical samp	les		,		
Blais et al. (2001)	142 outpatients with a diagnosis of MDD with no panic disorder	50 undergraduate students with no history of panic attacks	ASI 26.0 (11.3)	Patients with depression scored significantly higher on a fear of emotion measure compared with healthy controls	
Bravo and Silverman (2001)	53 older adults diagnosed with MDD	53 older adults with low scores on depression screening measure	ASI Depressed 27.21 (14.94) Control 18.30 (11.91)	Fear of emotion scores were significantly higher than in those who had low depression symptom scores	Fear of emotion scores in the depressed group were higher than previously published norms.
Cox, Enns, Freeman, and Walker (2001)	51 patients with major depressive disorder referred for treatment		ASI 31.29 (10.38)	Patients with MDD had elevated scores on a measure of fear of emotion compared with previous published norms.	Patients were diagnosed with comorbid dysthymia (18%), PD (18%) and SAD (16%).
Cox, Enns, and Taylor (2001)	142 outpatients referred to a mood disorders clinic for treatment. All patients had a primary diagnosis of major depressive disorders, and 53 met criteria for a comorbid anxiety disorder.		ASI Depression only 30.53 (12.27) Depression and anxiety 35.17 (10.43)	Patients with depression had elevated scores on a measure of fear of emotion. Those with depression and comorbid anxiety had significantly higher scores than those with depression and no comorbid anxiety.	Scores on Mental Concerns subscale predicted depression symptom severity.

Study	Population	Controls	Measures	Findings	Comments
Liverant, Brown, Barlow and Roemer (2008)	60 individuals presenting at an anxiety and mood disorders treatment clinic with a diagnosis of MDD or dysthymia.		Mean (Standard Deviation) ACS	Fear of emotion was significantly positively correlated with scores on two depression symptom measures.	83% of the sample met criteria for at least one comorbid anxiety disorder.
Otto, Pollack, Fava, Uccello and Rosenbaum (1995)	Two samples of depressed patients: Sample 1: 15 outpatients with depression with no current or past anxiety disorder	Sample 1: 26 healthy controls	ASI 25.3 (12.5)	For the first sample, scores on measure of fear of emotion were significantly elevated compared with control group (15.3 (12.4)).	
	Sample 2: 144 outpatients with depression, stratified into three groups: 63 with no comorbid anxiety disorder, 53 with comorbid anxiety disorder other than panic, 19 comorbid past or current panic disorder		No anxiety 25.2 (11.9) Non-panic 28.5 (12.2) Panic 31.2 (13.0)	For the second sample, scores between the three groups were not significantly different.	
Rector, Szacun- Shimizu and Leybman (2007)	126 patients referred to an anxiety disorder treatment clinic, of whom 48 met diagnostic criteria for primary PD, 50 for SAD, and 28 for GAD. Of these, 26 were experiencing a current depressive episode.		ASI subscales	In patients with a primary anxiety disorder diagnosis, scores on the ASI Mental Concerns subscale were significantly associated with depression symptom scores, and identified anxious patients with and without secondary MDD.	Subscale scores were not reported.

Study	Population	Controls	Measures	Findings	Comments
Taylor, Koch, Woody and McLean (1996)	135 people recruited for study of whom 52 met criteria for PD without depression, 46 for depression without PD, 37 for depression and PD.		Mean (Standard Deviation) ASI PD 31.4 (9.6) Depression 22.1 (10.6) Depression and PD 40.3 (11.3)	Compared with previously published norms, each group showed elevated scores on a measure of fear of emotion	
Student samp	les				
Schmidt, Lerew and Joiner (1998)	1401 military recruits undergoing basic training		ASI 4.0 (2.9)	Overall, fear of emotion did not predict depression scores. However, score on the Mental Concerns subscale did predict depression symptom scores.	Prospective study investigating relationship between fear of emotion and depression. Scores on measure of fear of emotion were low prior to the commencement of training compared with previously published community norms.
Tull, Gratz and Lecroce (2006)	79 undergraduate students who reported at least one panic attack in the last year	undergraduate students who reported no history of panic attacks	ASI-R subscales Panic Physical harm 11.89 (8.75) Social 17.06 (8.04) Mental 6.57 (5.76) Physical sensations 15.86 (7.17)	The students reporting panic symptoms scored higher on the subscales of the fear of emotion measure than those who reported no panic symptoms. Those with panic symptoms reported higher scores on a measure of depression symptoms.	Severity of depressive symptoms was significantly correlated with fear of emotion subscale scores. The Mental Concerns subscale scores were a significant predictor of depressive symptom severity.

Study	Population	Controls	Measures	Findings	Comments
			Mean (Standard Deviation) No panic Physical harm 6.92 (7.54) Social 11.46 (6.55) Mental 2.88 (3.63) Physical sensations 9.34 (6.31)		
Viana and Rabian (2009)	94 undergraduate students who scored in the moderate to high range on a measure of worry		ASI-R 48.20 (25.84)	Participants' fear of emotions scores were elevated compared to previously published norms.	Higher scores on the fear of emotion measure predicted depression scores, after controlling for worry and GAD symptoms.

SAD = social anxiety disorder; GAD = generalized anxiety disorder; PD = panic disorder; MDD = major depressive disorder

Fear of emotion as measured with the ACS is also associated with depression symptoms cores in patients with depression, most with comorbid anxiety (Liverant, Brown, Barlow, & Roemer, 2008).

Scores on measures of fear of emotion are elevated in individuals who report depressive symptoms, but who do not have a diagnosis of major depressive disorder. Undergraduate students with high scores on a worry measure reported elevated fear of emotion scores. The fear of emotion scores predicted depression symptoms scores after controlling for worry and symptoms of generalized anxiety disorder (Viana & Rabian, 2009). In another student sample reporting a recent history of panic attacks, fear of emotions scores were significantly associated with depression symptoms scores, and the Mental Concerns subscale scores of the ASI were a significant predictor of depressive symptom severity (Tull, Gratz, & Lecroce, 2006).

Fear of emotion scores predict depression symptom severity, but the relationship is not clear. While full scales scores did not predict depression symptom severity, scores on the Mental Concerns subscale predicted depression symptom severity both in patients with depression (Cox, Enns, & Taylor, 2001), and in an apparently healthy population of military recruits (Schmidt, Lerew, & Joiner, 1998). In the latter group, this is noteworthy as the scores on the fear of emotion measure were considerably lower than community norms.

In summary, although early work on fear of emotion described explicit links to the development and maintenance of anxiety disorders, a body of work is accumulating showing a similar association with depression. Elevated scores on measures of fear of emotion are seen in patients with a diagnosis of major depressive disorder, with and without comorbid anxiety disorders. Fear of emotion scores are also associated with depression symptoms in non-clinical groups and predict the onset of depression symptoms in an otherwise healthy group. The scores on measures of fear of emotion are

typically not as high as for those with panic disorder, but are similar to social anxiety disorder, generalized anxiety disorder and specific phobia, and greater than previously published norms.

Fear of emotion as a transdiagnostic construct. A growing body of evidence demonstrates core underlying processes behind anxiety and mood disorders, if not all emotional disorders (Barlow, Allen, & Choate, 2004; Norton, 2006). The argument for common underlying factors is also strengthened when considering the high levels of comorbidity among the anxiety disorders and between anxiety disorders and depression (Norton & Philipp, 2008).

These processes have been identified in domains of attention, memory, reasoning, thought and behaviour (Harvey, Watkins, Mansell, & Shafran, 2004). Fear of emotion can be considered a reasoning process, as there is an interpretational bias where the sensations and symptoms of emotional arousal are appraised as being dangerous or harmful.

Increasingly, researchers propose that fear of emotion may be a shared component across psychological disorders (Barlow et al., 2004; Mennin, Heimberg, Turk, & Fresco, 2005). Research shows that higher baseline anxiety about the experience of negative emotion, or fear of emotion, may exacerbate responses to emotion and predict behavioural responses during laboratory emotion induction tasks (Brown, Smits, Powers, & Telch, 2003).

The relationship between fear of emotion and psychopathology is likely to be complex. While the literature reviewed shows that fear of emotion is associated with anxiety and mood disorders, it is yet to be understood if fear of emotion is a risk factor for developing an emotional disorder, is a consequence of a disorder, or perhaps both, as originally purported by Goldstein and Chambless (1978).

A range of analytical methods has been employed to investigate how potential underlying mechanisms may explain fear of emotion as a transdiagnostic process maintaining anxiety and mood psychopathology. A proxy risk factor model was used in an attempt to explain the co-occurrence of non-clinical panic attacks and panic disorder with generalized anxiety disorder (Tull, Stipelman, Salters-Pedneault, & Gratz, 2009). This risk factor (fear of emotion) was also associated with difficulties in emotion regulation. In a confirmatory factor analysis study, fear of emotion, as measured by anxiety sensitivity, was associated with anxiety and depression (Dehon, Weems, Stickle, Costa, & Berman, 2005). The authors showed that anxiety sensitivity, along with each of the ASI subscales, was significantly associated with anxiety and depression. The Physical Concerns subscale had the largest unique association with anxiety, and the Mental Concerns subscale had the largest unique association with depression. Structural equation modelling was used to investigate the relationship between fear of emotion and emotional disorders (Lewis et al., 2010). In findings similar to the above studies, the authors concluded that a 'General Distress' factor was predicted by mental incapacitation in a hierarchical manner, with lower order factors associated with a range of psychological disorders. Similarly, a hierarchical model with a general negative affectivity factor and two mid-level or mediating factors (one of which was anxiety sensitivity) was tested and showed improved specificity for depression and anxiety disorders (Paulus, Talkovsky, Heggeness, & Norton, 2015). However in that model, anxiety sensitivity was constrained to panic disorder, having explained very little variance in an alternate model linking anxiety sensitivity to social anxiety disorder.

Fear of emotion as measured by other instruments accessing the construct, namely the Agoraphobic Cognitions Questionnaire (ACQ; Chambless, Caputo, Bright, & Gallagher, 1984) and the Body Sensations Questionnaire (BSQ; Chambless et al.,

1984), is also associated with mood and anxiety disorders. Chambless and Gracely (1989) reported that while patients with panic disorder and agoraphobia reported higher levels of fearful beliefs about panic symptoms, patients with a diagnosis of either panic disorder, agoraphobia, social anxiety disorder, generalized anxiety disorder, obsessive compulsive disorder or depression reported similar levels of fears of the social or behavioural consequences of experiencing anxiety. In addition, each patient with a clinical condition scored higher than normal controls.

Prospective naturalistic studies show that in individuals with no pre-existing disorder, those with high levels of fear of emotion are more likely to develop anxiety, mood and alcohol use disorders (Schmidt, Zvolensky, & Maner, 2006). Further with regard to substance use disorders, fear of emotion may have an underlying role in the conceptualisation and treatment of intolerance to emotions in these disorders (Otto, Powers, & Fischman, 2005). Indeed, patients with substance use disorders report elevated fear of emotion (Forsyth, Parker, & Finlay, 2003; Lejuez, Paulson, Daughters, Bornovalova, & Zvolensky, 2006).

Finally, a meta-analysis reported by Olatunji and Wolitzky-Taylor (2009) showed differences in fear of emotions, as measured by various forms of the ASI, between non-clinical controls and those with anxiety and mood disorders. The authors suggested that this may be consistent with the proposition that fear of emotions is an indicator for general psychological distress, and thus a "transdiagnostic" factor.

Therefore, despite fear of emotion being conceptualised as a central factor in panic disorder, the considerable research that has ensued suggests very strongly that fear of emotion is not uniquely associated with panic and anxiety, but represents a common underlying element of mood and anxiety disorders.

Summary. The psychological construct of fear of emotion was first proposed as a cognitive maintaining component of panic disorder. Over time, fear of emotion has

been the focus of a considerable body of evidence in a bid to further understand anxiety and mood disorders. This has resulted in fear of emotion being shown to be strongly associated with all anxiety disorders and unipolar depression. Given these findings, fear of emotion can be thought of as a 'transdiagnostic' maintaining factor in psychological disorders.

Along with theoretical and cross-sectional research ascertaining the association between fear of emotion and psychopathology, the construct is seen to manifest directly in clinical settings. Many studies, as cited above, have used treatment-seeking participants meeting diagnostic criteria of various disorders, and clinical experience sees fear of emotion present in avoidance of anxiety provoking tasks and withdrawal from situations evoking high levels of emotional arousal. Clients also frequently medicate, either with prescribed anxiolytics or self-medicate with alcohol and other substances, in an attempt to reduce this arousal in the short term.

Taken together, these findings suggest that treatments which affect fear of emotion may be important for successful and effective remediation of these common psychological disorders.

Impact of treatment on fear of emotion

As the interest in understanding more about the nature of fear of emotion has grown in the last decade, so too has interest grown in the role of treatment in addressing this important individual difference. Despite being described as a trait-like characteristic, fear of emotion has been shown to decrease over treatment, as presented in the following sections.

Some research (e.g. Salters-Pedeneault, 2007) suggests that traditional treatments for anxiety and depressive disorders need to be modified or improved in order to more directly target fear of emotion. As current treatments are efficacious in

improving anxiety and mood disorders, these treatments might ameliorate fear of emotion whether or not the treatment purports to specifically target this construct.

The effectiveness of existing treatments in treating co-morbid disorders further suggests that these treatment approaches can treat the underlying transdiagnostic factors (Norton & Philipp, 2008). For example, cognitive behaviour therapy, when applied to treat a primary disorder, will also have a beneficial impact on a comorbid disorder. Such is also the case with pharmacological treatments where, for example, a selective serotonin reuptake inhibitor will treat both the mood disorder and co-morbid anxiety disorder.

To find relevant studies a literature search was carried out using electronic databases (ProQuest, PubMed, and Google Scholar). The first series of search terms were cognitive behaviour therapy, cognitive behavior therapy, cognitive therapy or CBT. These keywords could appear anywhere in the article. The second series of search terms were pharmacotherapy, medication, antidepressants, or drug therapy. For combined therapy, these two series were used with the operator 'AND'. The fourth series of search terms were anxiety sensitivity index, ASI, affective control scale, ACS, agoraphobic cognitions questionnaire, ACQ, body sensations questionnaire, or BSQ. If possible this was selected as the 'Tests and Measures' field. Each of the first three series was combined with the fourth series with the operator 'AND'. Only peer-reviewed English language articles with adult participants were selected, providing that at least pre- and post-data were reported on the measures of interest. The databases were searched for relevant studies published up until 31st January 2017.

Cognitive Behaviour Therapy. Cognitive behaviour therapy (CBT) is an efficacious treatment for depression and anxiety disorders, and has been shown to reduce symptoms in both efficacy and effectiveness studies (e.g. De Rubeis & Crits-Cristoph, 1998; Butler, Chapman, Forman, & Beck, 2006). While there is some

variation in the exact composition in this family of therapies, they typically involve components such as psychoeducation about the disorder and it's maintaining factors, strategies to modify maladaptive thoughts and beliefs; and a behavioural component to introduce patients to feared situations in anxiety, or situations from which the patient has withdrawn in depression.

CBT can be delivered in either individual or group format. Each format offers the potential to exploit particular advantages. Individual CBT is able to provide a context for exploring a client's particularly distressing personal experience and allows flexibility of delivery of treatment protocols, whereas group treatment offers opportunities for shared experiences, vicarious learning and support (Tucker & Oei, 2006). The diminishing availability of resources, particularly in publicly funded healthcare settings, suggests that the more efficient group therapy options will be preferred, although the actual cost-effectiveness may be small (Tucker & Oei, 2006).

In a review of the efficacy and cost-effectiveness of group and individual therapy, Tucker and Oei (2006) identified that the benefits of one format of treatment over the other may differ depending on the psychological disorder being treated.

For patients diagnosed with panic disorder, no differences were found between individual, group or brief individual CBT, with each version of treatment proving effective compared with a wait list control (Marchand, Roberge, Primiano, & Germain, 2009). Sharp, Power and Swanson (2004) found that individual treatment for panic disorder was more effective than group therapy post-treatment, but this difference diminished by three month follow-up when there was no difference in outcomes.

For patients with generalized anxiety disorder, there have been very few published studies for group CBT, and none of these directly compared the effectiveness of the two approaches. In a recent study, group CBT for generalized anxiety disorder

demonstrated large effect sizes that were numerically comparable with those found in individual CBT (McEvoy et al., 2015).

In the treatment of health anxiety, CBT has been shown to be an effective therapy (e.g. Asmundson, Abramowitz, Richter & Whedon, 2010; Olatunji et al. 2014). Most studies have used an individual approach but three studies (Bouman, 2002; Stern & Fernandez, 1991; Hedman et al., 2010) have shown that group therapy is also effective for severe health anxiety.

In the treatment of depression with CBT, the seminal work by Beck, Rush, Shaw and Emery (1979) described protocols for treating depressed patients in either individual or group format. A very large number of treatment studies (in excess of 75) have shown CBT to be an effective intervention for treating depression (Butler, Chapman, Forman, & Beck, 2006). When delivered in group format, CBT remains an effective treatment with outcomes at three months post-treatment being no different than for those patients receiving individual treatment, although at post treatment, there is an advantage for those patients receiving individual therapy (Huntley, Araya, & Salisbury, 2012).

For the treatment of social anxiety disorder, a recent meta-analysis (Mayo-Wilson et al., 2014) concluded that individual CBT produced the largest effect sizes when comparing a range of psychological and pharmacological interventions. While group CBT was effective, the effect sizes were significantly less than those for individual therapy.

Individual CBT has been shown to be an efficacious treatment for posttraumatic stress disorder, and is considered the treatment of choice in expert consensus guidelines (NICE, 2005). There are some preliminary findings indicating that group CBT is also effective compared with a minimum contact control group (Beck, Coffey, Foy, Keane, & Blanchard, 2009). There have been no direct comparisons between individual and

group treatment approaches (Phoenix Australia – Centre for Posttraumatic Mental Health, 2013), but a recent meta-analysis indicated that the effect sizes for group CBT were much less than those for individual treatment (Watts et al., 2013).

For patients with obsessive compulsive disorder, a number of studies have attested to the effectiveness of both individual and group treatment approaches (Fals-Stewart, Marks, & Schafer, 1993; Anderson & Rees, 2007; Jaurrieta et al., 2008; Jonsson, Hougaard, & Bennedsen, 2011). A meta-analysis of these four comparative studies suggests there was a small advantage to individual therapy (Jonsson et al., 2011).

Overall, CBT delivered in either individual or group format is effective in the treatment of depression and anxiety disorders. There is some evidence that an individual approach may be advantageous for social anxiety disorder, posttraumatic stress disorder and obsessive compulsive disorder. There is little difference in outcomes between the two formats for patients with panic disorder, generalized anxiety disorder or depression.

CBT and fear of emotion. As described in the previous section, fear of emotion can be considered as a cognitive construct that is reinforced by cognitive, behavioural and emotional avoidance. Given the nature of CBT treatment, it is likely that CBT will address both the cognitive and behavioural elements of the construct, either explicitly or implicitly by assisting the patient to develop some confidence in managing the experience of distressing emotions (such as anxiety and depression), leading to a reduction in their fear of emotions. Therefore it is expected that CBT for anxiety and depressive disorders will result in a reduction in scores of fear of emotion.

Change in fear of emotion with CBT for anxiety and depression. In an early review article, Chambless and Gillis (1993) investigated effect sizes for change measures in the treatment of anxiety disorders and depression. They reported that in the CBT treatment of panic disorder and agoraphobia, large effect sizes were seen for

anxiety symptoms and for fear of fear symptoms measured by a range of psychometric measures (e.g. ACQ, BSIQ).

Two meta-analyses have been published describing the impact of CBT on fear of emotion as measured by the ASI. In the first (Otto & Reilly-Harrington, 1999), the impact of treatment on fear of emotion was investigated along with the need to target the treatment specifically at the anxiety sensations. Ten treatment studies were included in the report. Of these, four studies randomly allocated patients with a diagnosis of panic disorder to either CBT or control; one study comprised individuals with elevated anxiety sensitivity (who, therefore, were considered 'at risk' of developing panic disorder), and the remaining five studies were open trials of CBT, pharmacotherapy or their combination for panic disorder or major depressive disorder. Across these 10 studies, levels of fear of emotion as measured by ASI scores decreased significantly over treatment with CBT. The reduction in ASI scores in the seven studies with a CBT treatment component (totalling 160 participants) was approximately 14 points from pretreatment to post-treatment. The treatment varied considerably from 10 to 12 weeks of group CBT to self-help bibliotherapy approaches. The greater reductions were seen over the longer treatments, with a five-week CBT program (Gould, Clum, & Shapiro, 1993) producing much smaller changes (approximately two points on the ASI) compared with longer treatment programs showing 12 to 20 point reductions in ASI scores (Telch et al., 1993; Shear, Pilkonis, Cloitre, & Leon, 1994; Hazen, Walker, & Eldridge, 1996; McNally & Lorenz 1987; Penava, Otto, Maki, & Pollack, 1998).

In the second meta-analysis, Smits et al. (2008) updated the analysis previously reported by Otto and Reilly-Harrington (1999), and investigated the role of variables which may potentially mediate the relationship between treatment and reduction in fear of emotion as measured by the ASI. Given the number of studies that had been completed in the years between the meta-analyses, Smits et al. (2008) stratified the

studies into those with patients at risk (adults with elevated anxiety sensitivity) and those who were seeking treatment for a particular disorder when comparing the efficacy of CBT against control conditions. The secondary analyses investigated the amount of therapist contact, the type of control condition (waitlist or psychological control condition) and year of publication. The meta-analysis was conducted on 24 studies that met the inclusion criteria of participants aged over 18, random assignment to CBT or a control condition, and measurement of anxiety sensitivity with one of the versions of the ASI. Exclusion criteria were single case studies and the use of combined CBT and pharmacotherapy.

Of the 24 studies included, 16 studies involved patients seeking treatment for conditions of panic disorder, tinnitus, claustrophobia and social anxiety disorder. There was some variability in the CBT used in the studies with the predominant approach including cognitive therapy with some exposure component (10 of the 16), other approaches being either a cognitive or exposure component used alone. The remaining eight studies included participants who met criteria for being 'at risk' (that is, having elevated scores on the ASI), which varied across studies from being in excess of 16 to in excess of 25 on the ASI-16 or in excess of 28 in the study that used the ASI-R.

An effect size of Hedges' g of 1.40 was found for the treatment seeking studies, representing a large effect for CBT over control conditions. The weighted pre-treatment score was 31.18 (SD=10.29) and the post–treatment score was 20.28 (SD=9.89), representing a change score on the ASI of 10.89 (SD= 3.14). For the control condition, the pre-treatment score was 31.89 (SD=9.71), and the post-treatment score was 30.74 (SD=10.51), showing a change score of 1.15 (SD=3.15). In other analyses reported from the same study, there was no evidence of publication bias, and larger reductions in anxiety sensitivity were seen with more hours of therapist contact, particularly in the CBT treatment conditions. One suggestion from this study was that further studies are

required to identify the mechanisms underlying change from different treatment modalities (such as CBT and pharmacotherapy).

While no further meta-analyses have been published, more recent studies have continued to show reductions in fear of emotion scores in patients treated with CBT for anxiety disorders or depression. In a study that met the inclusion criteria for the Smits et al. (2008) meta-analysis, patients diagnosed with panic disorder and treated with CBT showed a large decrease in ASI scores, from a mean of 29.90 (SD=8.94) to 15.03 (SD=7.58) (Liebscher et al., 2016). Other studies have also shown large decreases in ASI scores in patients undergoing CBT treatment with a primary diagnosis of an anxiety disorder. Mean ASI scores decreased from 36.75 to 26.79 in patients undergoing manualised group CBT (Heatherington, Harrington, Harrington, Niemeyer, Weinberg, & Friedlander, 2014), from 31.54 (SD=9.46) to 21.51 (SD=7.94) in patients undergoing a unified treatment protocol (Schmidt, Buckner, Pusser, Woolaway-Bickel, Preston, & Norr, 2012) and from 31.81 to 19.47 in patients undergoing a transdiagnostic CBT treatment (Talkovsky & Norton, 2014). In two of these studies (Heatherington et al., 2014; Schmidt et al., 2012), most patients were medicated (74% and 57% respectively) and in the third study (Talkovsky & Norton, 2014) medication status was not reported.

CBT for fear of emotion. As described above, much of the research investigating the impact of CBT on fear of emotion has come about as an incidental finding of treatments designed to ameliorate specific psychological disorders, most commonly panic disorder.

In a study to investigate possible mechanisms by which these improvements occur, a mediation analysis was performed on data collected from earlier studies (Smits, Powers, Cho, & Telch, 2004). The results from these analyses showed that fear of emotion (termed 'fear of fear') significantly mediated the effects of CBT on several outcome measures of panic disorder, including anxiety symptoms, frequency of panic

attacks, agoraphobic avoidance, and global disability. The authors noted that the role of pharmacotherapy in addressing fear of emotion also needed to be investigated.

Some studies have specifically investigated the impact of CBT on fear of emotion. Schmidt et al. (2007) described a study aimed at developing a program to reduce fear of emotion, and monitored the outcomes over a 24 month period to determine if there was an effect on the development of anxiety and mood problems. Four hundred and four participants scoring at a moderately elevated level on the ASI (and a community sample screened to exclude individuals with a current psychiatric illness) were randomly assigned to either the anxiety sensitivity reduction program or to a control condition. The treatment condition involved participants viewing a video with educational information presented about the nature of anxiety and the benign nature of symptoms and physical signs of emotion. The presentation also explained exposure exercises to reduce the interoceptive conditioning. Following the presentation, an experimenter reviewed the video material and provided further information about the exposure exercises, but the amount of practice was not monitored. The participants in the control condition received a presentation on health and nutrition to equate time spent with the experimenters. The findings of this study indicated that targeting fear of emotion reduced the incidence of a diagnosed anxiety and mood disorder. The intervention led to a large change in fear of emotion with ASI scores changing from a mean of 17.0 (SD=8.2) at pre-treatment to 11.9 (SD=7.2) at the completion of the treatment.

A series of studies reported on the relationship between fear of emotion, as operationalised by anxiety sensitivity, and pain sensitivity (Watt, Stewart, Lefaivre, & Uman, 2006). As part of these studies, Watt et al. used a brief CBT program with the intention of reducing fear of emotion. This program consisted of three 50-minute group sessions of six to 10 participants. The material presented in the sessions involved

psychoeducation about anxiety, fear of emotions, interpretation of emotion arousal sensations, identifying and reframing dysfunctional thinking, and a brief interoceptive exposure exercise (running). Across treatment, participants with elevated anxiety sensitivity levels were seen to reduce their fear of emotion scores significantly (Watt, Stewart, Conrod, & Schmidt, 2008). This research group also used the same brief CBT intervention to significantly reduce fear of emotion in participants when investigating the relationship between fear of emotion and beliefs and behaviours associated with problematic alcohol use (Watt, Stewart, Birch, & Bernier, 2006).

In measuring the impact of CBT treatment on fear of emotion, the vast majority of studies have used the ASI as the measure of fear of emotion. In one of the few published studies using the ACS, Roemer & Orsillo (2007) reported on the treatment of 16 individuals with a diagnosis of generalized anxiety disorder using a CBT treatment augmented with therapy components based on the principles of Acceptance and Commitment Therapy. Following treatment, the authors noted a significant difference between pre- and post-treatment for the ACS total score (3.97, SD=.62 to 2.94, SD=.77), with large effect sizes (partial eta squared =.68). Of the 16 clients, six were taking psychotropic medication throughout the treatment program (anxiolytic, antidepressant, or both). At three-month follow-up, there was a significant difference between pre-treatment and follow-up scores, maintaining the large effect size.

Summary. Cognitive behaviour therapy is an effective treatment for mood and anxiety disorders, which can be delivered in individual or group format. This type of psychological therapy is likely to be effective in reducing patients' fear of emotion as it involves cognitive and behavioural strategies which target the construct directly (e.g., interoceptive exposure) or indirectly (e.g., emotion monitoring, exposure to emotion inducing situations). Treating patients with anxiety and depressive disorders with CBT

results in lower scores on measures of fear of emotion. CBT has also been used to target fear of emotion directly with large effect sizes.

Pharmacotherapy. Pharmacotherapy is an evidence-based intervention for the treatment of anxiety and mood disorders (Roy-Byrne & Cowley, 2007; Nemeroff & Schatzberg, 2007). Commonly used classes of medications used in treating these disorders are anxiolytics (such as benzodiazepines) and antidepressants (such as selective serotonin reuptake inhibitors (SSRIs)). While these classes of drugs generate improvements in outcome measures, such as symptom severity, they may also have an effect on fear of emotion.

Pharmacotherapy and fear of emotion. Benzodiazepines exert their action by potentiating the effect of gamma-aminobutyric acid (GABA) in the central nervous system (CNS). Specifically, benzodiazepines bind to the GABA receptors at different subunits, thus accounting for their slightly differing effects depending on their chemical structure. GABA is a major inhibitory neurotransmitter in the CNS. Benzodiazepines exert their effect on GABA at various structures within the CNS including spinal cord, hypothalamus, hippocampus, substantia nigra, cerebellar cortex, and cerebral cortex. These changes at CNS level manifest as sedation, reduction in the physical symptoms associated with anxiety, a depressant effect on psychomotor and cognitive functions, induction of sleep, along with anticonvulsant effects, muscle relaxation, and respiratory depression (Trevor & Way, 2009). Given that fear of emotion is in response to concerns about the ability control emotion, medication that reduces physiological arousal and cognitive hypervigilance could be seen as helpful in overcoming such a fear as the emotion can now be considered manageable by the user of the medication. While benzodiazepines have been used in the management of anxiety and depression since their discovery in the 1960s, they are not currently recommended for first line treatment (Rossi, 2014). Despite this, long term use remains common (National Prescribing Service, 1999).

The precise mechanisms by which SSRI medications reduce anxiety and depression are not precisely understood. Despite being different chemical moieties, this class of medications all have the same property of increasing the amount of serotonin (5-HT) in synaptic clefts in the central nervous system, leading to increased serotonergic neurotransmission. Three pathways have been described that lead to a reduction in anxiety (Gorman, Kent, Sullivan, & Coplan, 2000). First, the activation of the serotonergic pathway from the brainstem raphe region to the locus ceruleus results in inhibition of noradrenaline activity. It is proposed that this reduction of activity leads to a reduction in adrenergic symptoms such as increased heart rate and blood pressure. Secondly, the pathway from the raphe nuclei to the periaqueductal grey area appears to have a role in reducing the tendency to escape from anxiety provoking situations. It has been shown that increasing serotonin in the dorsal raphe nucleus leads to an increase in serotonin in the periaqueductal grey area with an attendant reduction in activity in this region. Serotonergic agonism in the dorsal periaqueductal grey area elicits an anxiolytic effect with a reduction in heart rate and blood pressure. Serotonin agonism in the ventrolateral periaqueductal grey area leads to a heightened fear response, suggesting specificity for serotonin receptors (Yamashita, de Bortoli, & Zangrossi, 2011). The third pathway involves a reduction in the release of corticotropin releasing factor (CRF) from the hypothalamus following prolonged treatment with a serotonergic agent. CRF is ultimately responsible for the release of cortisol from the adrenal glands and is shown to be elevated in subjects with anxiety and depression. CRF also has a role as a neurotransmitter in the CNS and, in animal models, elevated CRF levels are seen to be associated with increased levels of fear with physiological and behavioural

consequences. Conversely agents which are CRF antagonists have been shown to reduce these effects (Gorman et al., 2000).

Finally, serotonergic agents such as SSRIs have been shown to have a direct effect on the central nucleus of the amygdala. Increased levels of serotonin have an inhibitory effect on sensory inputs from thalamic and cortical pathways. Thus the anxiolytic effect of SSRI medications is likely due to a primary effect of reducing the sensitivity of the amygdala to excitatory inputs, and a reduction in physiological symptoms triggered by cortisol and noradrenaline. A secondary indirect effect is also likely as individuals realise that anticipated catastrophic physical or mental outcomes do not occur, with a consequent reduction in anticipatory anxiety and avoidance.

Change in fear of emotion with pharmacotherapy for anxiety and depression. Few studies have reported on changes in fear of emotion measures when patients have been treated with pharmacotherapy. The following five studies investigated the impact of various medications in patients with anxiety disorders and depression. In these studies, fear of emotion (as measured by the ASI) was a secondary outcome measure.

Three studies have investigated changes in fear of emotion during pharmacological treatment of patients with panic disorder. Otto, Pollack, Sachs and Rosenbaum (1991, cited in Otto & Reilly-Harrington, 1999) reported on the treatment of 24 individuals with panic disorder treated with naturalistic pharmacotherapy. The drug treatment consisted of benzodiazepines (67%), antidepressants (8%) or a combination (25%). After six months of treatment, fear of emotion scores, as measured by ASI, decreased from a mean of 31.2 to 22.2. A similar magnitude of reduction in fear of emotion scores was reported for 26 patients with panic disorder with agoraphobia taking either SSRIs or SNRIs with scores decreasing from 31.18 at pre-treatment to 25.21 after a mean 53 days treatment (Liebscher et al., 2016). In the third study, larger reductions in fear of emotion were reported for patients with panic disorder and

agoraphobia being treated solely with an antidepressant drug (Mavissakalian, Perel, Talbott-Green, & Sloan, 1998). In a study of 110 patients, fear of emotion scores decreased from 33.6 to 12.5 after 24 weeks of treatment for the 59 treatment completers. In this study, the pharmacotherapy used was imipramine, a strongly serotonergic tricyclic antidepressant with demonstrated efficacy for treating the disorder. This change occurred early in therapy with an ASI score of 20 being achieved after eight weeks of treatment.

Olatunji et al. (2008) reported on an investigation into changes in fear of emotion in generalized anxiety disorder during treatment with pharmacotherapy. They examined the effect of pharmacotherapy, specifically fluoxetine, an SSRI, on fear of emotion in 38 patients diagnosed with generalized anxiety disorder. The aim of the study was to investigate changes in symptoms of anxiety and depression during the treatment of generalized anxiety disorder for which SSRIs such as fluoxetine had previously been shown to be effective. The authors also identified that the role of fear of emotion, despite being associated with generalized anxiety disorder, had not been examined across treatment with pharmacotherapy. The results showed a small reduction in mean (SD) scores of fear of emotion with ASI at pre-treatment of 24.97 (9.80) reducing to 19.23 (10.05) at post-treatment. Scores were also reported for the ASI subscales, showing a reduction in social concerns and mental concerns. The scores for physical concerns did not decrease significantly, which was somewhat surprising as models suggest that reduction of anxiety symptoms would lead to a reduction of fear of that emotion. This may suggest that in the maintenance of anxiety disorders, the fear of emotion persists regardless of the presence or absence of physical symptoms.

In patients being treated with pharmacotherapy for depression, fear of emotion scores were assessed by Otto et al. (1995). Eighty-six patients meeting diagnostic criteria for depression were part of an eight week open trial of fluoxetine. At pre-

treatment there was no difference in fear of emotion scores between patients with a comorbid anxiety disorder and those only with depression. Following treatment, fear of emotion scores decreased significantly from a mean of 27.1 (SD=12.3) to a mean of 20.1 (12.1). This change also correlated with changes in depression severity rating and dysfunctional thinking.

Pharmacotherapy for fear of emotion in panic disorder. Two studies were specifically designed to investigate changes in fear of emotion in patients with panic disorder being treated with pharmacotherapy.

In a small study (Romano, van Beek, Cucchi, & Perna, 2004), 20 patients with a diagnosis of panic disorder, but no other psychiatric comorbidities, were treated for six weeks with citalopram (an SSRI). Each patient received the same dose of citalopram and they did not receive any other treatments (psychiatric or psychological) during the trial. The authors reported a change in ASI scores from a mean (SD) of 26.6 (8.7) at pretreatment, to a mean (SD) of 17.2 (7.7) at post-treatment. They concluded that pharmacologically modifying the serotonergic system inhibited physiological and cognitive symptoms of anxiety. It should be noted that only 10 of the 20 patients recorded pre-treatment scores that could be considered in the 'high' range (greater than or equal to 27), which is somewhat surprising in a group of patients meeting criteria for panic disorder. Also, the decrease in fear of emotion scores did not predict a decrease in measures of panic severity, which is in stark contrast to the considerable literature showing a high association between fear of emotion and the symptoms of panic disorder. The authors suggested that a reduction in symptoms of anxiety is separate from the fear associated with experiencing the symptoms.

A combination of pharmacotherapeutic agents was investigated in a study reported by Simon et al. (2004), who re-analysed data from an earlier study (Pollack et al., 2003). Fifty-nine participants with a primary diagnosis of panic disorder with or

without agoraphobia were randomly allocated to three treatment groups; one receiving paroxetine (an SSRI) alone, one with paroxetine and continuous clonazepam (a benzodiazepine) treatment, or one with paroxetine and tapering benzodiazepine treatment. Across the twelve weeks of therapy, there was no significant difference between the three treatment conditions with regard to changes in ASI scores. Across all conditions, ASI scores changed from baseline mean (SD) 32.8 (12.9) to a mean of 23.2 (12.0) at the end of treatment. For treatment completers (n=33), scores decreased from 30.7 (12.2.) to 18.4 (9.1). There were differences, however, in the pattern of change between the group who continued benzodiazepine treatment, and the group who ceased gradually. While not reaching statistical significance, there was a large effect size (d=.77) in favour of the taper group. This change did reach significance when controlling for pre-treatment differences in panic severity.

In investigating the impact of pharmacotherapy on fear of emotion as measured by the ACS, no published literature was found.

Pharmacotherapy interfering with treatment for fear of emotion. Despite the above investigations demonstrating the usefulness of pharmacotherapy in reducing fear of emotion, specifically as measured by ASI, there are some suggestions that medications may interfere with the process of reducing this fear. In a letter to the American Journal of Psychiatry, Fava (1996), citing his group's own research (Fava et al., 1994) into benzodiazepine withdrawal, asserted that continued use of benzodiazepines compromised the ability of patients to learn to tolerate uncomfortable symptoms of emotion, thus leading to maintenance of the underlying disorder.

In a comprehensive review examining the benefits each of cognitive behavioural therapy and pharmacotherapy in the treatment of anxiety, Westra and Stewart (1998) described three key maintaining factors of anxiety disorders (arousal, behavioural avoidance, and catastrophic cognitions) and concluded that pharmacotherapy essentially

focussed on the somatic arousal component, whereas CBT focused explicitly on the avoidance and cognitive components. While it seems logical that combining the two therapies may prove advantageous, their review of published data suggested that pharmacotherapy could interfere with the cognitive variables, such as catastrophic beliefs, self-efficacy, hypervigilance, and learning and memory.

With regard to catastrophic beliefs, Westra and Stewart (1998) proposed that anxiolytic medication (i.e. benzodiazepines) interfered with changing maladaptive beliefs about the symptoms of emotion by preventing adaptive learning experiences. That is, by using anxiolytic medication, patients were deprived of the opportunity to learn that feared outcomes may not arise. Secondly, the use of anxiolytic medication leads to the elimination of these feared sensations, as opposed to a more adaptive approach of learning to tolerate the distress associated with the emotion. The proposed mechanism was also suggested to be a function of the potency of the benzodiazepine medication (high potency being more problematic than lower potency) and the increasing use of 'prn' or 'as required' dosing regimens.

A second cognitive variable that might be adversely affected by the use of pharmacotherapy is that of self-efficacy for managing symptoms of emotion. Westra and Stewart (1998) proposed that the use of anxiolytic medication would serve to undermine the development of a patient's self-efficacy for managing their distressing emotions. Once again they asserted that this was a function of the potency and dosing regimen of the benzodiazepine medication.

With regard to hypervigilance, Westra and Stewart (1998) derived different conclusions based on the nature of the medication. They suggested that anxiolytic benzodiazepine medication affected the patient's capacity to fully attend to the predicted feared outcome of the emotional experience, thus interfering with adaptive learning. Secondly, particularly when 'prn' dosing is utilised, a patient is inadvertently

trained to increase their awareness of symptoms of emotion in order to identify the appropriate time for dosing. These issues were not expected to arise with antidepressant medication which does not have such a direct effect on anxious arousal, nor is dosing linked to identification of somatic symptoms. This hypothesis was supported in a study by Stewart, Westra, Thompson, and Conrad (2000) who investigated the use of benzodiazepines on attention to threat cues using a Stroop task paradigm in a group of patients with a range of anxiety disorders. In this study the group using benzodiazepine medication was more attentive to the emotional threat words, particularly those characterizing physical threat, than matched patients not taking benzodiazepines. This effect was particularly pronounced in the sub-group of benzodiazepine users who took their medication on a 'prn' basis. Given the quasi-experimental design of the study, the correlational analyses need to be interpreted carefully.

Finally, Westra and Stewart (1998) summarised a body of literature suggesting multiple pathways through which benzodiazepine medication may interfere with learning. Of significance is the phenomenon of state-dependent learning, where a patient may learn to tolerate anxiety in the context of dosing with medication, but cannot generalise this tolerance to a medication-free state.

In considering fear of emotion specifically, Westra, Stewart and Conrad (2002) investigated patients with panic disorder with agoraphobia being treated with regular versus 'prn' benzodiazepines. They found that greater 'prn' use of benzodiazepine medication at pre-treatment was associated with less reduction in anxiety sensitivity. The 'as needed' use of medication was a better predictor of anxiety sensitivity than pre-treatment anxiety sensitivity scores. The naturalistic design of this study limits the interpretation of these findings, and the authors noted the need for a randomised controlled study to validate their conclusions.

In a study investigating augmentation of CBT with d-cycloserine for the treatment of panic disorder, benzodiazepine use was associated with poorer outcomes from CBT treatment regardless of augmentation but antidepressant use was not (Otto et al., 2016). In an analysis of recurrence of anxiety in the landmark Coordinated Anxiety Learning and Management (CALM) trial, benzodiazepine use was found to be the most discriminant predictor of recurrence of anxiety symptoms in patients receiving a combination of pharmacotherapy and cognitive behaviour therapy (Taylor, Jakubovski, & Bloch, 2015). This study also showed that high scores on fear of emotion six months into treatment was the second strongest predictor of anxiety recurrence. As the use of benzodiazepines was a second-line pharmacotherapy, it is possible that the association was an indicator of treatment resistance.

Summary. Various medications are useful in treating depression and anxiety disorders. These medications are categorised into different classes and have distinct chemical moieties. When used primarily for the treatment of depression and anxiety, fear of emotion scores decrease to previously reported community norms. In two small studies, fear of emotion scores decreased in patients being treated with an SSRI alone or in combination with a benzodiazepine. However, benzodiazepines may also have a deleterious effect on reducing fear of emotion by impacting on beliefs, self-efficacy, hypervigilance, and learning and memory.

Combined therapy. Given the efficacy of CBT and pharmacotherapy for treating anxiety and mood disorders, it is not surprising that clinicians would utilise some combination of these therapies with the aim of achieving better outcomes. Expert consensus guidelines have been published which recommend the use of a single therapeutic approach, at least as a first step, in managing these disorders. Despite the publication of such guidelines advocating monotherapy as first choice treatment, clinical practice is increasingly making use of the concurrent or sequential use of

pharmacotherapy and psychotherapy (Otto, Smits, & Reese, 2005). The two types of treatment can be used together in an integrated format (two types of treatment provided by one clinician, typically a psychiatrist) or in a combined format (two types of treatment provided by two clinicians). A number of studies suggest benefits to the use of combined therapy. It may increase the rate of response in the acute phase, increase the rate of remission, minimise relapse, enhance the acceptability of each treatment, and provide a broader response as each treatment retains its specific benefits (Fava & Ruini, 2005; Otto et al., 2005; Petersen, 2006).

As shown in the preceding sections, CBT and pharmacotherapy also reduce fear of emotion in patients with anxiety and depression, whether the treatment is targeted at the symptoms of the psychological disorder, or at fear of emotion itself. Therefore, it is possible that combining these approaches will also reduce fear of emotion in individuals with anxiety or mood disorders. Indeed, it is possible that the two treatments may have a synergistic effect on decreasing negative affect. However, should pharmacotherapy, particularly benzodiazepines, interfere with the learning opportunities available in CBT, then combined therapy may result in poorer outcomes. Studies attempting to unravel these hypotheses have been carried out, some comparing combined therapy to CBT alone.

Combined therapy and pharmacotherapy. In one of the early effectiveness studies for the treatment of panic, Roy-Byrne et al (2005) reported on a randomised control trial investigating the role of combined therapy for the treatment of panic disorder. They used a measure of fear of emotion (ASI) as one of their numerous outcome data. This large study (N=232) compared a treatment-as-usual condition, primarily pharmacotherapy, to a combined pharmacotherapy/CBT regimen specifically designed for use in the primary care setting. The pharmacotherapy component was an algorithm prepared by a psychiatrist and involved the use of an SSRI for at least six

weeks, unless previous attempts with that class of medication had been unsuccessful, in which case other classes of antidepressant medication were used with adjunctive medications, such as benzodiazepines, if necessary. The CBT program was modified from a format previously shown to be efficacious and involved six sessions in the first 3 months, of which at least three were face-to-face contact. The remainder of the sessions could be delivered by telephone. The therapists used in the study were considered to be novices in the sense that they had basic masters or doctoral level training and minimal, if any, experience in delivering CBT interventions. In the combined therapy group, 68.1% of participants had between four and six sessions of CBT; both groups received similar anti-panic pharmacotherapy. Scores in fear of emotion for the group treated with pharmacotherapy alone changed from a mean of 32.3 at pre-treatment to a mean of 28.94 at 3 months, and 27.73 at 6 months. For the group treated with combined therapy, mean pre-treatment scores of 34.4 decreased to 23.11 at 3 months and 21.63 at 6 months. While the authors noted that the objective of the study was to test the effectiveness of treatments for panic disorder, they noted that many of the participants had a comorbid disorder (for example, over 70% of clients also met criteria for another anxiety disorder or for major depressive disorder), and thus the broadening of the CBT component to address aspects of other disorders may be useful.

Craske et al (2005) reported on a subset of the data taken from the above study investigating specifically changes in fear of emotion. This study compared patients with a primary diagnosis of panic disorder (but with comorbid psychological disorders) being treated with pharmacotherapy, or pharmacotherapy with the addition of CBT. The primary outcome measure was ASI. The authors reported that adding CBT, or indeed at least one component of CBT, to pre-existing medication regimens led to improvement in fear of emotion, which continued to 1-year follow-up. The definition of CBT for this paper was broad with components of CBT being defined as relaxation, situational

exposure, homework, interoceptive exposure, psychoeducation, identification of cognitive biases, and restructuring of cognitive distortions.

Combined therapy and CBT. The impact of combined therapy on fear of emotion was reported in a seminal paper investigating the efficacy of an individual CBT treatment applied in a group program (Telch et al., 1993) for patients with a primary diagnosis of panic disorder. The main finding was that delivering the CBT treatment in a group format was effective. Among the battery of self-report measures used, fear of emotion was assessed with the ASI. The medication being taken was described as 41.2% benzodiazepines only, 5.9% antidepressants only, 5.9% benzodiazepines and antidepressants, and 2.9% taking other unspecified anxiolytic medication. Given that a large percentage of the patients were medicated (55.9%), the authors also commented on a post-hoc analysis of the effect of medication in the group receiving the CBT treatment. The authors found no differences between the medicated and un-medicated groups on any of the clinical measures at pre-treatment, post-treatment, or at follow-up.

A similar conclusion was drawn from a study into the treatment of 37 patients with panic disorder in a group CBT program administered through an anxiety disorders clinic at a general hospital (Penava, Otto, Maki, & Pollack, 1998). Each of the patients participated in a structured 12 week CBT program, and 27 of the patients were taking medication. Across the course of treatment, there was a significant reduction in fear of emotion (ASI) from a mean (SD) of 35.0 (12.5) to 21.5 (12.9). Data were not provided to differentiate medication users from non-users, and the authors concluded that use of medication was not detrimental to the improvements in the severity scores of ASI scores of patients. In this study, patients were taking benzodiazepines (16), SSRIs (14), tricyclic antidepressants (7) and a monoamine oxidase inhibitor (1).

In a study that did provide differential data for medication users and non-users (Westra et al., 2002), 43 patients presenting for treatment with panic disorder with

agoraphobia received a 10-session CBT program for anxiety disorders presented in group format. Thirty-three patients were medicated and 10 were not taking medication. Eleven of the benzodiazepine users were also taking antidepressant medication and one a beta-blocker. Patients were stratified into three groups; those not taking benzodiazepines, those taking benzodiazepines regularly, and those taking benzodiazepines on a 'prn' (as required) basis. The authors found that fear of emotion decreased over treatment from a mean (SD) of 40.20 (6.33) to 18.64 (5.87) for the unmedicated patients, from 43.77 (8.63) to 19.97 (12.98) for the regular benzodiazepine users, and from 44.60 (10.11) to 34.18 (9.58) for the 'prn' benzodiazepine users. It was concluded that the non-medicated patients were not significantly different from the regular benzodiazepine users; however, the 'prn' benzodiazepine users remained significantly higher on the fear of emotion measure (ASI). The authors also reported on clinical significance and found a similar pattern of results with the 'prn' benzodiazepine users scoring higher on levels of fear of emotion.

Arch and Craske (2007) reported on an analysis of data from two treatment studies of a manualised CBT treatment for panic disorder, with the majority of patients reporting using pharmacotherapy with naturalistic dosing regimens. Their study addressed several questions; the most pertinent to the current research looked at whether use of medication at commencement of treatment with CBT was associated with different treatment outcomes compared with those not using medication at the commencement of CBT in a sample of treatment seeking individuals diagnosed with panic disorder, with or without agoraphobia. In examining the impact of medication use, the researchers divided medication use into three classes; those taking SSRIs only, those taking benzodiazepines only, and those taking any antidepressant or anti-anxiety medication. In relation to changes in fear of emotion (measured by ASI), the researchers found that there were no differences for patients taking benzodiazepines across CBT

treatment at post–treatment or at six-month follow-up. For those taking SSRIs, there was little difference between ASI scores for both groups at pre- and post-treatment, but at 6-month follow-up, those taking SSRIs scored 4.5 points higher than those who did not use SSRIs. The authors also noted that for 'other' antidepressants, there was a similar pattern of results, although this did not reach statistical significance. Thus, the use of pre-treatment SSRI or other antidepressant medication resulted in significantly higher levels of fear of emotion at 6-month follow-up, compared with those not receiving the medication.

In investigating the impact of combined therapy on fear of emotion as measured by the ACS, no published literature could be found.

Summary. Combining efficacious therapies is becoming commonplace in clinical practice, despite expert guidelines promoting monotherapy as first line treatment for anxiety disorders and depression. As the monotherapies are effective in reducing fear of emotion, it is likely that combined therapy also reduces fear of emotion. In studies comparing combined therapy to pharmacotherapy alone, combined therapy achieved greater reductions in fear of emotion than pharmacotherapy alone. When comparing combined therapy to CBT alone, there appears to be little difference between treatment types, although patients treated with SSRIs reported higher scores of fear of emotion at six-month follow-up.

Summary of impact of treatment on fear of emotion. Fear of emotion is a feature of emotional disorders that is amenable to treatment effects when addressed indirectly or directly. Treatments that have been shown to be efficacious and effective for treating anxiety and mood disorders have also been shown to reduce fear of emotion. Two evidence-based treatments are cognitive behavioural therapy and pharmacotherapy. When applied to the treatment of anxiety disorders or depression, fear of emotion is significantly reduced when either treatment is utilised. Therefore, when

individuals with an anxiety disorder or depression are treated using a CBT approach, scores on fear of emotion consistently decrease regardless of whether that is an explicit focus of therapy. That is, fear of emotion decreases over treatment with CBT.

Given the widespread use of combination therapy in clinical practice, research has investigated the impact of combined therapy on fear of emotion. While fear of emotion has not been addressed specifically, the research summarised above suggests that patients receiving combinations of CBT and pharmacotherapy also experience a reduction in fear of emotion.

However, there have been suggestions that combining pharmacotherapy with CBT may reduce the effectiveness of the CBT treatment, and some preliminary studies have shown that patients treated with combined therapy have less favourable outcomes than those treated with CBT alone.

Rationale for Study 1

Fear of emotion is a construct that describes an individual's predisposition to notice sensations associated with the experience of emotion and to react fearfully to these sensations. The current study aimed to replicate and extend previous research by exploring the relationship between treatment gains and changes in fear of emotion in the treatment of anxiety disorders and depression in a naturalistic setting.

Fear of emotion, as measured by the ASI, is associated with anxiety disorders and depression. The parallel development of the ACS suggests that this measure assesses the same construct. Much of the research using the ACS as a measure of fear of emotion has relied on 'analogue' samples; that is participants, usually university students, have been selected based on their score on a screening measure. The current study used data collected from patients attending a community mental health centre who had been diagnosed with a psychological disorder. In order to validate the fear of

emotion construct, this study investigated the relationship between measures of fear of emotion in a clinical population.

In previous research, the association between fear of emotion and emotional disorders was undertaken on patients with a single psychological disorder. A further aim of the current study was to determine whether fear of emotion is associated with psychological disorders in a general clinical sample. This adds to existing literature in that community samples often display high levels of comorbidity, which is a common exclusion criterion in research studies.

Efficacious treatments for anxiety disorders and depression have been developed and include CBT and pharmacotherapy. Treatment of anxiety and depression with these therapies also results in reductions in fear of emotion. The research concerning the combined use of these two therapies is in its early stages. While combined therapy is a recommended clinical approach in some circumstances (e.g., severe depression), its use in other disorders is largely ahead of the evidence. Some studies show that combined therapy is no more effective than CBT alone and some preliminary studies show that combining pharmacotherapy with CBT may reduce the effectiveness of the CBT treatment. An important aim of the current study was to determine if an underlying transdiagnostic mechanism, that is fear of emotion, was associated with differing outcomes to help clarify the role of the two therapeutic approaches. Further, the pharmacotherapy that has been reported in previous studies has tended to reflect the availability of agents at the time of the research, often in the 1980s and 1990s. Since then, newer agents have been developed and there has been a shift to prescribing these drugs. The pharmacotherapy profiles of patients presenting for treatment to a community mental health centre are more representative of current clinical practice.

This study built on what is known about fear of emotion as measured by the ASI, and assessed this construct further by using the ACS. This allowed for a more

precise understanding of the construct, its association with psychological disorders and its change over treatment. This was particularly important as currently no published literature was found assessing the impact of treatment on fear of emotion measured by the ACS in medicated patients.

In summary, the purpose of this study was to investigate fear of emotion using two measures, one well established (ASI), and the other less so (ACS). The study sought to determine whether fear of emotion was specific to particular disorders, or whether it represented a more global transdiagnostic construct. The study also investigated changes in fear of emotion over treatment with either CBT alone, or in combination with naturalistic pharmacotherapy. A further exploratory aim was to investigate whether the change in fear of emotion over treatment differed between groups of patients undergoing CBT in individual or group format.

The study provided data to answer the following questions: (1) Are ASI and ACS measuring a similar construct (fear of emotion)? (2) Is fear of emotion associated with anxiety and depression in patients presenting for treatment at a community mental health centre? (3) Does fear of emotion change over treatment with either CBT alone or combined with pharmacotherapy? (4) Do scores decrease more in patients receiving CBT without pharmacotherapy than in those receiving pharmacotherapy in addition to CBT?

Chapter 3 Method for Study 1

Participants

Six hundred and fifty two adults, who had received treatment at the Centre for Clinical Interventions (CCI) in Perth, Western Australia, between July 2000 and April 2011, and for whom pre-treatment and post-treatment data were available, consented to participate in the current study. Of these, 452 were receiving antidepressant or anxiolytic medication prescribed by their medical practitioner.

CCI is a government-funded clinical psychology service treating patients for anxiety, mood and eating disorders. The clinical staff have all undertaken post-graduate studies in clinical psychology and, where eligible, are endorsed by the Australian Health Practitioner Regulation Agency for clinical psychology. The service provides individual treatment for eating disorders, individual and group treatment programs for bipolar disorder (as an adjunctive treatment to psychiatric management), and social anxiety disorder. Transdiagnostic treatment groups are also provided for patients with anxiety and depression and for those presenting with pathological repetitive negative thinking. The service accepts referrals for adults from the entire state of Western Australia, although the majority of patients live in the Perth metropolitan area.

Participants were referred to CCI from a variety of sources including GPs, psychiatrists, and other government agencies. Participants were assessed at an initial screening interview to determine clinical diagnosis. Those with a primary diagnosis of depressive disorder (depressive episode, recurrent depressive disorder, or dysthymia) or anxiety disorder (panic disorder with or without agoraphobia, generalized anxiety disorder, social anxiety disorder, specific phobia, obsessive compulsive disorder, post-traumatic stress disorder, agoraphobia, or mixed anxiety-depressive disorder) as defined by the DSM-IV (American Psychiatric Association, 1994) criteria were included.

Potential participants with co-morbid psychosis, substance misuse, or eating disorders

were excluded. Participants completed an intake assessment questionnaire which gathered details about their presenting problems and included information about their age, gender, country of birth, marital status, educational background and current employment.

The characteristics of the participants are displayed in Table 9. The most common primary diagnosis was a depressive disorder (51.1%), followed by social anxiety disorder (14.0%), generalised anxiety disorder (10.4%), specific phobia (9.5%), other anxiety disorders (8.1%), and panic with or without agoraphobia (6.7%). Just over 60% of patients met criteria for more than one disorder, with depressive disorder being the most common comorbid diagnosis (20.0%), followed by generalised anxiety disorder (11.6%), social anxiety disorder (8.5%) and specific phobia (4.4%). The mean time since onset of the current problems was 12 years and seven months.

Of the 452 patients who were medicated, 334 reported taking antidepressants, 20 taking anxiolytics, and 98 taking a combination of an antidepressant and an anxiolytic. For those taking antidepressant medication, 211 were taking an SSRI, 19 a TCA, 72 a combination of antidepressants and 130 were taking other antidepressants (e.g. venlafaxine, desvenlafaxine, mirtazapine). This pharmacotherapy was considered 'naturalistic' as it represented typical prescribing patterns as was not regulated for this study.

Measures

Diagnostic interview. The Mini International Neuropsychiatric Interview-PLUS (MINI PLUS Version 5.0; Sheehan et al., 2001) was administered as part of a clinical assessment interview by therapists at CCI to determine a primary diagnosis. The MINI is a structured interview used to diagnose Axis I disorders based on the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 2000)*. It has good inter-rater and test-retest reliability, and has

been found to converge with other diagnostic interviews, including the Structured Clinical Interview for DSM (SCID) and Composite International Diagnostic Interview (CIDI, Lecrubier et al., 1997; Sheehan et al., 2001).

Symptom measures. Beck Depression Inventory Revised: The Beck Depression Inventory Revised (BDI-II; Beck, Steer, & Brown, 1996) is a widely used, 21-item self-report measure of depressive symptom severity. Scores range from 0 to 63, with higher scores representing more symptoms of greater severity. The BDI-II has high one-week test-retest reliability, r = .93, and high internal consistency, Cronbach's $\alpha = .91$ (Beck, Steer, Ball, & Ranieri, 1996; Beck, Steer, & Brown, 1996). This instrument was administered at assessment (pre-treatment), and at the completion of treatment (post-treatment).

Beck Anxiety Inventory: The Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) is similar in format to the BDI-II, and is a self-report measure of anxiety symptoms. The test has adequate one-week test-retest reliability, r = .75, and high internal consistency, $\alpha = .92$ (Beck, Epstein, Brown, & Steer, 1988; Steer, Ranieri, Beck, & Clark, 1993). The BAI was administered at the same times as the BDI-II.

Fear of emotion measures. Anxiety Sensitivity Index: The Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986) is a 16-item self-report questionnaire designed to assess the dispositional tendency to feared physiological and cognitive symptoms of anxiety due to the belief that these symptoms may be harmful or dangerous. It is currently the most widely used measure of the anxiety sensitivity construct. Each item is rated on a five-point Likert scale ranging from 0 (very little) to 4 (very much). The measure has moderate to high internal consistently (α =.82 to .91) and moderate test-retest reliability over two weeks (r=.75; Reiss et al., 1986) and over three years (r=.71; Maller & Reiss, 1992).

Despite being developed as a measure of a single construct, the ASI has a hierarchical structure, consisting of a single higher-order factor, and three lower order factors. Using factor analyses, these lower order dimensions are described as Mental Concerns, Physical Concerns and Social Concerns. Mental Concerns represents a fear of mental impairment or loss of cognitive control, Physical Concerns represents fear of physical sensations of emotion, and Social Concerns represents the fear that symptoms of emotion will be observable to others (Zinbarg, Barlow, & Brown, 1997; Rodriguez, Bruce, Pagano, Spencer, & Keller, 2004).

Affective Control Scale: The Affective Control Scale (ACS; Williams, Chambless, & Ahrens, 1997) is a 42-item self-report instrument designed to assess anxiety about loss of control over a person's emotions and a person's reactions to those emotions (Williams et al., 1997). The scale is composed of four subscales measuring apprehension about the experience of different emotions: Fear of Anger, Fear of Positive Emotion, Fear of Depression, and Fear of Anxiety. Individual scale items are rated on a 7-point Likert scale with choices ranging from "very strongly disagree" (1) to "very strongly agree" (7), with some items being reverse scored. Scoring of the scale provides for a total mean score and a score for each of the four subscales. Higher scores indicate higher levels of fearful responding to emotions.

The ACS and each of its subscales have good internal consistency and test-retest reliability in a non-clinical sample (Williams et al., 1997) and this was replicated by Berg, Shapiro, Chambless and Ahrens (1998) in a non-clinical sample. For the total scale, Cronbach's α = .92 and the two week test-retest reliability, r = .78. The subscales show similar properties with Cronbach's α ranging between .72 and .91, and two-week test-retest reliability ranging between .66 and .77 in an undergraduate sample (Williams et al., 1997; Berg et al., 1998). Validation studies show that Fear of Positive Emotion, Depression, and Anger predict panic sensations over and above the Fear of Anxiety

subscale, while controlling for trait and state anxiety (Williams et al., 1997; Berg et al., 1998).

The two fear of emotion measures were administered at initial assessment (pretreatment), and at the completion of treatment (post-treatment).

Procedure

Data from the symptom and fear of emotion measures analysed in the current study were collected from the participants as part of their treatment at CCI. Upon referral to CCI, patients were screened at an intake assessment interview with the MINI PLUS and completed the self-report measures. Patients were then assigned for either individual therapy or group therapy depending on their presenting problem or availability to attend group therapy sessions. For individual therapy, patients received a program of manualised cognitive behavioural therapy (Nathan, Rees, Lim, & O'Donnell, 2001) based on Beck's (1979) manual for depression. Patients receiving individual therapy were treated by a clinical psychologist or clinical psychologist registrar for between two and 30 sessions (*M*=12.16, *SD*=5.0). The patients receiving group therapy attended a manualised Mood Management Course (Nathan, Rees, & Smith, 2001) consisting of 10 two-hour weekly sessions, and a follow-up session after one month. These were facilitated by at least one clinical psychologist and a post-graduate clinical psychology trainee.

The patients receiving pharmacotherapy were taking various regimens of antidepressant and anxiolytic medications as prescribed and monitored by their referring general practitioner or psychiatrist. The assessing clinician at CCI had recorded each patient's self-reported regimen. This information was reviewed after treatment had been completed and was coded according to current Australian prescribing guidelines (National Prescribing Service Limited, 2004). Antidepressant medications were coded as tricyclic antidepressants (TCA; e.g. amitriptyline, imipramine, dothiepin), selective

serotonin re-uptake inhibitors (SSRI; e.g. citalopram, fluoxetine, sertraline), or other (e.g. venlafaxine, mirtazepine).

The author was responsible for the design of the study in consultation with the clinic director, and was a co-facilitator of the group treatment programs from 2008 to 2011. The author had sole responsibility for the pharmacotherapy review.

Analyses

To answer the questions posed in the rationale for Study 1 a series of analyses were carried out using SPSS version 22. Firstly, to determine whether demographic or clinical characteristics differed between medicated and unmedicated patients on entry to treatment, independent samples t-tests were performed. Two-way between-groups analyses of variance (ANOVA) with post-hoc comparisons using the Tukey HSD test were conducted to determine differences between medicated and unmedicated patients on fear of emotion measures and their subscales according to primary diagnosis.

The first research question asked if the ASI and ACS were measuring a similar construct (i.e. fear of emotion). It was expected that there would be a moderate to strong association between the total and subscale scores. The relationships between measures of fear of emotion were computed for the ASI and ACS using Pearson correlations to assess this. The second question asked if fear of emotion was associated with anxiety and depression in patients presenting for treatment. It was hypothesised that in patients diagnosed with depression and/or an anxiety disorder, scores on measures of fear of emotion would moderately predict severity of depression and anxiety symptom scores. To test this, a series of multiple regression analyses were performed using both the total scale scores and the subscale scores of each measure. The third and fourth questions asked if fear of emotion changed over treatment with either CBT alone or combined with pharmacotherapy, and whether scores decreased with one type of treatment more than the other. It was expected that fear of emotion scores would decrease with either

treatment, and that patients undergoing CBT alone would show larger decreases in fear of emotion scores than those undergoing CBT while taking naturalistic pharmacotherapy. Change in fear of emotion over treatment was assessed with a 2 x 2 x 2 mixed design ANOVA with medication as the between subject variable with two levels (medicated and not medicated) and time as the within subject variable with two levels (pre-treatment and post-treatment). To examine change in fear of emotion using subscale scores, a doubly-multivariate analysis of variance (MANOVA) was performed with medication as the between subjects independent variable with two levels (medication, no medication). The within subjects variable was time with two levels (pre-treatment and post-treatment). Similarly, to investigate differences in types of medication, two 2 x 2 mixed design ANOVAs were conducted with type of medication as one between subjects variable with four levels (no medication, antidepressant only, anxiolytic only and antidepressant with anxiolytic). The second between subjects variable was type of therapy (individual or group). As with the previous analysis, this was followed by a doubly-multivariate analysis of variance (MANOVA) performed on the subscale scores of fear of emotion. Finally, clinically significant change was calculated using methods described by Jacobson and Truax (1991), and defined as scores within two standard deviations of the non-distressed population mean for the ASI and ACS measures.

Chapter 4 Results for Study 1

Participant characteristics

The characteristics of the participants are displayed in Table 9. To determine whether demographic or clinical characteristics differed between medicated and unmedicated patients on entry to treatment, independent samples t-tests were performed on the fear of emotion measures (ASI, ACS) along with patients' age, duration of presenting problem, and number of treatment sessions. For those patients with ASI data, there was no significant difference between the pre-treatment scores for those who were medicated and those who received only CBT, t(650) = .999, p = .318. There was also no significant difference between the two groups for duration of problem (t(569)=-1.22,p=.224), or for the number of treatment sessions (t(633)=-.26, p=.794). There was, however, a significant difference for age, t(650)=3.03, p<.05, with the medicated group being significantly older than the CBT-alone group. Chi-square tests of independence were performed on the variables of gender, individual or group therapy, marital status, education, and comorbidity. The proportion of females to males in the medicated group was not significantly different to the proportion of females to males in the CBT group, χ^2 (1, N=652) = .15, p=.698, and there was no significant difference between the groups for marital status (χ^2 (5, N=645) = 4.58, p=.469), or comorbidity (χ^2 (16, N=408) =16.11, p=.445). The proportion of patients receiving individual rather than group CBT was higher for the unmedicated than medicated group (χ^2 (1, N=652) = 5.71, p < .05), and the proportion of patients who completed education to Year 12 level was higher for the unmedicated group than the medicated group (χ^2 (4, N=597) = 11.55, p<.05).

For those patients with ACS data, there was a significant difference in pretreatment scores, ACS-total t(274)=2.20, p<.05, with the medicated group having higher scores (mean=4.15, SD=.63) than the unmedicated group (mean=3.96, SD=.76). There was again a significant difference for age, t(274)=2.06, p<.05, with the medicated

group being significantly older than the CBT group. There was no significant difference between the two groups for duration of problem (t (241) = -.33, p=.743), or for the number of treatment sessions (t (261) = .24, p=.807). Chi-square tests of independence showed that the patients who were medicated and receiving CBT were similar to those receiving CBT-alone in terms of the proportion of females in each group (χ^2 (1, N=276) = .69, p=.408), marital status (χ^2 (5, N=272) = 10.22, p=.069), education (χ^2 (4, N=251) = 8.76, p=.067), and comorbidity (χ^2 (12, N=202) = 11.20, p=.512). Unlike those patients who had completed the ASI measure, there was no significant difference between the proportion of those receiving individual or group therapy, χ^2 (1, N=276) = 3.47, p=.063.

In summary, for those who completed the ASI, medicated and unmedicated patients were similar for most variables except age but a greater proportion of those who received CBT alone received individual therapy and had been educated to Year 12 level. For those who completed the ACS, the groups were also similar, although the medicated group had higher total scale scores at pre-treatment and were also older.

Table 9

Characteristics of Patients by Treatment-type Group.

Variable	ASI		ACS	
	CBT + Medicated	CBT	CBT + Medicated	CBT
Number of patients	452	200	192	84
Gender (% female)	63.1	65.0	63.0	69.0
Mean age in years (SD)	38.3 (12.7)*	35.0 (13.0)*	38.5 (12.7)*	35.5 (13.0)*
Marital status (%)				
Single (never married)	42.3	41.8	45.5	40.7
Married	24.7	21.9	25.7	21.0
Divorced/separated	18.1	15.8	15.7	21.0
Education (highest level %)				
Completed to year 10	17.1	13.8	14.3	7.9
Completed to year 12	21.6*	34.3*	21.1	38.2
Technical/trade	21.2	16.0	22.9	17.1
Completed tertiary	36.5	31.5	37.7	32.9
Comorbid Axis 1 diagnosis (%)	54.9	51.0	59.9	52.4
Duration of problem in months (SD)	49.2 (81.4)	58.2 (79.0)	66.0 (107.0)	70.8 (94.7)
Number of treatment sessions (SD)	11.1 (5.8)	11.2 (5.8)	12.4 (6.2)	12.2 (5.7)
Individual CBT (%)	39.6*	50.0*	34.9	47.6

^{*} indicates significant difference between group means

Two-way between-groups analyses of variance (ANOVA) were conducted to determine differences between medicated and unmedicated patients on fear of emotion measures according to their primary diagnosis (see Table 10). For ASI scores, the main effect for primary diagnosis (F(5, 640)=.99, p=.42), medication (F(1, 640)=1.72, p=.19) and the interaction effect (F(5,640)=1.29, p=.27) did not reach statistical significance. For ACS scores, there was a significant main effect for primary diagnosis (F(5, 264)=2.56, p=.03) with a small to moderate effect size ($\eta^2=.04$). Post-hoc comparisons using the Tukey HSD test indicated that the mean score for patients with a primary diagnosis of a depressive disorder (M=4.20, SD=.65) was significantly greater than the mean score for patients with a primary diagnosis of panic disorder with or without agoraphobia (M=3.65, SD=.83) and those with generalized anxiety disorder (M=3.81, SD=.60). The main effect for medication (F(1,264)=1.53, p=.22) and the interaction effect (F(5,264)=.92, p=.47) did not reach statistical significance.

To explore this further and to test whether fear of particular emotions differed between primary diagnoses in those patients who were medicated and those who were not, two-way between-groups ANOVAs were conducted. The dependent variables were the subscale scores of the ASI and the ACS. The means and standard deviations are presented in Tables 11 and 12. For the ASI Mental Concerns subscale, the main effect for primary diagnosis was not significant (F(5, 640)=.70, p=.62), but the main effect for medication (F(1, 640)=4.91, p=.03) and the interaction effect (F(5,640)=2.26, p=.05) did reach statistical significance. Post hoc comparison with the Tukey HSD test showed that the mean score on the subscale for patients with a primary diagnosis of panic disorder with or without agoraphobia disorder were higher than those patients diagnosed with a generalized anxiety disorder, but differed depending on medication status with medicated patients with panic disorder having higher scores (M=9.48, SD=3.72) than unmedicated patients (M=5.50, SD=4.41), and unmedicated patients with generalized

anxiety disorder having higher scores (M=7.24, SD=3.75) than medicated patients (M=5.93, SD=4.19). For the ASI Physical Concerns subscale, the main effect for primary diagnosis (F(5, 640)=1.46, p=.20), medication (F(1, 640)=.70, p=.40) and the interaction effect (F(5,640)=.78, p=.56) did not reach statistical significance. For the ASI Social Concerns subscale, there was a significant main effect for primary diagnosis (F(5, 640)=4.13, p=.01) with a small effect size (q²=.03). Post-hoc comparisons using the Tukey HSD test indicated that the mean score for patients with a primary diagnosis of social anxiety disorder (M=10.03, SD=3.05) was significantly greater than the mean score for patients with a primary diagnosis of panic disorder with or without agoraphobia (M=8.18, SD=3.39), generalized anxiety disorder (M=7.98, SD=3.44) or a depressive disorder (M=8.68, SD=3.26).

For the ACS-anger subscale, the main effect for primary diagnosis (F(5, 264)=1.83, p=.11), medication (F(1, 264)=.16, p=.59) and the interaction effect (F(5,264)=1.27, p=.28) did not reach statistical significance. Similarly, for the ACS-anxiety subscale, the main effect for primary diagnosis (F(5, 264)=1.54, p=.18), medication (F(1, 264)=.19, p=.67) and the interaction effect (F(5,264)=.79, p=.56) did not reach statistical significance. For the ACS-positive subscale, there was a significant main effect for primary diagnosis (F(5, 264)=2.84, p=.02) with a small to moderate effect size (q2=.04). Post-hoc comparisons using the Tukey HSD test indicated that the mean score for patients with a primary diagnosis of a depressive disorder (M=3.55, SD=.82) was significantly greater than the mean score for patients with a primary diagnosis of generalized anxiety disorder (M=3.09, SD=.89). The main effect for medication (F(1,264)=.47, p=.49) and the interaction effect (F(5, 264)=.25, p=.94) did not reach statistical significance. Finally, for the ACS-depression subscale, there was a significant main effect for primary diagnosis (F(5, 264)=6.75, p<.01) with a moderate effect size (q2=.09). Post-hoc comparisons using the Tukey HSD test indicated that the mean score

for patients with a primary diagnosis of a depressive disorder (M=5.04, SD=.94) were significantly greater than the mean score for patients with a primary diagnosis of panic disorder with or without agoraphobia (M=3.76, SD=1.24), generalized anxiety disorder (M=4.28, SD=1.09) or specific phobia (M=4.31, SD=1.17). The main effect for medication (F(1,264)=5.00, p=.03) was also significant with mean scores for medicated patients higher than for unmedicated patients. The interaction effect (F(5, 264)=1.23, p=.29) did not reach statistical significance.

In summary, an interaction effect between primary diagnosis and medication status was observed for the ASI Mental Concerns subscale, however the effect size was small. A main effect for medication was found for the ASI Mental Concerns subscale and the ACS-depression subscale with patients who were medicated reporting higher scores on the fear of emotion measures than the patients who were unmedicated. A main effect for primary diagnosis was found for the ASI Social Concerns, ACS-positive and ACS-depression subscales, and the ACS-total scale scores.

Table 10

Mean (SD) Scores on Fear of Emotion Scores by Primary Diagnosis and Treatment-type Group.

Primary Diagnosis	ASI Mean (SD)				ACS Mean (SD)			
	N	CBT + Medicated	N	CBT	N	CBT + Medicated	N	CBT
Panic disorder with or without	24	35.42 (11.64)	20	27.85 (12.14)	8	3.83 (.83)	7	3.44 (.84)
agoraphobia								
Social anxiety disorder	50	32.12 (11.75)	41	27.27 (13.24)	16	4.16 (.55)	7	3.97 (.47)
Generalized anxiety disorder	45	25.53 (13.49)	23	28.48 (13.12)	27	3.82 (.64)	14	3.80 (.53)
Specific phobia	37	28.27 (13.00)	25	27.64 (11.59)	18	4.18 (.77)	12	3.99 (.74)
Other anxiety disorder	37	27.51 (13.98)	17	27.29 (14.58)	18	4.34 (.60)	6	3.33 (.71)
Depressive disorder	259	29.61 (12.65)	74	29.57 (17.73)	105	4.27 (.57)	38	3.98 (.80)

Table 11

Mean (SD) Scores on ASI Subscale Scores by Primary Diagnosis and Treatment-type Group.

Primary Diagnosis		Mental Concerns Mean (SD)			Physical Concer	ns Mean (SD)	Social Concerns Mean (SD)	
	N	CBT + Medicated	N	CBT	CBT + Medicated	CBT	CBT + Medicated	CBT
Panic Disorder with or without Agoraphobia	24	9.48 (3.72)	20	5.50 (4.41)	7.81 (3.36)	7.50 (4.15)	8.81 (3.39)	7.44 (3.31)
Social anxiety disorder	50	8.57 (3.99)	41	6.38 (4.39)	7.10 (3.24)	5.73 (3.89)	10.43 (2.64)	9.58 (3.43)
Generalized anxiety disorder	45	5.93 (4.19)	23	7.24 (3.75)	6.08 (4.10)	6.90 (3.81)	7.76 (3.40)	8.43 (3.57)
Specific phobia	37	6.86 (4.40)	25	6.80 (3.95)	6.04 (3.67)	5.38 (3.61)	9.32 (3.02)	10.08 (3.15)
Other anxiety disorder	37	6.94 (4.35)	17	6.47 (5.04)	6.58 (4.23)	5.90 (4.57)	8.21 (3.25)	9.07 (2.92)
Depressive disorder	259	7.82 (4.27)	74	7.14 (4.83)	6.69 (3.66)	6.86 (4.24)	8.67 (3.18)	8.69 (3.52)

Table 12

Mean (SD) Scores on ACS Subscale Scores by Primary Diagnosis and Treatment-type Group.

Primary Diagnosis	Anger Mean (SD)		Positive 1	Mean (SD)	Depression	Mean (SD)	Anxiety l	Anxiety Mean (SD)		
	N	CBT + Med	N	CBT	CBT + Med	CBT	CBT + Med	CBT	CBT + Med	CBT
Panic Disorder w or w/o Ag	8	3.69 (1.12)	7	3.34 (1.35)	2.87 (.84)	2.86 (.92)	4.27 (1.22)	3.18 (1.04)	4.61 (.81)	4.26 (.84)
Social anxiety disorder	16	3.85 (.70)	7	3.62 (1.02)	3.62 (1.11)	3.42 (.54)	4.60 (1.17)	4.13 (.78)	4.62 (.59)	4.65 (.41)
Generalized anxiety disorder	27	3.91 (.95)	14	3.88 (.92)	3.17 (.95)	2.93 (.76)	4.29 (1.01)	4.26 (1.26)	4.13 (.85)	4.36 (.85)
Specific phobia	18	4.34 (1.05)	12	3.92 (.93)	3.47 (1.00)	3.51 (1.16)	4.47 (1.20)	4.08 (1.11)	4.64 (.80)	4.46 (.76)
Other anxiety disorder	18	3.80 (.75)	6	4.65 (.88)	3.58 (.86)	3.64 (.86)	4.36 (.97)	4.73 (.74)	4.22 (.95)	4.46 (.88)
Depressive disorder	105	4.23 (.97)	38	3.94 (1.00)	3.61 (.74)	3.39 (1.03)	5.17 (.84)	4.64 (1.12)	4.41 (.82)	4.12 (.99)

Measuring fear of emotion

To determine if there is a shared relationship between measures of fear of emotion a correlation matrix was computed for the ASI and ACS. The correlation matrix in Table 13 shows the relationships between each of the total scores for the measures and the sub-scale scores. The values of Pearson's *r* ranged from .169 to .918, indicating a wide range of strengths in the relationships among the sub-scale scores.

The correlations between the ASI and its subscales showed high values with the total scale score strongly related to each of the three sub-scale scores. The values of Pearson's r ranged between .740 and .918, n=605, p<.05. The relationship between the ASI subscales was somewhat weaker, but still strong with values between .527 and .591, n=605, p<.05.

The relationship between the total score of the ACS and its subscales was also strong with values of Pearson's r between .638 and .773, n=334, p<.05. Between the sub-scale scores, the values were lower, showing moderately strong relationships with vales of r between .289 and .411, n=334, p<.05.

The total scale scores show a moderate correlation, r=.459, n=334, p<.05, indicating some shared variance across the fear of emotion construct. However, the relationship between total and sub-scale scores between measures shows a less consistent pattern. Large values for Pearson's r were seen between the total score of the ASI (ASI-total) and the anxiety subscale of the ACS (ACS-anxiety); the total score of the ACS (ACS-total) and the mental subscale of the ASI (ASI-mental); and ASI-mental and ACS-anxiety. Moderate values for Pearson's r were seen between ASI-mental and the depression subscale of the ACS (ACS-depression); the physical subscale of the ASI (ASI-physical) and ACS-anxiety; ASI-physical and ACS-total; and the social subscale of the ASI (ASI-social) and ACS-anxiety (see Table 13 for values).

Thus, there was considerable overlap on the total scale scores of each measure, suggesting both measures access a fear of emotion construct. Each measure's subscales appear to have some specificity although there were considerable overlaps between the ASI subscales and the anxiety subscale on the ACS (ACS-anxiety).

Table 13

Pearson Product-Moment Correlations Between Total and Subscale Scores on ASI and ACS.

	ASI	ASI	ASI	ASI	ACS	ACS	ACS	ACS	ACS
	Total	Mental	Physical	Social	Total	Anger	Positive	Depression	Anxiety
ASI Total									
ASI Mental	.817*								
ASI Physical	.918*	.591*							
ASI Social	.740*	.533*	.527*						
ACS Total	.459*	.500*	.346*	.367*					
ACS Anger	.233*	.241*	.169*	.217*	.666*				
ACS Positive	.262*	.297*	.199*	.188*	.773*	.384*			
ACS Depression	.278*	.327*	.215*	.178*	.638*	.298*	.289*		
ACS Anxiety	.512*	.533*	.384*	.445*	.756*	.345*	.411*	.322*	

For correlations between ASI-total and ASI subscales, n=605, for remainder, n=334.

^{*}p<.01

Predicting severity of anxiety and depression symptoms by fear of emotion

To determine how strongly fear of emotion is related to depression and anxiety, a series of multiple regression analyses were performed.

Firstly, a hierarchical multiple regression analysis was performed with pretreatment BDI scores as the dependent variable and the total scores on the ASI and ACS at pre-treatment as predictor variables. Medication use was entered in the first step to control for any effect it may have on symptom scores. Table 14 shows the standardised regression coefficients (β), the variance and the change in variance accounted for by the model. In the final model, R for regression was significant, F(3,277)=39.25, p<.05. Both ASI-total and ACS-total pre-treatment scores were significant predictors of depressive symptoms. Together, scores on ACS-total and ASI-total at pre-treatment accounted for 29.8% variance in the pre-treatment BDI scores.

A second hierarchical multiple regression was performed with pre-treatment BAI scores as the dependent variable, and the same predictor variables as for the first regression analysis. Medication use was again entered in the first step. The coefficients and probability values are also shown in Table 14. R for regression was significant, F(3,273)=47.74, p<.05. Both ASI-total and ACS-total scores at pre-treatment were significant predictors of anxiety symptoms. Together, scores on pre-treatment ASI-total and ACS-total scales accounted for 34.4% of variance in the pre-treatment BAI scores.

These results suggest that fear of emotion is a weak to moderate predictor of depression and anxiety symptoms, independent of medication status.

Table 14
Summary of Stepwise Multiple Regression Predicting BDI and BAI Scores from ASI-total and ACS total Pre-treatment Scores.

	β	t	R^2 (adjusted R^2)	ΔR^2	\overline{F}
BDI					
Step 1			.02 (.01)	.02	5.06*
Medication	133*	-2.25			
Step 2			.30 (.29)	.28*	39.25*
ASI-total	.169*	2.98			
ACS-total	.436*	7.60			
BAI					
Step 1			.00 (01)	.00	.04
Medication	.01	.19			
Step 2			.34 (.34)	.34*	47.74*
ASI-total	.524*	9.49			
ACS-total	.118*	2.11			

^{*}*p*<.05

To explore this further and to test whether this fear of emotion is specific to particular emotions or is more representative of emotion vulnerability, the subscales of the ASI and ACS were used in multiple regression analyses.

A hierarchical multiple regression analysis was performed with pre-treatment BDI scores as the dependent variable and the sub-scale scores on the ASI and ACS at pre-treatment as predictor variables. Medication use was entered in the first step to control for any effect on the dependent variable. Table 15 shows the standardised regression coefficients (β), the variance and the change in variance accounted for by the

model. R for regression was significant, F(8,280)=21.34, p<.05. Together, scores on ASI subscales and ACS subscales explained 38.6% of the variance in the pre-treatment BDI scores. Only three of the sub-scales contributed significantly to the prediction of depressive symptoms. They were ACS-depression (β =.37, p<.05), ASI-mental (β =.21, p<.05), and ACS-anger (β =.17, p<.05).

The analysis was repeated with pre-treatment BAI scores as the dependent variable. The coefficient values and variance are shown in Table 15. R for regression was significant, F(8,268)=19.84, p<.05. The scores on the subscales of the two measures accounted for 37.2% of the variance in pre-treatment BAI scores. The three subscales that uniquely contributed to the prediction of anxiety symptoms were ASI-mental ($\beta=.26$, p<.05), ASI-physical ($\beta=.28$, p<.05) and ACS-anxiety ($\beta=.20$, p<.05).

Table 15

Summary of Stepwise Multiple Regression Predicting BDI and BAI Scores from ASI subscale and ACS subscale Pre-treatment Scores.

	β	t	R^2 (adjusted R^2)	ΔR^2	\overline{F}
BDI	•		<u> </u>		
Step 1			.02 (.01)	.02	5.06*
Medication	133*	-2.25			
Step 2			.39 (.37)	.37*	21.34*
ASI-mental	.31*	3.07			
ASI-physical	04	71			
ASI-social	.10	1.72			
ACS-anger	.17*	3.20			
ACS-positive	.05	.92			
ACS-					
depression	.37*	6.96			
ACS-anxiety	.01	.10			
BAI					
Step 1			.00 (01)	.00	.04
Medication	.01	.19			
Step 2			.37 (.35)	.37*	19.84*
ASI-mental	.26*	3.70			
ASI-physical	.28*	4.39			
ASI-social	.03	.50			
ACS-anger	.06	1.13			
ACS-positive	07	-1.29			
ACS-					
depression	02	35			
ACS-anxiety	.20*	3.14			

^{*}*p*<.05

In both analyses, the subscale scores account for a greater proportion of the variance in symptoms than the total scale scores. For depression symptoms, the subscale scores accounted for 39% of the variance compared with the total scale scores of 30%. Similarly for anxiety symptoms, the subscale scores accounted for 37% of the variance compared with 34% for the total scale scores. These results also indicate some specificity in fear to particular emotions in that scores on the ACS-depression subscale were the strongest independent significant predictor of depressive symptoms (along with ASI-mental and ACS-anger). For anxiety symptoms, ASI-physical was the strongest independent predictor, along with ASI-mental and ACS-anxiety.

When considering the two sets of analyses together, the results showed that fear of emotion as measured by the total scores on the ASI and ACS was a weak to moderate predictor of depression and anxiety symptoms, independent of medication status. The subscales explained a greater proportion of variance in symptoms, and suggested some specificity in the subscales in differentiating between the types of symptoms, with depression symptoms being predicted by scores on the ACS-depression, ASI-mental, and ACS-anger subscales, and anxiety symptom scores being predicted by scores on ASI-physical, ASI-mental and ACS-anxiety subscales.

Change in fear of emotion over treatment

CBT and pharmacotherapy, alone or in combination, have been shown to be useful treatments for many psychological disorders including anxiety and depression. The usefulness of these treatments has been demonstrated in efficacy studies where active treatments are compared with control groups in strictly controlled conditions (e.g., randomised controlled trials), such that conclusions can be drawn on the effect of the treatment on the defined outcome. Further evidence for the usefulness of CBT and pharmacotherapy has been derived from effectiveness studies, where the treatments are evaluated as they are actually delivered in clinical practice, and can be considered to have high external validity. As fear of emotion is associated with anxiety and depression, it would be expected to change over treatment, with scores on measures of fear of emotion lower at post-treatment than at pre-treatment.

Further, those receiving CBT without pharmacotherapy may reduce their scores to a greater extent than those receiving pharmacotherapy in addition to CBT (since medications such as anxiolytics combined with CBT have been shown to be less effective than CBT alone, particularly when taken on a 'prn' (as required) basis).

To test these hypotheses, consideration was given to the use of mixed betweenwithin subjects analyses of covariance (ANCOVA) to test for differences between groups while controlling for differences in the pre-treatment variable of age. For this analysis to render meaningful results, three assumptions need to be met (Stevens, 2002). These assumptions are a linear relationship between the dependent variables and the covariate, homogeneity of the regression slopes, and reliability of the measurement of the covariate. To check for linearity for each group, scatterplots with fit lines were generated for age and each dependent variable (ASI-total and ACS-total). For both dependent variables no relationship was shown between them and age with fit lines running almost horizontally. The lack of relationship was further demonstrated by very small R squared values for both the medicated and unmedicated groups. For ASI-total, R squared for the medicated group was .007 and for the unmedicated group was .012. For ACS-total, R squared for the medicated group was .003 and for the unmedicated group .008. In determining homogeneity of regression slopes, no significant interaction was found between the treatment groups and the covariate of age. While this shows that the assumption of homogeneity was not violated, it has little meaning given the weakness of the relationship between age and the dependent variables. With regard to the reliability of the measurement of the covariate, age was measured without error. Therefore, despite two of the three assumptions being met, the lack of any meaningful relationship between age and ASI-total and ACS-total rendered the use of an ANCOVA approach unsuitable.

A 2 x 2 x 2 mixed design ANOVA was conducted with medication as the between subject variable with two levels (medicated and not medicated) and time as the within subject variable with two levels (pre-treatment and post-treatment). As the proportion of patients receiving individual therapy differed between the medicated and non-medicated groups, an additional between subjects variable was included with two levels (individual and group). The dependent variables were the total scores on the ASI and the ACS. The means and standard deviations are presented in Table 16.

For the ASI-total, there was a significant main effect for time, Wilks' Lambda=.602, F(1,648)=428.62, p<.05. This indicates that scores on the ASI-total were significantly lower at post-treatment than they were at pre-treatment. Partial eta squared had a value of .40 indicating a large effect size according to Cohen's (1988) criteria. The main effect for medication approached significance, F(1,648)=3.70, p=.06, with a small effect size, η^2 =.01, suggesting that those receiving CBT-alone had lower scores at pre- and post-treatment than those receiving CBT in addition to their pharmacotherapy. The main effect for individual or group therapy was not significant, F(1,648)=2.48, p=.12, with a very small effect size, η^2 =.004. The interaction terms were not significant for Time x Medication (F(1,648)=1.96, p=.16), Time x Individual/Group (F(1,648)=2.78, p=.10), Medication x Individual/Group (F(1,648)=.34, p=.56), or Time x Medication x Individual/Group (F(1,648)=.76, p=.38).

For the ACS-total, there was a significant main effect for time, Wilks' Lambda=.689, F(1,272)=122.59, p<.05. This indicates that scores on the ACS-total were significantly lower at post-treatment than they were at pre-treatment. Partial eta squared had a value of .31 indicating a very large effect size. There was a significant main effect for medication, F(1,272)=4.20, p<.05, η^2 =.02, with those taking medication having higher scores than those who did not take medication, a small to moderate effect size. There also was a significant main effect for individual or group therapy, F(1,272)=14.34, p<.05, η^2 =.05, with those receiving individual therapy showing lower scores than those receiving group therapy with a small to moderate effect size.

The interaction effect between Time x Individual/Group was significant, Wilks' Lambda=.983, F(1,272)=4.72, p<.05, η^2 =.02. The patients participating in individual therapy showed larger decreases in their scores from pre-treatment to post-treatment than did those participating in group therapy.

More patients in the unmedicated than medicated group had completed education to year 12 level. This is unlikely to be important since the few studies examining demographic variables as predictors of response to CBT and medication have found no differences between age, gender, race, marital status, education or occupation (Hamilton & Dobson, 2002).

Table 16

Descriptive Statistics for Scores on Measures of Fear of Emotion at Pre- and Post-treatment.

Measure	Medicated	Individual or Group	N	Mean	Standard deviation
ASI-total					
Pre-tx	Yes	Individual	179	29.17	13.23
		Group	273	29.60	12.54
	No	Individual	100	28.03	13.49
		Group	100	28.70	13.57
Post-tx	Yes	Individual	179	18.56	12.42
		Group	273	19.91	12.21
	No	Individual	100	15.03	10.12
		Group	100	18.38	12.19
ACS-total		•			
Pre-tx	Yes	Individual	67	4.10	.65
		Group	125	4.18	.62
	No	Individual	40	3.78	.80
		Group	44	4.12	.68
Post-tx	Yes	Individual	67	3.47	.84
		Group	125	3.75	.74
	No	Individual	40	3.19	.68
		Group	44	3.73	.67

A doubly-multivariate analysis of variance (MANOVA) was performed on the subscale measures of fear of emotion to determine the effects of medication. The between subjects independent variable was medication with two levels (medication, no medication). Given the pre-treatment differences in the proportion of patients receiving individual or group therapy, a dummy between-subjects variable was included with two levels (individual, group). The within subjects variable was time with two levels (pretreatment and post-treatment). The dependent variables were the scores on the ASI subscales and the scores on the ACS subscales. Both sets of subscale scores were used in the analysis as the previous results from the correlation analyses showed that the subscale scores were measuring different aspects of fear of emotion. To avoid problems with multicollinearity and singularity, total scores on the ASI and ACS were not included as dependent variables due to the high positive correlations with the respective subscale scores. Only one r value was above the suggested cut off of 0.9 (Grice & Iwasaki, 2007), but the decision to exclude total scores was made on theoretical grounds as the sub-scale scores are derived from the same items making up the total scale scores. The means and standard deviations are presented in Table 17.

There was a significant multivariate effect for time, Wilks' Lambda=.527, F(7,269)=34.50, p<.05, indicating that scores on the measures of fear of emotion were significantly lower at post-treatment than they were at pre-treatment. Partial eta squared had a value of .473 indicating a very large effect size. The observed power was strong, 1.000. Univariate analysis of each individual dependent variable, using a Bonferroni adjusted alpha level of .007, showed that ASI-mental (η^2 =.35), ASI-physical (η^2 =.33), ASI-social (η^2 =.27), ACS-anger (η^2 =.14), ACS-positive (η^2 =.06), ACS-depression (η^2 =.26), and ACS-anxiety (η^2 =.38) decreased significantly over time with moderate to strong effect sizes.

The multivariate effect for medication was not significant, Wilks' Lambda=.963, F(7,269)=1.46, p=.18. Univariate analysis of each individual dependent variable, using a Bonferroni adjusted alpha level of .007, showed that only ACS-depression (η^2 =.02) and ACS-anxiety (η^2 =.01), were significantly associated with medication use.

There was a significant multivariate effect for individual versus group, Wilks' Lambda=.918, F(7,269)=3.42, p<.05, η^2 =.08, on the combined dependent variable fear of emotion. The observed power was strong, .963.

Each of the interaction effects failed to reach significance. For time by medication, Wilks' Lambda=.991, F(7,269)=.35, p=.93, η^2 =.01, for time by individual or group, Wilks' Lambda=.969, F(7,269)=1.23, p=.29, η^2 =.03, for medication by individual or group, Wilks' Lambda=.976, F(7,269)=.96, p=.46, η^2 =.02, and for time by medication by individual or group, Wilks' Lambda=.975, F(7,269)=.97, p=.45, η^2 =.02.

In summary, these results show that across treatment, measures of fear of emotion decreased for those receiving CBT alone and for those receiving CBT with medication. The results also suggest that there was a difference both before and after treatment between those receiving medication and those receiving CBT alone, which may be related to the emotions of depression and anxiety specifically. The multivariate interaction effects did not reach significance.

Table 17

Descriptive Statistics for Subscale Scores on Measures of Fear of Emotion at Pre- and Post-treatment.

Measure	Medicated	Individual or Group	N	Mean	Standard deviation
ASI-mental					
Pre-tx	Yes	Individual	69	7.20	4.35
		Group	124	7.92	4.09
	No	Individual	42	6.67	4.32
		Group	44	7.11	3.83
Post-tx	Yes	Individual	69	4.17	4.02
		Group	124	4.40	3.66
	No	Individual	42	3.10	3.15
		Group	44	4.18	3.02
ASI-physical					
Pre-tx	Yes	Individual	69	6.67	3.95
		Group	124	6.68	3.58
	No	Individual	42	6.56	4.02
		Group	44	6.32	3.56
Post-tx	Yes	Individual	69	4.28	3.34
		Group	124	3.87	3.39
	No	Individual	42	3.61	3.03
		Group	44	3.61	3.34
ASI-social					
Pre-tx	Yes	Individual	69	8.29	2.86
		Group	124	9.15	3.11
	No	Individual	42	8.76	3.04
		Group	44	9.05	3.36
Post-tx	Yes	Individual	69	6.36	3.20
		Group	124	6.74	3.51
	No	Individual	42	6.50	3.65
		Group	44	6.55	3.41

Measure	Medicated	Individual or Group	N	Mean	Standard deviation
ACS-anger					
Pre-tx	Yes	Individual	69	3.95	.84
		Group	124	4.22	1.03
	No	Individual	42	3.71	1.16
		Group	44	4.16	.86
Post-tx	Yes	Individual	69	3.53	.94
		Group	124	3.87	1.05
	No	Individual	42	3.17	.92
		Group	44	3.80	.87
ACS-positive					
Pre-tx	Yes	Individual	69	3.51	.86
		Group	124	3.45	.88
	No	Individual	42	3.09	.99
		Group	44	3.57	1.05
Post-tx	Yes	Individual	69	3.15	.97
		Group	124	3.30	.91
	No	Individual	42	2.90	.79
		Group	44	3.45	.98
ACS-depression					
Pre-tx	Yes	Individual	69	4.53	1.08
		Group	124	4.87	.99
	No	Individual	42	4.22	1.26
		Group	44	4.53	1.00
Post-tx	Yes	Individual	69	3.80	1.32
		Group	124	4.19	1.17
	No	Individual	42	3.33	1.09
		Group	44	4.03	1.00
ACS-anxiety					
Pre-tx	Yes	Individual	69	4.49	.91
		Group	124	4.43	.75
	No	Individual	42	4.17	.90
		Group	44	4.40	.80
Post-tx	Yes	Individual	69	3.59	.92
		Group	124	3.85	.82
	No	Individual	42	3.34	.86
		Group	44	3.74	.65

Next, two 2 x 2 mixed design ANOVAs were conducted with type of medication as one between subjects variable with four levels (no medication, antidepressant only, anxiolytic only and antidepressant with anxiolytic). The second between subjects variable was type of therapy (individual or group). The within subjects variable was time with two levels (pre-treatment and post-treatment). The dependent variables were the total scores on the ASI and the ACS. The means and standard deviations are presented in Table 18.

For the ASI-total, there was a significant main effect for time, Wilks' Lambda=.789, F(1,638)=171.13, p<.05. This indicates that scores on the ASI-total were significantly lower at post-treatment than they were at pre-treatment. Partial eta squared had a value of .211 indicating a large effect size according to Cohen's (1988) criteria. There was also a significant main effect for type of medication, F(3,638)=9.00, p<.05, η^2 =.04. Post hoc comparisons showed that those taking the combination of antidepressant and anxiolytic medication had higher scores than those taking antidepressant medication only, anxiolytic medication only, or no medication. The main effect for type of therapy was not significant, F(1,638)=.15, p=.70. The interaction effect between type of medication and time was not significant (F(3,638)=.63, p=.60, η^2 =.003).

For the ACS-total patients taking only anxiolytic medication (N=7) were not included in the analysis due to the inadequate cell size. There was a significant main effect for time, Wilks' Lambda=.731, F(1,261)=95.85, p<.05. This indicates that scores on the ACS-total were significantly lower at post-treatment than they were at pretreatment. Partial eta squared had a value of .269 indicating a large effect size. The main effect for medication did not reach significance, F(2,261)=2.05, p=.131, $\eta^2=.02$. The main effect for type of therapy was significant, F(1,261)=5.56, p<.05, $\eta^2=.02$, and the interaction effect between time and type of therapy was significant, Wilks'

Lambda=.979, F(1,261)=5.62, p<.05, η^2 =.02, with those receiving individual therapy reporting lower scores on the fear of emotion measure at post-treatment. The interaction effect between type of medication and time was not significant (F(2,261)=1.63, p=.20, η^2 =.01).

Table 18

Descriptive Statistics for Scores on Measures of Fear of Emotion at Pre- and Post-treatment.

Measure	Type of	Individual	N	Mean	Standard
	Medication	or Group			Deviation
ASI-total		-			
Pre-tx	None	Individual	100	28.03	13.49
		Group	102	28.60	13.73
	Antidepressant	Individual	133	27.21	13.14
	-	Group	193	29.21	12.45
	Anxiolytic	Individual	8	25.88	10.58
	·	Group	12	28.17	11.75
	Antidepressant	Individual	37	37.32	11.11
	and anxiolytic				
	Ž	Group	61	31.75	12.84
Post-tx	None	Individual	100	15.03	10.12
		Group	102	18.50	12.30
	Antidepressant	Individual	133	17.33	11.70
	•	Group	193	19.03	12.23
	Anxiolytic	Individual	8	16.13	10.54
	,	Group	12	16.50	11.10
	Antidepressant	Individual	37	23.86	14.10
	and anxiolytic				
	Ž	Group	61	23.46	11.72
ACS-total		1			
Pre-tx	None	Individual	40	3.78	.80
		Group	45	4.14	.68
	Antidepressant	Individual	48	4.06	.61
	1	Group	89	4.25	.62
	Antidepressant	Individual	16	4.25	.78
	and anxiolytic				
	J	Group	29	3.97	.65
Post-tx	None	Individual	40	3.19	.68
		Group	45	3.77	.70
	Antidepressant	Individual	48	3.43	.82
	1	Group	89	3.73	.76
	Antidepressant	Individual	16	3.70	.90
	and anxiolytic				•
	- J	Group	29	3.79	.70

A doubly-multivariate analysis of variance (MANOVA) was performed on the subscale scores of fear of emotion to determine the effects of types of medication. Once again, both sets of subscale scores were used to investigate changes in overall fear of emotion as well as changes to specific aspects of fear of emotion. The between subjects independent variables were type of medication with three levels (no medication, antidepressant only and antidepressant with anxiolytic), and type of therapy. Patients only taking anxiolytic medication were not included in the analysis due to the inadequate cell size. The within subjects variable was time with two levels (pretreatment and post-treatment). The dependent variables were the scores on the ASI subscales and the scores on the ACS subscales. Total scores on the ASI and ACS were once again not included as dependent variables to avoid problems of multicollinearity. The means and standard deviations are presented in Table 19.

There was a significant multivariate effect for time, Wilks' Lambda=.549, F(7,258)=30.32, p<.05, indicating that scores on the combined dependent variable of fear of emotion were significantly lower at post-treatment than they were at pretreatment. Partial eta squared had a value of .451 indicating a very large effect size. The observed power was strong, 1.00. Univariate analysis of each individual dependent variable, using a Bonferroni adjusted alpha level of .007, showed that ASI-mental $(\eta^2=.35)$, ASI-physical $(\eta^2=.30)$, ASI-social $(\eta^2=.23)$, ACS-anger $(\eta^2=.11)$, ACS-positive $(\eta^2=.04)$, ACS-depression $(\eta^2=.21)$, and ACS-anxiety $(\eta^2=.34)$ decreased significantly after treatment.

The results also showed a significant multivariate effect for medication class, Wilks' Lambda=.891, F(14,516)=2.12, p<.05, indicating that scores on the combined fear of emotion variable were significantly different across those not taking medication, those taking antidepressants, and those taking a combination of anti-depressants and anxiolytic medication. The effect size was moderate with a value of partial eta squared

of .06. Univariate analysis of each individual dependent variable, using a Bonferroni adjusted alpha level of .007, showed that only ASI-mental (η^2 =.04) was significantly associated with medication use with patients taking a combination of anti-depressants and anxiolytic medication having the highest scores.

The interaction terms did not reach significance for time by medication class (Wilks' Lambda=.948, F(14,516)=1.00, p=.45, η^2 =.03), time by type of therapy (Wilks' Lambda=.955, F(7, 258)=1.74, p=.10, η^2 =.04), or medication class by type of therapy (Wilks' Lambda=.939, F(14,516)=1.78, p=.29, η^2 =.03).

Table 19

Descriptive Statistics for Subscale Scores on Measures of Fear of Emotion at Pre- and Post-treatment.

Measure	Type of	Individual	N	Mean	Standard
	Medication	or Group			Deviation
ASI-mental					_
Pre-tx	None	Individual	42	6.67	4.32
		Group	45	7.20	3.83
	Antidepressant	Individual	50	6.34	4.34
		Group	88	7.83	4.02
	Antidepressant and anxiolytic	Individual	16	10.38	2.73
	·	Group	29	8.17	4.51
Post-tx	None	Individual	42	3.10	3.15
		Group	45	4.38	3.26
	Antidepressant	Individual	50	3.74	3.72
		Group	88	4.11	3.44
	Antidepressant and anxiolytic	Individual	16	6.13	4.60
	3	Group	29	4.83	4.12
ASI-physical		1			
Pre-tx	None	Individual	42	6.56	4.02
		Group	45	6.41	3.58
	Antidepressant	Individual	50	6.10	3.90
	-	Group	88	6.64	3.69
	Antidepressant and anxiolytic	Individual	16	8.84	3.33
	J	Group	29	6.38	3.34
Post-tx	None	Individual	42	3.61	3.03
		Group	45	3.72	3.38
	Antidepressant	Individual	50	3.92	3.14
	•	Group	88	3.49	3.30
	Antidepressant	Individual	16	5.94	3.60

Measure	Type of Medication	Individual or Group	N	Mean	Standard Deviation
	and anxiolytic		- 0		
ACI 1		Group	29	4.76	3.54
ASI-social	None	Individual	42	9.76	2.04
Pre-tx	None		42 45	8.76	3.04
	A mti dammagaamt	Group	43 50	9.09	3.34
	Antidepressant	Individual		7.70 9.09	2.72 3.11
	A mti damma a a a mt	Group Individual	88		
	Antidepressant and anxiolytic	maividuai	16	10.19	2.37
		Group	29	9.00	3.27
Post-tx	None	Individual	42	6.50	3.65
		Group	45	6.62	3.41
	Antidepressant	Individual	50	5.92	2.98
		Group	88	6.44	3.48
	Antidepressant and anxiolytic	Individual	16	7.88	3.22
	,	Group	29	7.48	3.70
ACS-anger		1			
Pre-tx	None	Individual	42	3.71	1.16
		Group	45	4.19	.87
	Antidepressant	Individual	50	3.95	.81
	•	Group	88	4.23	.97
	Antidepressant and anxiolytic	Individual	16	3.93	.92
	j	Group	29	4.18	1.23
Post-tx	None	Individual	42	3.17	.92
		Group	45	3.83	.90
	Antidepressant	Individual	50	3.51	.88
	PP	Group	88	3.86	1.07
	Antidepressant and anxiolytic	Individual	16	3.64	1.02
	and anxiotytic	Group	29	3.90	1.04
ACS-positive		Group	2)	3.70	1.01
Pre-tx	None	Individual	42	3.09	.99
TIC tx	TVOILE	Group	45	3.59	1.05
	Antidepressant	Individual	50	3.53	.74
	rintiacpressant	Group	88	3.61	.87
	Antidepressant	Individual	16	3.50	1.24
	and anxiolytic				
D4 4	Mana	Group	29	2.97	.80
Post-tx	None	Individual	42	2.90	.79
	A	Group	45	3.49	1.01
	Antidepressant	Individual	50	3.18	.96
	A4: 4	Group	88	3.33	.97
	Antidepressant and anxiolytic	Individual	16	3.21	1.06
		Group	29	3.12	.71

Measure	Type of Medication	Individual	N	Mean	Standard Deviation
ACS-	Medication	or Group			Deviation
depression Pre-tx	None	Individual	42	4.22	1.26
Pie-tx	None		42	4.22	1.20
	A4: .1	Group			
	Antidepressant	Individual	50	4.50	1.05
	A .:1	Group	88	4.90	1.00
	Antidepressant and anxiolytic	Individual	16	4.62	1.26
		Group	29	4.84	.98
Post-tx	None	Individual	42	3.33	1.09
		Group	45	4.07	1.03
	Antidepressant	Individual	50	3.75	1.24
	_	Group	88	4.08	1.14
	Antidepressant and anxiolytic	Individual	16	4.13	1.51
	,	Group	29	4.45	1.26
ACS-anxiety		··r			
Pre-tx	None	Individual	42	4.17	.90
		Group	45	4.39	.79
	Antidepressant	Individual	50	4.33	.93
	1	Group	88	4.48	.75
	Antidepressant and anxiolytic	Individual	16	4.98	.77
	and anxiorytic	Group	29	4.30	.78
Post-tx	None	Individual	42	3.34	.86
r ost-tx	None	Group	45	3.76	.66
	Antidanraggant	Individual	50	3.70	.84
	Antidepressant		88	3.80	.81
	Antidonnagant	Group			
	Antidepressant and anxiolytic	Individual	16	3.98	1.09
		Group	29	3.99	.93

Clinically significant change has been put forward as a more informative indicator of clinical utility than statistically significant differences in determining the difference between group averages. The analyses allow for patient outcomes from treatment to be reported as recovered, no change, or deteriorated. This is based on the theoretical conceptualisation that a treatment can be considered as moving a person's score on a measure for the condition from a point on a distribution of a dysfunctional population to a point on a distribution of a functional population.

Clinical significance of the differences seen in the previous analysis was determined according to the procedure outlined by Jacobson and Truax (1991) and

Jacobson and colleagues (1999). Patients' scores for each treatment type were classified as deteriorated, unchanged or improved and expressed as a percentage of the group (see Table 20).

Three methods have been described for determining clinically significant change (Jacobson & Truax, 1991). The choice of method depends upon whether norms are available for distressed and non-distressed populations on the outcome measure. Such norms exist for the ASI and for the ACS; hence, clinically significant change was defined as scores within two standard deviations of the non-distressed population mean for these measures. The number and percentage of patients attaining these cut-off values are also shown in Table 20. A proportion of patients also recorded pre-treatment scores that were below the clinical cut-off scores. That is, prior to CBT treatment, their scores on the measures were less than the cut-off score obtained from a non-clinical population. These proportions are also reported in Table 20.

Table 20

Number (Percentage) of Patients who Deteriorated, Remained Unchanged, Improved, or Achieved Clinically Significant Change at Post-treatment

Outcome	Change	Medicated		Not		χ^2	p
variable		n	(%) medic		cated		
				n	(%)		
ASI							
	Deteriorated	4	(1)	1	(1)	.27	.60
	Unchanged	175	(39)	58	(29)	5.70	.02
	Improved	111	(24)	58	(29)	1.42	.23
	Clinically significant	93	(84)	52	(90)	2.36	.12
	improvement						
	Pre-tx score less than clinical	162	(36)	83	(41)	1.89	.17
	cut-off						
ACS							
	Deteriorated	3	(2)	3	(3)	1.11	.29
	Unchanged	135	(70)	56	(67)	.36	.55
	Improved	50	(26)	20	(24)	.15	.70
	Clinically significant	13	(26)	3	(15)	1.10	.30
	improvement						
	Pre-tx score less than clinical	4	(2)	5	(6)	2.77	.10
	cut-off						

The results showed that using the ASI as the outcome measure, 24% of those taking medication and receiving CBT achieved reliable improvement compared with 29% of those receiving CBT alone. Of those patients who improved, 84% of those

receiving medication and CBT achieved clinically significant change and 90% of those receiving CBT alone achieved clinically significant change. For patients receiving medication and CBT, 39% remained unchanged compared with 29% of those receiving CBT alone.

In considering the results using the ACS as the outcome measure, similar proportions achieved improved scores (26% for those receiving medication and CBT and 24% for those receiving CBT alone). Of those patients who improved, a higher proportion of those receiving medication and CBT achieved clinically significant change compared with those receiving CBT alone (26% and 15% respectively), although the numbers in each group were relatively small. Greater proportions of patients remained unchanged at post-treatment with 70% of those receiving medication and CBT and 67% of those receiving CBT alone indicating that the change in their scores was not clinically significant.

Differences between the proportions of those who deteriorated, remain unchanged or improved were tested using a chi-square test for independence for each of the outcome measures. For both the ASI and the ACS, the overall test failed to reach significance indicating that the proportion of patients deteriorating, not changing, improving, attaining clinical significant change, or being below post-treatment cut-off at pre-treatment were not significantly different between medicated and undedicated groups. For each category of change, only the proportion of patients who remained unchanged using the ASI as the fear of emotion measure was significant ($\chi^2(1, N = 652) = 5.70$, p = .02), showing that a greater proportion of those who were medicated remained unchanged compared with the proportion who were not medicated.

Chapter 5 Discussion for Study 1

The purpose of this study was to investigate fear of emotion in a sample of anxious and depressed patients, and how it changes after treatment with CBT alone or combined therapy (CBT with naturalistic pharmacotherapy). To do this, fear of emotion was measured using two different questionnaires, assessed across psychological disorders, and was evaluated after treatment by examining statistically significant and clinically meaningful differences.

Measuring Fear of Emotion

The Anxiety Sensitivity Index (ASI) and the Affective Control Scale (ACS) were both developed to assess the fearful response an individual may have to their emotions. Although originally being developed for panic disorder, both have been used to assess fear of emotion in patients with depression and a range of anxiety disorders. As they were developed to assess similar constructs, a strong correlation between these measures would be expected. Indeed, the results of this study showed that the total scale scores from these two measures were moderately correlated.

Within each of the measures, the subscale scores were strongly correlated with the total scale scores, and moderately to strongly associated with each other, consistent with published psychometric validity studies (Zinbarg, Barlow, & Brown, 1997; Rodriguez et al., 2004; Berg, Shapiro, Chambless, & Ahrens, 1998).

Across measures, there was a moderately strong to strong association between the anxiety subscale of the ACS (ACS-anxiety) and the ASI scale and subscales. This was also expected as both scales were developed to initially assess various responses to the experience of anxiety. Total scores on the ACS (ACS-total) were also moderately to strongly correlated with two ASI subscales, Mental Concerns and Physical Concerns. The depression subscale of the ACS (ACS-depression) was also moderately correlated with the Mental Concerns subscale of the ASI (ASI-mental). This considerable overlap

between the ACS and the ASI and its subscales suggests that while the ACS may offer the prospect of assessing fear of specific emotions, a broader fear of emotion construct appears to capture an individual's response to any negative emotion. As the ACS is a 42 item measure and the original and most recent revision of the ASI has far fewer items (the ASI comprises 16 items and the ASI-3 comprises 18 items), then the marginal benefits of the ACS appear to be offset by the overlap with the ASI and the user-friendliness of fewer items. Correspondingly, despite the development of the ASI as a measure to assess responses to anxiety symptoms, the current data show a much broader application of the measure to other negative emotions. The current study did not assess whether patients also presented with specific concerns about anger or positive emotions and, as such, these subscales of the ACS may demonstrate a more thorough assessment of fear of emotion.

While there is a degree of overlap between the two measures, it is not possible from the current study to determine if both measures are capturing the same construct. To formally answer that question, a more robust series of construct validation studies needs to be carried out.

Fear of Emotion in Anxious and Depressed Patients

Fear of emotion is a cognitive construct that was borne out of research to identify predisposing and maintaining factors of panic disorder, and indeed remains a core factor in current cognitive models of panic. Accumulating evidence from subsequent research has shown fear of emotion scores to be elevated in depressive and anxiety disorders. As such, it was expected in the current study that patients with a primary diagnosis of an anxiety or depressive disorder presenting for treatment at a community mental health clinic would have elevated scores compared with previously published norms on fear of emotion measures. Results from this study showed elevated scores on both the ASI and ACS, and these elevated scores were of similar magnitude

or higher than those reviewed in Chapter 2, which is most likely a reflection of the high degree of comorbidity in the clinical sample.

Results of regression analyses showed that fear of emotion scores were a weak to moderate predictor of symptoms of anxiety and depression, accounting for 34.4% and 29.8% of variance in pre-treatment scores, a finding entirely consistent with extant literature (e.g., Taylor et al., 1992, Rodriguez et al., 2004, Rector et al., 2007, Naragon-Gainey, 2010).

The development of the Affective Control Scale (ACS) was an attempt to expand the concept of fear of anxiety to other emotions. The development of the subscales of the ACS was designed to assess fear of depression, anger and positive emotion, along with fear of anxiety. The current study investigated the specificity of these subscales in predicting symptoms of anxiety and depression in patients with an anxiety or mood disorder.

Results of regression analyses showed that the subscale scores explained a higher proportion of variance in symptom scores than total scale scores (37.2% as against 34.4% for anxiety symptoms, and 38.6% as against 29.8% for depression symptoms), showing some support for the specificity of the subscales. Examination of the ACS subscales showed that, as expected (Roemer et al., 2005), the ACS-anxiety subscale was the only unique predictor of anxiety symptoms. The ACS-depression subscales also uniquely predicted depression symptoms (Liverant et al., 2008). The ACS-anger subscale also uniquely predicted depression symptoms, which suggests that these subscale items may not differentiate depressive irritability from anger more generally. When examining differences in fear of emotion scores across primary diagnoses, patients with a primary diagnosis of a depressive disorder reported higher scores on the ACS-depression subscale; a finding consistent with previously shown association between depression symptom scores and the subscale (Liverant, Brown,

Barlow, & Roemer, 2008). While this suggests some specificity of the subscale, patients with a primary diagnosis of a depressive disorder also reported higher scores on the ACS-positive subscale compared with patients diagnosed with generalized anxiety disorder.

In consideration of the ASI subscales, the ASI Physical Concerns subscale was found to be a unique predictor of anxiety symptoms, consistent with the earlier reviewed studies (e.g., Zinbarg, Barlow, & Brown, 1997). This is also consistent with cognitive theories of anxiety disorders where attention and reaction to the physical symptoms of anxiety are consistently included (Barlow, 1988; Rapee & Heimberg, 1997; Taylor, 2003). It is possible this finding is confounded by the construction of the Physical Concerns subscale which consists of eight of the 16 items of the ASI (Taylor, et al., 2007).

However, findings from the current study did not support the specificity of subscales with regard to depressive symptoms. While the ASI Mental Concerns was a unique predictor of depression symptoms, it also uniquely predicted anxiety symptoms. This is consistent with findings from studies where patients with a diagnosis of Major Depressive Disorder with or without comorbid anxiety disorders scored highly on the ASI Mental Concerns subscale, along with patients with anxiety disorders (Rodriguez et al., 2004; Rector et al., 2007). Patients with a primary diagnosis of social anxiety disorder reported higher scores on the ASI Social Concerns subscale compared with those with a primary diagnosis of panic disorder with or without agoraphobia, generalized anxiety disorder or depression consistent with previous studies (Zinbarg et al., 1997; Rector, Szacun-Shimizu, & Leybman, 2007).

The interaction between diagnosis and medication status for scores on the ASI Mental Concerns subscale was unexpected but could potentially be important. For example, it is tempting to speculate that the alarming symptoms of panic disorder may

prompt these patients to attend their medical practitioner, who would probably prescribe medication and refer them for psychological intervention (hence those with higher ASI Mental Concerns subscale scores will be medicated). In patients with generalized anxiety disorder, their worries may include whether medication is going to be a problem (e.g. dependence, adverse effects) so they may choose not to take medication (hence those with higher ASI Mental Concern subscale scores will be unmedicated).

There has been ongoing debate about the validity of the ASI subscales, as they were initially derived from post-hoc factor analysis as opposed to being theoretically constructed (e.g., Zinbarg, Mohlman & Hong, 1999). This has led to revisions of the measure, and adding items to increase the reliability and validity of the subscales. The most recent revision, Anxiety Sensitivity-3 (ASI-3; Taylor, Zvolensky, Cox, Deacon, Heimberg, Ledley, et al., 2007) has been shown to have better internal consistency and a robust factor and dimensional structure (Broman-Fulks, Deacon, Olatunji, Bondy, Abramowitz, & Tolin, 2010) than earlier versions of the ASI. A further study using this instrument replicating the current study would clarify questions of subscale specificity.

In summary, in patients presenting for treatment, fear of emotion is associated with depressive and anxiety disorders, with total scores on fear of emotion measures being weak to moderate predictors of anxiety and depressive symptoms. This strongly suggests that fear of emotion plays a role in many emotional disorders, and not just panic disorder. The subscales of both fear of emotion measures account for a small increase in total variance and, in the case of the ACS, show some specificity for predicting anxiety and depression symptom scores from the anxiety and depression subscales respectively.

Changes in Fear of Emotion after Treatment

Despite fear of emotion being initially proposed as a dispositional tendency, early research showed that it was amenable to treatment, either indirectly when

measured during treatment for psychological disorders, or directly (e.g., Otto & Reilly-Harrington, 1999; Smits et al., 2008). Fear of emotion scores decreased in patients treated with efficacious treatments such as CBT or various pharmacotherapies (e.g., Romano et al., 2004; Simon et al., 2004). The change in fear of emotion scores for patients receiving both types of therapy simultaneously is not as clear.

In the current study, fear of emotion scores decreased from pre-treatment to post-treatment on both measures. For the ASI, mean (SD) scores decreased from 29.13 (13.03) to 18.56 (12.06) with a large effect size. The analyses also showed a small effect size for medication, such that those patients who were unmedicated and underwent CBT therapy had lower scores both before and after treatment than those medicated patients who underwent CBT therapy, although the difference in the mean scores failed to reach significance (p=.06).

These results are consistent with reported studies on other clinical samples. In the meta-analyses of patients with panic disorder being treated with CBT, ASI scores decreased by 14 points in the first meta-analysis (Otto & Reilly-Harrington, 1999) and from 31.18 (10.29) to 20.28 (9.89) in the second (Smits et al., 2008), a change of 10.9 points. In the study by Smits et al., patients with a diagnosed anxiety disorder or depression showed a reduction from 28.36 (13.50) to 16.71 (11.30), a change of 11.65 points. The results from this study lend further weight to the suggestion that fear of emotion is a transdiagnostic construct, as the aforementioned review and meta-analysis were conducted on treatment seeking samples with anxiety disorders or tinnitus. This study found similar magnitude of change scores in a highly comorbid population.

In those patients who received a combination of pre-existing naturalistic pharmacotherapy along with CBT, scores on the ASI in this study decreased from 29.47 (12.82) to 19.38 (12.30), a change of 10.09 points. The magnitude of this decrease in ASI scores from pre-treatment to post-treatment was similar to that reported in previous

studies (Roy-Byrne et al., 2005; Arch & Craske, 2007). The co-morbid panic disorder patients in the Roy-Byrne study who received CBT and pharmacotherapy according to a clinical algorithm showed change scores from 34.4 to 23.11, a change of 11.29 points. In the Arch and Craske study, ASI scores decreased from 31.71 (9.38) to 17.85 (11.94), a change of 13.86 points in patients undergoing CBT therapy and taking SSRI/SNRI medication; and from 30.54 (11.45) to 19.59 (13.24), a change of 10.95 in patients undergoing CBT and taking any antidepressant medication. Of note in the current study is the much larger sample size of 452 patients, compared with 119 in the Roy-Byrne et al. (2005) study and 27 in the Arch and Craske (2007) study. Given that the patients in the current study presented with a range of primary anxiety disorders and depression, the results support the transdiagnostic nature of fear emotion and the robustness of the change in response to treatment, be it with CBT alone or in combination with pharmacotherapy.

Scores on the ACS also decreased over treatment, consistent with the pattern of reduction in ASI scores. Fear of emotion scores decreased over treatment from a mean of 4.09 (.68) to a mean of 3.56 (.77). This is an important finding given the paucity of research using the ACS in clinical populations. Indeed, only one study was found where changes in ACS scores were reported in clinical samples. In that study (Roemer & Orsillo, 2007), the researchers found that in 16 individuals undertaking a cognitive behavioural therapy, scores decreased from 3.97 (.62) to a mean of 2.94 (.77), a change of 1.03 points. The smaller decrease in change scores in this study may be attributable to the much larger and more diverse sample (276 participants in the current study compared with 16 in the Roemer and Orsillo study). Further, the majority (14) of participants in the Roemer and Orsillo study met criteria for generalized anxiety disorder, and the focus of the CBT administered for that study was to specifically address the emotional avoidance theorised to be a key maintaining factor.

In the current study, for those patients who only undertook CBT treatment, scores decreased from 3.96 (.76) at pre-treatment to 3.48 (.72) at post-treatment, showing a change of .48 points. For those patients undertaking combined therapy, scores decreased from 4.15 (.63) at pre-treatment to 3.65 (.79) at post-treatment, showing a similar change of 0.5 points. Of the 16 participants in the Roemer & Orsillo study, six were taking stable regimens of pharmacotherapy in addition to undertaking CBT, but the results of these six individuals were not reported separately to the entire sample, so a comparison to the current study cannot be made.

The mean ACS-total pre- and post-treatment scores of patients undergoing CBT treatment and not taking medications were lower than those who were; the difference reaching significance and showing a small to moderate effect size. This finding may have several explanations: Patients taking medications may have a higher baseline fear of emotion, leading to their presentation to medical practitioners and resulting in pharmacotherapy. They may also have more severe symptoms of an anxiety or depressive disorder, and therefore be more fearful, the medication they were taking might have been ineffective in reducing their emotional symptoms, thereby leaving them more fearful of those symptoms, or the medication may have increased their fear of emotion. Measuring fear of emotion prior to commencing pharmacotherapy would be helpful to answer these questions.

The results from the ACS data also showed that those patients receiving individual CBT treatment had lower sores than those receiving group treatment, and that those participating in individual therapy showed greater reductions in scores across treatment than those participating in group therapy. The ASI data did not support this finding. These equivocal results are consistent with extant literature showing either no difference between individual and group therapy, or a slight advantage to individual treatment over group treatment.

Changes on the subscale scores. To extend the findings of previous research, the effects of treatment on the subscales of the fear of emotion measures were examined. Over treatment, significant reductions were shown on the composite variable, consistent with reductions seen in total scale scores, also with a large effect size. The follow-up analyses showed that each of the subscale scores decreased over treatment.

With regard to the changes in the ASI subscale scores, the current study showed a different pattern of results compared with previously reported studies. In this study each of the three subscales, ASI-physical, ASI-mental, and ASI-social, all decreased over treatment with either CBT alone or CBT combined with naturalistic pharmacotherapy. This contrasts with the findings of Schmidt et al. (2007) who observed a decrease only in ASI-physical and ASI-social scores after a CBT treatment for fear of emotion in an at-risk grip for anxiety disorders (i.e., those with elevated ASI scores). Olatunji et al. (2008) reported a reduction in ASI-mental and ASI-social scores, but not ASI-physical when treating 38 patients with generalized anxiety disorder with fluoxetine. The current study may show a different pattern of findings due to the higher level of comorbidity in the participants. Olatunji et al. noted that the patients diagnosed with generalized anxiety disorder and comorbid depression in their study reported only mild levels of depressive symptoms. This suggests that more emotional distress is associated with greater fear of emotion generally, and that the physical concerns subscale is associated with depression.

There was no effect for medication, although interestingly two of the ACS subscales, ACS-depression and ACS-anxiety, were significantly associated with medication use. While it is tempting to suggest that future studies investigate medication use on these two subscales specifically to further understand this relationship, there appears to be little theoretical rationale for these particular subscales to be sensitive to medication effects. In practical terms, patients with elevated fear of

anxiety or fear of depression may be more likely to present to their medical practitioner and be prescribed pharmacotherapy, compared with those with fear of anger, and those with a fear of positive emotions may be unlikely to present to their medical practitioner at all.

Also consistent with the data from the total scale scores was a significant difference between those receiving group CBT treatment and those receiving individual CBT, with those receiving individual CBT reporting greater reductions in subscale scores. As none of the remaining interaction terms were significant, there was no difference between those who were medicated and those who were not on the subscales scores, across individual and group treatment.

In summary, these results show that across treatment, measures of fear of emotion decreased for those receiving CBT alone and for those receiving CBT with medication. The results also suggest that there is a difference between those receiving medication and those receiving CBT alone, which may be related to the emotions of depression and anxiety specifically.

Type of medication. This study also investigated the effect of the type of medication on fear of emotion over treatment. The patient sample was stratified into three groups: Those not taking medication, those taking an antidepressant medication (typically SSRIs), and those taking a combination of antidepressant and anxiolytic medication (typically SSRIs with benzodiazepines). As described in the previous section, there was a reduction in scores on both measures of fear of emotion from pretreatment to post-treatment across all conditions. On the ASI measure, patients taking a combination of antidepressant and anxiolytic medication recorded higher scores at preand post-treatment than those taking antidepressant medication alone or not taking any medication. Medication had no effect on ACS scores.

This finding is in contrast to that of Arch and Craske (2007) who found that patients medicated with SSRI antidepressant medication did not show changes in ASI scores over CBT treatment. The current study had many more participants in the antidepressant groups (238 vs 14 in the Arch and Craske (2007) study), which allows for greater power to detect differences. Furthermore, the current study included a range of antidepressant medication, as opposed to SSRIs only in the Arch and Craske (2007) study.

Patients taking an antidepressant combined with a benzodiazepine recorded higher scores before and after CBT treatment, but reported a similar magnitude of change over treatment. This finding is consistent with that of Telch et al. (1993), who found that medication use – including benzodiazepines – made no difference to treatment outcome on a range of measures including fear of emotions (ASI). However, Simon et al. (2004), who reported that patients taking a combination of an antidepressant with a benzodiazepine showed a smaller magnitude of reduction in fear of emotion scores than those taking the antidepressant alone or taking the antidepressant with a reducing dose of benzodiazepine. Westra, Stewart and Conrad (2002) reported that use of benzodiazepines 'as required' produced less change in ASI scores than in those patients with panic disorder who were not medicated, or those who were taking benzodiazepines regularly. Indeed, those taking benzodiazepines regularly reported outcomes similar to those patients with panic disorder who were unmedicated. Thus, the type of benzodiazepine use appears to be important with regard to changes in fear of emotion.

Clinically significant change. Clinically significant change provides a more clinically meaningful description of change in response to a clinical intervention than statistically significant change. In considering clinical outcomes, the current study showed trends that for patients receiving CBT alone, more showed reliable

improvement on the ASI (29% vs. 24%), more achieved clinically significant change (90% vs. 84%) and fewer remained unchanged (29% vs 39%) compared with those receiving medication and CBT.

On the ACS fear of emotion measure, there was little difference between the patients undergoing CBT treatment and those receiving combined CBT and medication with similar proportions remaining unchanged (67% vs. 70%) and showing reliable improvement (24% vs. 26%). There was a trend for a higher proportion of those receiving combined treatment to achieve clinically significant change (26% vs. 15%), but these groups were very small (13 and three patients respectively). It is important to note that these differences were not significant for either measure. This is not surprising as the standard deviations for this measure were much higher than the ASI, requiring large changes in scores for a patient to move from a clinical distribution to a healthy distribution.

Limitations

The primary context for this study was examining fear of emotion with two measures in a community sample of mental health patients with anxiety disorders and/or depression being treated with CBT with and without pharmacotherapy. While this allows for greater generalisability of the findings than for highly structured studies, the findings of this study are subject to several limitations.

Neither of the two types of treatment can be considered to be optimised in that no treatment fidelity procedures were instituted. Nevertheless, it is likely that the CBT treatment provided was consistent given that it was guided by treatment manuals and was carried out by a small number of clinicians working at the same clinic, and who were all experienced in carrying out the interventions and who participated in clinical supervision with a senior clinician.

Another limitation was that the pharmacotherapy used in the combined treatment group was recorded based on patients' self-report. While patients were prompted during clinical interview to disclose each of their medications, dosages and duration of medication use, no data was collected from the prescribers, and no objective measure of medication use (e.g. blood assays, dose counting) was obtained.

Furthermore, patients' adherence to their prescribed therapy was not monitored.

However, as effectiveness studies have shown high correlation between patients' self-report and pharmacy records (Katon et al., 1996; Katon et al., 1999), it is likely that the medication effects reported here were fairly accurate.

For patients taking anxiolytic medications, the use of 'prn' or 'as required' dosing was not established. Since this type of dosing has been shown to be problematic (Westra, Stewart & Conrad, 2002), it is possible that the higher scores in this group were attributable to the nature of the dosing of the benzodiazepines as opposed to the administration of the combination of benzodiazepines with antidepressant medication.

As no restrictions were placed on prescribing antidepressant and anxiolytic medication, it is not clear which protocols were used by the various prescribers. Indeed, almost half of the patients receiving pharmacotherapy were dosed outside Australian prescribing guidelines (National Prescribing Service Limited, 2004). Finally, many of the participants in this study were taking other drugs for a variety of conditions.

Inspection of the patient files revealed a range of medical conditions ranging from coronary bypass surgery, hypothyroidism, sleep disorders, osteoarthritis, ulcerative colitis and oesophageal reflux. It is also likely that patients did not offer information they did not see as relevant to their application for treatment to a community mental health service. Therefore, some patients may have taken drugs that interacted with their antidepressant medication, potentially altering its effectiveness.

Certain elements of the research design also limit the interpretation of the findings. In comparing the impact of CBT and pharmacotherapy on fear of emotion, patients were not randomly assigned to treatment groups. Thus, selection biases cannot be ruled out. For example, those patients who were medicated may have had more severe psychopathology which had not responded adequately to pharmacotherapy, prompting their referral for additional psychological therapy. However, the two groups did not differ markedly on measures of severity or duration of symptoms prior to undergoing CBT treatment. Patients were also not 'blind' as to which treatment they were receiving. The research design also did not include a control group so it was not possible to determine if either treatment (medication with CBT or CBT alone) accounted for the reductions in fear of emotion scores over time. However, the reviewed literature for this study strongly suggests that the changes in fear of emotion were due to the treatments as implemented in this study.

To answer the questions posed in this study, a number of statistical analyses were performed on the same data set. Except where stated, alpha protection was not used to reduce the risk of Type 1 'family-wise' errors. Therefore it possible that the significant findings reported were spurious. However, it is arguable that most of the analyses were not 'family-wise' as they were testing theoretically distinct hypotheses (O'Keefe, 2003).

Finally, this study used the original 16-item version of the ASI as one of the measures of fear of emotion. The most recent version of this measure, the ASI-3, has improved psychometric properties particularly with regard to the three subscales and should be used in future studies.

Conclusions

Fear of emotion can be measured with the ASI or the ACS. The two scales are moderately correlated and provide some discriminant validity in those patients with

depression or anxiety symptoms. Fear of emotion is associated with anxiety and depressive disorders and can therefore be considered a transdiagnostic construct. Fear of emotion appears to respond to treatment with CBT alone or combined with pharmacotherapy. While the decrease in fear of emotion was statistically significant, only a small proportion of patients achieved clinically significant change.

Chapter 6 Introduction to Study 2

In the previous study, fear of emotion was shown to be a cognitive construct associated with anxiety and depressive symptoms in patients with anxiety and depressive disorders presenting for treatment at a community mental health centre.

Further, these elevated scores decreased over the course of CBT treatment, regardless of whether the patients were concurrently being treated with naturalistic pharmacotherapy.

Given that both CBT and pharmacotherapy are effective treatments for treating anxiety and depressive disorders, it was expected that fear of emotion scores would decrease following treatment, and this study confirmed that combining pharmacotherapy with CBT did not disrupt outcomes.

Cognitive models of panic disorder posit that long-term outcome will be dependent on patients being less fearful of the consequences of experiencing anxious emotion (Clark et al., 1994). If this is the case in panic disorder, then it is possibly also the case in other disorders given that fear of emotion is elevated in patients with most anxiety and depressive disorders (as per introduction to Study 1). That is, as fear of emotion contributes to the maintenance of these disorders, elevated fear of emotion at the completion of treatment may be an ongoing vulnerability for individuals with anxiety and depression, and elevated scores on fear of emotion measures may increase risk of relapse in those patients.

As with most early work on fear of emotion, preliminary studies investigating the relationship between fear of emotion after treatment and continued symptom improvement at follow-up focused on patients with panic disorder. More recent studies have extended this to other anxiety disorders and unipolar depression.

Panic disorder. In 63 patients with a primary diagnosis of panic disorder, fear of emotion scores (as measured with the ASI) after a 12-week program of CBT predicted anxiety symptoms, panic intensity, anticipatory anxiety and avoidance of

physical sensations associated with panic at six-month follow-up (Schmidt and Bates, 2003). A naturalistic prospective study, investigating the factors that maintain panic disorder, showed that fear of emotion (as measured by the ASI) predicted panic attacks at one-year follow-up (Ehlers, 1995). Of the 169 patients this study, 22 had undergone treatment for panic disorder with either medication or psychotherapy.

In contrast, in examining relapse in patients with panic disorder with agoraphobia following successful treatment with pharmacotherapy, Mavisskalan and Guo (2004) found that fear of emotions did not predict relapse, but general fearfulness did, along with anxiety symptoms and disability in work or family settings.

Any anxiety disorder. In a large study of 429 individuals, fear of emotion (as measured by the ASI), along with disability (impairment of functioning) each independently predicted relapse after two years in a group presenting with panic disorder with or without agoraphobia, agoraphobia alone, social anxiety disorder, generalized anxiety disorder or a combination of these anxiety disorders (Scholten et al., 2013).

A prospective study in the primary care setting (Taylor, Jakubovski & Bloch, 2015) showed that in patients meeting diagnostic criteria for an anxiety disorder (generalized anxiety disorder, social anxiety disorder, panic disorder, or posttraumatic stress disorder), recurrence of symptoms was predicted by post-treatment scores on a fear of emotion measure (ASI), following treatment with pharmacotherapy and/or cognitive behaviour therapy.

Similarly, Boswell et al. (2013) investigated fear of emotion in a sample of 37 treatment-seeking patients with diagnoses of panic disorder, social anxiety disorder, generalized anxiety disorder, obsessive-compulsive disorder, posttraumatic stress disorder and anxiety disorder not otherwise specified. Reduction in fear of emotion during treatment with a transdiagnostic CBT protocol was associated with lower scores

on measures of symptom severity at six-month follow-up. Approximately half of the participants were stable on psychotropic medication.

Depression. Elevated fear of emotion scores have also been hypothesized to be associated with increased vulnerability to further depressive episodes. In examination of the so called "scar effect", Cassin and Rector (2012) examined fear of emotion scores (specifically the Mental Concerns subscale of the ASI) in a group of patients with a primary anxiety disorder presenting for treatment. They found that fear of emotion scores were elevated in those with concurrent secondary depression and those with a history of depression compared with those who had never been depressed. These findings provided preliminary support for models of relapse suggesting that, once an individual has experienced a depressive episode, they remain fearful of further episodes, despite experiencing a reduction of depressive symptoms with treatment.

Response to Treatment

These studies provide support for the importance of fear of emotion to remain stable, or to reduce further, at follow-up following successful treatment. What is less clear is the role of the type of treatment in maintaining the improvements in measures of fear of emotion and symptoms of anxiety and depression at follow-up. As indicated in the above studies, most patients had undergone CBT, some pharmacotherapy and some combined CBT and pharmacotherapy.

As elucidated in Chapter 2, CBT, and other psychological therapies that promote exposure to emotion, may be more effective than pharmacotherapy over the longer term as they typically teach a range of techniques and strategies to patients to enable them to manage their distressing emotions as they arise in the future (Taylor et al., 2003). That is, exposure to emotion and learning skills to manage these emotions leads to a reduction in fear of emotions.

The medication status of people undertaking CBT treatment may influence the effectiveness of treatment gains over the longer term. Patients undergoing pharmacotherapy in addition to CBT may not benefit in the same way. As also previously discussed, pharmacotherapy could interfere with the cognitive variables, such as catastrophic beliefs, self-efficacy, hypervigilance, and learning and memory, and these effects may become more evident over time due to continued inhibition of learning-based mechanisms (Hollon, Stewart & Strunk, 2006). Additionally, gains from CBT treatment may also be undermined by creating state-dependent learning in the presence of antidepressant or anxiolytic medication (Craske and Mystkowski, 2006), or even as a safety behaviour; i.e., taking medication prevents exposure to the feared outcomes and this interferes with the ability to learn that the feared outcome is tolerable.

A literature search was carried out to identify relevant studies. A similar search strategy was used as for Study 1, with the difference being that studies were selected only if at least pre-treatment and follow-up data were reported on the measures of interest. The databases were searched for relevant studies published up until 4th February 2017.

Effect of CBT with or without medication on fear of emotion following treatment. Few studies have reported on the ongoing effect of CBT on fear of emotion following treatment. Table 21 summarises seven studies that report on fear of emotion scores as measured by the Anxiety Sensitivity Index before and after treatment and at a follow-up period. Similarly, Table 22 summarises two studies measuring fear of emotion using the Affective Control Scale. The participants in these studies all met criteria for an anxiety disorder.

Table 21
Summary and Features of Studies Investigating the Effect of Treatment on Fear of Emotion as Measured by Anxiety Sensitivity Index.

Study	Treatment	nent Number of Patients Medicated/ Unmedicated	Primary Diagnosis	Period of Follow-up (Months)				Comments	
					Pre-treatment	Post-treatment	Follow-up		
Telch et al., (1993).	CBT Group	19/15	Panic Disorder with or without Agoraphobia	6	33.74 (11.15)	13.94 (8.52)	14.23 (10.15)	No significant difference between those who were medicated and those who were not (separate data were not reported)	
Marchand et al.,	CBT Group	18/12	Panic Disorder with	12	37.1 (8.9)	18.4 (13.2)	15.1 (11.5)	No significant difference between	
(2009).	CBT Brief	13/11	Agoraphobia	Agoraphobia		30.3 (10.1)	21.9 (12.4)	16.3 (10.0)	those who were medicated and those
	CBT Standard	16/14			33.3 (11.5)	20.5 (13.2)	15.0 (10.6)	who were not at post-treatment and follow-up (separate data were not reported)	
Craske et al. (2007)	CBT for Panic Disorder CBT for PD and	9/14	Panic Disorder with or without Agoraphobia	6	31.6 (10.7)	17.3 (6.8)	16.2 (10.6)	No analyses reported on differences between those who	
	comorbidity 12/10	12/10			31.6 (10.4)	21.8 (15.2)	17.4 (14.8)	were medicated and those who were not	

Study	Treatment		Primary Diagnosis	Period of Follow-up (Months)	Mean Scores (Standard Deviation)			Comments
					Pre-treatment	Post-treatment	Follow-up	
Hedman et al. (2010)	CBT Group	8/16	Health Anxiety	6	27.88 (14.48)	17.92 (10.87)	13.42 (9.15)	Authors did not comment on differences, if any, between those who were medicated and those who were not
Arch et al. (2012)	CBT	36/35	Various Anxiety Disorders	6	27.60 (11.81)	18.68 (11.16)	20.47 (12.90)	Authors did not comment on differences, if any, between those who were medicated and those who were not
Roy-Byrne et al. (2005)	Pharmacotherapy	113	Panic Disorder	6	32.3	28.94	26.52	Standard deviation data were not
(2000)	CBT +	110			34.3	23.11	20.16	reported
	Pharmacotherapy	119						
Arch & Craske (2007)	CBT + SSRI CBT only	14 71	Panic Disorder with or without Agoraphobia	6	31.71 (9.38) 32.56 (12.23)	17.85 (11.94) 17.66 (11.19)	21.50 (11.94) 16.92 (12.38)	Pre-treatment use of SSRI resulted in smaller
(=007)	CBT + Antidepressant	37			30.54 (11.45)	19.59 (13.24)	21.32 (12.19)	improvements in fear of emotion
	CBT only 48	48			33.44 (11.63)	16.89 (9.26)	15.96 (11.91)	scores. Authors reported similar non- significant trend for any antidepressant use at pre-treatment

Table 22
Summary and Features of Studies Investigating the Effect of Treatment on Fear of Emotion as Measured by Affective Control Scale.

Study	Treatment	Number of Patients Medicated/ Unmedicated	Primary Period of Diagnosis Follow-up (Months)	Mean Scores (Standard Deviation)			Comments	
					Pre-treatment	Post-treatment	Follow-up	
Treanor et al. (2011)	CBT	9/22	Generalized Anxiety	3	3.84 (.72)	3.47 (.68)	3.10 (.70)	Authors did not comment on
` '			Disorder	9			2.73 (.78)	differences, if any, between those who were medicated and those who were not
Roemer & Orsillo (2007)	СВТ	6/10	Generalized Anxiety Disorder	3	3.97 (.62)	2.94 (.77)	3.31 (.54)	Authors did not comment on differences, if any, between those who were medicated and those who were not

In investigating the efficacy of CBT group treatment for panic disorder, Telch et al. (1993) reported on the impact of treatment at 6-month follow-up in 34 patients with a principal diagnosis of panic disorder with or without agoraphobia, of whom 19 were medicated predominantly with benzodiazepines. Fear of emotion was assessed with the ASI and scores were shown to decrease across treatment and remain stable at follow-up. Mean scores at post-treatment were 13.94 (8.52) and 14.23 (10.15) at follow-up. The authors reported that medicated and non-medicated patients showed no differences in fear of emotion scores.

Marchand et al. (2009) reported on a study of three variants of CBT for treating panic disorder. They found that 'standard' individual CBT, 'standard' group CBT and a brief seven session individual CBT each reduced measures associated with panic disorder, including fear of emotion. In their study follow-up scores were reported for 12 and 24-months post-treatment. For standard CBT, ASI at post-treatment was 20.5 (13.2) and at 12 month follow-up was 15.0 (10.6). For standard group CBT, ASI at post-treatment was 18.4 (13.2) and at 12-month follow-up was 15.1 (11.5), and for brief CBT, ASI at post-treatment was 21.9 (12.4) and at follow-up 16.3 (10.0). In this study, 62% of participants were medicated, with 55% taking an anxiolytic medication, 21% an antidepressant medication, and 14% taking both types of medication. There was no difference between those who were medicated and those who were not at post-treatment or at follow-up.

Craske et al. (2007) compared two variants of CBT treatment for patients with a principal diagnosis of panic disorder with or without agoraphobia. Regardless of treatment variant, fear of emotion scores remained reduced at post-treatment. For those receiving a typical CBT program, fear of emotion scores (as measured with the ASI) at post treatment were 17.3 (6.8) and at follow-up 16.2 (10.6). For the CBT program, which also targeted co-morbid disorders, the post-treatment scores were 21.8 (15.2) and

17.4 (14.8) at follow-up. The follow-up periods in this study varied from either 6 or 12 months. Approximately half of the groups were medicated, and a quarter received other psychotherapy during the time they were in the study. The effect of medication on scores at follow-up was not reported.

In a treatment trial of group CBT for health anxiety in 24 patients (8 of whom were medicated), fear of emotion scores decreased during treatment from 27.88 (14.48) to 17.92 (10.87) at post treatment (Hedman et al., 2010). At 6-month follow-up, these scores significantly decreased further to 13.42 (9.15). As for the previous study, the effect of medication on scores at follow-up was not reported.

Stability in fear of emotion scores between post-treatment and follow-up was also seen in a study with patients presenting with a range of anxiety disorders (Arch et al., 2012). Following a CBT treatment, participants reported a mean ASI score of 17.63 (10.87) and a mean score of 18.72 (8.96) at 6 months follow-up. Just under half of the patients (48%) were medicated although the effect on scores at follow-up was not reported.

Effect of combined therapy on fear of emotion following treatment. In the Roy-Byrne et al. (2005) study comparing combined therapy (CBT with an algorithm of prescribed pharmacotherapy) to treatment as usual (naturalistic pharmacotherapy) for patients with panic disorder, fear of emotion, as assessed with ASI, decreased over treatment and continued to decrease or remained stable over a follow-up period. The pharmacotherapy component was an algorithm prepared by a psychiatrist and involved the use of an SSRI for at least six weeks, unless previous attempts with that class of medication had been unsuccessful, in which case other classes of antidepressant medication were used with adjunctive medications, such as benzodiazepines, if necessary. For the patients receiving combined therapy, mean pre-treatment scores of 34.4 decreased to 23.11 at three-month follow-up and 21.63 at six-month follow-up.

The scores at follow-up were significantly lower than the scores of fear of emotion for the group treated with pharmacotherapy alone, which changed from a mean of 32.3 at pre-treatment to a mean of 28.94 at 3 months, and 27.73 at 6 months.

Arch and Craske (2007) reported on change in fear of emotion (as measured by the ASI) over time in patients with panic disorder being treated with CBT and pharmacotherapy. In their study, results were reported for different medication classes. For patients taking SSRI medication, ASI scores at post treatment were 17.85 (11.94) and 21.50 (11.94) at 6-month follow-up. For those not taking SSRIs, post treatment scores on the ASI were 17.66 (11.19) at post-treatment and 16.92 (12.38) at follow-up. This difference approached significance (p=.08) controlling for pre-treatment ASI scores. For those taking other antidepressant medication (i.e. an antidepressant that was not an SSRI), mean ASI scores at post-treatment were 19.59 (13.24) and at six-month follow-up were 21.32 (12.19). For those not taking antidepressant medication, post-treatment scores were 16.89 (9.26) and at six-month follow-up were 15.96 (11.91).

Two studies have also reported on the ongoing benefits of CBT on fear of emotion using the ACS as the measure. Treanor et al. (2011) investigated an enhanced CBT for generalized anxiety disorder featuring components of mindfulness-based therapies. They found that CBT produced a decrease in ACS scores over treatment, which further decreased over three and 9 months follow-up. Total ACS scores at post-treatment were 3.47 (.68), 3.10 (.70) at three-month follow-up, and 2.73 (.78) at 9-month follow-up. In this study nine of the 31 participants were taking psychotropic medication, but the effect of medication on scores at follow-up was not reported. Roemer and Orsillo (2007) also reported that the reduction in fear of emotion during treatment was sustained at follow-up. In their study of 16 patients with a primary diagnosis of generalized anxiety disorder, fear of emotions (as measured by total score on the ACS) was 2.94 (.77) at post-treatment and increased to 3.31 (0.54) at follow-up,

but both scores were significantly lower than pre-treatment scores. Six clients were taking antidepressant or anxiolytic medication or a combination of both. The effect of medication on scores at follow-up was not reported.

As summarised in Tables 18 and 19, fear of emotion decreased over treatment and was maintained at follow-up across each of the studies. With regard to the differences between treatments, two studies (Telch et al., 1993; Marchand et al., 2009) found no differences between those who received CBT and those who received combined therapy (CBT and medication), one study (Roy-Byrne et al., 2005) found that combined therapy was superior to medication alone, and another study (Arch & Craske, 2007) found that CBT was superior to combined therapy (CBT with an SSRI or CBT with any antidepressant medication). The remaining five studies did not report separate data for those who received CBT alone and those who received combined therapy.

Summary

Fear of emotion is theorised to be a risk factor for relapse in individuals with mood and anxiety disorders. A developing literature has found this to be the case, with lower fear of emotion scores at follow-up being associated with lower scores on measures of problematic symptoms. It is important that effective treatment for fear of emotion continues to have a lasting effect to help reduce the likelihood of relapse.

Research shows that CBT provides lasting reductions in scores of fear of emotion at follow-up assessment. The effect of medication on fear of emotion may lead to different outcomes at follow-up. These findings are less clear as there are very few studies reporting specifically on the outcomes of patients receiving CBT alone compared with those receiving CBT combined with pharmacotherapy.

Thus, the aim of this study was to determine whether fear of emotion was associated with symptoms of anxiety and depression at follow-up and whether individuals undergoing pharmacotherapy in addition to CBT reported different

outcomes to those undergoing a program of CBT alone in patients presenting with depression or anxiety disorder in a community mental health centre.

This study addressed two questions: Firstly, is fear of emotion at post-treatment associated with symptom severity at six-month follow-up; and secondly, does fear of emotion decrease more over time in patients who received CBT alone than those who received naturalistic pharmacotherapy in addition to CBT.

Chapter 7 Method for Study 2

Participants

One hundred and eight adults who were offered, and had accepted, treatment at the Centre for Clinical Interventions (CCI) in Perth, Western Australia, between March 2008 and April 2010 participated in this study. The protocol for acceptance of referrals and assessment was the same as for those participants described in Chapter 3, as were the inclusion and exclusion criteria. The patients provided consent (as for Study 1) and additional consent was obtained to contact these patients six months after completing treatment. A sample of this additional consent form is provided in Appendix A.

Seventy-seven of the 108 patients completed the transdiagnostic group treatment program, which was defined as completing 5 sessions or more, and were invited to complete a further follow-up questionnaire assessment package. The assessment package contained four self-report measures, and a brief questionnaire ascertaining any treatment they had undertaken since completing treatment at CCI and any changes to their medication regimen, be that addition of new medication, changes to existing medication, or ceasing pharmacotherapy (see Appendix B). Forty-one patients completed and returned the package, representing a response rate of 53%. Of these, 28 had been diagnosed with a depressive disorder (depressive episode, recurrent depressive disorder, or dysthymia), five with generalized anxiety disorder, three with social anxiety disorder, one with panic disorder with agoraphobia, one with simple phobia and three with mixed anxiety-depressive disorder. The majority (83%) were diagnosed with more than one psychological disorder, with depressive disorders being the most common (47.0%), followed by generalised anxiety disorder (17.6%), social anxiety disorder (14.7%) and panic disorder (8.8%). The mean time since onset of the current problems was 12 years and 10 months. Most (92.6%) had previously receives psychological or psychiatric treatment and about a third (34.1%) had previously been hospitalised for

psychological problems. Twenty-eight patients were taking antidepressant or anxiolytic medication.

Measures

Symptom measures. Symptom measures for depression (Beck Depression Inventory Revised (BDI-II); Beck, Steer, & Brown, 1996) and anxiety (Beck Anxiety Inventory (BAI); Beck, Epstein, Brown, & Steer, 1988) were as for Study 1 (see Chapter 3, Method).

Fear of emotion measures. Fear of emotion was assessed with the Anxiety Sensitivity Index (ASI; Reiss, Peterson, Gursky, & McNally, 1986) and the Affective Control Scale (ACS; Williams, Chambless, & Ahrens, 1997). A description of these measures and their properties was included in Study 1 (see Chapter 3, Method).

Procedure

Patients underwent assessment as part of their treatment at CCI, as detailed in Chapter 3. In brief, at an assessment interview, they were screened with a diagnostic structured interview and completed an intake questionnaire providing demographic and biographical details and a number of questionnaires including the symptom measures and fear of emotion measures. If patients met the service's inclusion criteria, they were offered a place in the group treatment program (the Mood Management Course) if their primary presenting problem was depression or anxiety, and if they were able to attend the treatment program (i.e., able to commit to attending each of the 10 sessions and completing between session tasks). The patients attended one of 12 manualised Mood Management Courses (Nathan, Rees, & Smith, 2001) consisting of 10 two-hour weekly sessions, and a follow-up session after one month. These were facilitated by at least one clinical psychologist and a post-graduate clinical psychology trainee. At the completion of the program, patients were provided with a copy of the study information letter to remind them of the follow-up assessment.

Six months after completing the therapy program, patients were sent an assessment package containing a cover letter (see Appendix C) and a further copy of the information letter, the therapy questionnaire and the four symptom and fear of emotion measures. If the questionnaires were not returned after two weeks a reminder letter was sent (Appendix D). If the questionnaires had not been returned after another two weeks, a telephone call was made to encourage completion and to check on progress. Once the questionnaires were received, a feedback letter was provided to the patient (see samples in Appendix E). The letter sent differed depending on whether the patient had maintained gains made in treatment or whether they had deteriorated following treatment. Five patients of the 41 patients who returned measures reported deterioration in their scores. For the 28 patients taking a pharmacotherapy regimen, coding was undertaken as for the same criteria in Study 1 for class of medication (e.g. tricyclic antidepressant, selective serotonin re-uptake inhibitor) and dose. Any other treatment received by any participant in the follow-up period was also coded.

Analyses

To answer the questions posed in the rationale for Study 2 a series of analyses were carried out using SPSS version 22. Firstly, to determine whether demographic or clinical characteristics differed between medicated and unmedicated patients on entry to treatment, independent samples t-tests were performed. Two-way between-groups analyses of variance (ANOVA) with post-hoc comparisons using the Tukey HSD test were conducted to determine differences between medicated and unmedicated patients on fear of emotion measures and their subscales according to primary diagnosis.

To determine whether there was a difference in symptoms over time between those who received CBT only and those who received CBT and pharmacotherapy, 2 x 3 mixed design ANOVAs were conducted with medication status at six-month follow-up as the between subjects variable with two levels (medicated and not medicated). The

within subjects variable was time with three levels (pre-treatment, post-treatment and six-month follow-up). The dependent variables were the total scale scores on the BDI and the BAI.

To determine if fear of emotion predicted severity of anxiety and depression symptoms six months after the completion of CBT treatment, two sets of multiple regression analyses were performed. Firstly, multiple regression analyses were performed with depression and anxiety symptom scores (BDI and BAI) at follow-up as the dependent variables and fear of emotion scores on the ASI and ACS at posttreatment as predictor variables. In replicating the analysis by Arch and Craske (2007), pre-treatment symptom scores were also entered into the regression as the first step to control for pre-treatment symptom severity. A second pair of hierarchical multiple regression analyses were performed with symptom scored at follow-up (BDI and BAI) as the dependent variables and fear of emotion scores (ASI and ACS) at post-treatment as predictor variables. In contrast to Arch and Craske (2007), post-treatment symptom scores were entered as the first step to control for symptom severity at the completion of CBT treatment. These regression analyses were repeated to determine if change in fear of emotion scores from pre-treatment to post-treatment predicted severity of anxiety and depression symptoms six months after the completion of CBT treatment. For these analyses, the predictor variables were defined as the change in fear of emotion scores over CBT treatment. These were calculated as the post-treatment score on each fear of emotion measure subtracted from the corresponding pre-treatment score. The same pattern of results was found.

Chapter 8 Results for Study 2

Participant characteristics

The characteristics of the participants are displayed in Table 23. To determine whether demographic or clinical characteristics differed between medicated and unmedicated patients six months after completing CBT treatment, independent samples t-tests were performed on patients' age, duration of presenting problem, and number of treatment sessions. There was no significant difference between the groups for age (t(39)=1.74, p=.09), duration of presenting problem (t(39)=-1.33, p=.19), or for the number of treatment sessions (t(39)=-.26, p=.80). Chi-square tests of independence were performed to determine whether gender, marital status, education, and comorbidity differed between medicated and unmedicated patients at the six-month follow-up time point. The proportion of females to males in the medicated group was not significantly different to the proportion of females to males in the CBT group, $\chi^2(1, N=41)=.74$, p=.39, and there was no significant difference between the groups for marital status $(\chi^2(5, N=41)=4.76, p=.45)$, education $(\chi^2(4, N=41)=6.37, p=.17)$ or comorbidity $(\chi^2(1, N=41)=.08, p=.78)$.

Thus, patients who were medicated at six month follow-up were similar to those who were not medicated on major demographic characteristics.

Of the patients who were taking medication following CBT treatment, all continued to be medicated at six-month follow-up. Fourteen reported changes with two increasing the dose, six decreasing the dose, and six changing to different medication. None of the unmedicated patients during CBT treatment commenced medication in the six-month follow-up period.

During the follow-up period, 22 patients had attended services on 169 occasions for their mental health problems, with assistance being sought from psychologists (nine patients), psychiatrists (three patients), counsellors (four patients), general practitioners

(four patents), and group programs (two patients). The mean number of visits was 8.45 per patient although this was skewed by seven patients attending services on 110 occasions.

Table 23

Characteristics of Patients by Treatment-type Group.

Characteristic	Medicated	Not Medicated
Number of patients	29	12
Gender (% female)	79.3	66.7
Mean age in years (SD)	40.9 (12.0)	33.8 (11.5)
Marital status (%)		
Single (never married)	24.1	33.3
Married	37.9	16.7
Divorced/separated	24.1	41.7
Education (highest level %)		
Completed to year 10	6.9	16.7
Completed to year 12	20.7	33.3
Technical/trade	34.5	0.0
Completed tertiary	31.0	33.3
Comorbid Axis 1 diagnosis (%)	62.1	66.7
Duration of problem in months (SD)	41.3 (62.0)	72.4 (81.5)
Number of treatment sessions (SD)	9.6 (1.4)	9.8 (1.5)

The characteristics of those who responded to the follow-up questionnaires and those who did not are displayed in Table 24.

Table 24

Characteristics of Responders and Non-responders to Follow-up Questionnaires.

Characteristic	Responders	Non-responders
Number of patients	41	36
Gender (% female)	78.0	75.0
Mean age in years (SD)	38.8 (12.2)	33.5 (11.4)
Marital status (%)		
Single (never married)	30.0	50.0
Married	30.0	13.9
Divorced/separated	20.0	19.5
Education (highest level %)		
Completed to year 10	25.6	22.9
Completed to year 12	23.1	45.7
Technical/trade	17.9	5.7
Completed tertiary	33.3	25.7
Comorbid Axis 1 diagnosis (%)	53.2	46.8
Duration of problem in months (SD)	37.4 (57.5)	31.2 (33.8)
Number of treatment sessions (SD)	10.1 (2.5)	8.9 (3.3)
Mean BDI pre-treatment (SD)	27.7 (11.0)	32.0 (10.6)
Mean BAI pre-treatment (SD)	21.1 (12.0)	24.5 (13.5)
Mean ASI pre-treatment (SD)	31.7 (12.5)	32.6 (11.5)
Mean ACS pre-treatment (SD)	4.18 (.63)	4.35 (.51)
Mean BDI post-treatment (SD)	16.3 (13.0)	24.0 (15.7)
Mean BAI post-treatment (SD)	13.8 (8.2)	17.6 (11.5)
Mean ASI post-treatment (SD)	22.8 (12.2)	23.7 (14.1)
Mean ACS post-treatment (SD)	3.88 (.75)	4.12 (.58)

To determine whether demographic or clinical characteristics differed between responders and non-responders to the follow-up questionnaires, independent samples t-tests were performed on patients' age, duration of presenting problem, number of treatment sessions and pre- and post-treatment measured of depression and anxiety symptoms and fear of emotion.. After correction for multiple comparisons, there was no significant difference between the groups for age (t(75)=-1.98, p=.05), duration of presenting problem (t(75)=-.55, p=.59), the number of treatment sessions (t(75)=-1.71, p=.09), pre-treatment depression symptoms (t(75)=1.64, p=.11), pre-treatment anxiety symptoms (t(75)=1.09, p=.28), pre-treatment ASI scores (t(75)=.32, t=.75), pre-treatment ACS scores (t(75)=1.30, t=.20), post-treatment depression symptoms (t=.10).

Chi-square tests of independence were performed to determine whether gender, marital status, education, and comorbidity differed between those who returned the sixmonth follow-up questionnaires and those who did not. The proportion of females to males in the medicated group was not significantly different to the proportion of females to males in the CBT group, $\chi^2(1, N=77) = .10$, p=.75, and there was no significant difference between the groups for marital status ($\chi^2(5, N=76) = 6.06$, p=.30), education ($\chi^2(3, N=74) = 5.49$, p=.14) or comorbidity ($\chi^2(9, N=77) = 3.21$, p=.96).

Change in anxiety and depression symptoms over treatment

To determine whether there was a difference in symptoms over time between those who received CBT only and those who received CBT and pharmacotherapy, 2 x 3 mixed design ANOVAs were conducted with medication status at six-month follow-up as the between subjects variable with two levels (medicated and not medicated). The within subjects variable was time with three levels (pre-treatment, post-treatment and

six-month follow-up). The dependent variables were the total scale scores on the BDI and the BAI. The means and standard deviations are presented in Table 25.

Table 25

Depression and Anxiety Symptom Scores at Pre-treatment, Post-treatment and Sixmonth Follow-up.

Measure	Medicated	N	Mean	Standard
				deviation
BDI				
Pre-tx	Yes	29	28.00	11.63
	No	12	27.00	11.63
Post-tx	Yes	28	16.31	13.22
	No	12	18.25	15.23
Follow-up	Yes	28	15.76	13.73
	No	12	18.08	15.03
BAI				
Pre-tx	Yes	29	21.31	11.33
	No	12	21.75	13.61
Post-tx	Yes	25	13.59	8.81
	No	12	15.17	8.93
Follow-up	Yes	25	13.14	9.41
	No	12	14.92	10.51

For depression symptoms (BDI), there was a significant main effect for time, Wilks' Lambda=.541, F(2,38)=16.12, p<.05. This indicates that there was a change in depression symptom scores over time. Partial eta squared had a value of .46 indicating a large effect size according to Cohen's (1988) criteria. The main effect for medication

was not significant, F(1,39)=.07, p=.79, η^2 =.002, nor was the interaction between medication and time, F(2,38)=.37, p=.69, η^2 =.02. To better understand the main effect of time, a post-hoc 2x2 mixed design ANOVA was conducted. The between subjects variable was medication status at the six-month follow-up (medicated, not medicated) and the within subjects variable was time (post-treatment, six-month follow-up). The main effect for time was not significant (F(1,39)=.07, p=.79, η^2 =.002), neither were the main effects for medication (F(1,39)=.21, p=.65, η^2 =.01), nor the interaction between medication status and time (F(1,39)=.02, p=.89, η^2 =.001).

For anxiety symptoms (BAI), there was a significant main effect for time, Wilks' Lambda=.601, F(2,38)=12.60, p<.05. This indicates that there was a change in anxiety symptoms over time. Partial eta squared had a value of .40 indicating a large effect size. The main effect for medication was not significant, F(1,39)=.16, p=.69, η^2 =.004, nor was the interaction between medication and time, F(2,38)=.10, p=.91, η^2 =.005. As with BDI data, a post-hoc 2x2 mixed design ANOVA was conducted with the BAI as the dependent variable. The between subjects variable was medication status at the six-month follow-up (medicated, not medicated) and the within subjects variable was time (post-treatment, six-month follow-up). The main effect for time was not significant (F(1,39)=.11, p=.74, η^2 =.003), neither were the main effects for medication (F(1,39)=.31, p=.58, η^2 =.01), nor the interaction between medication status and time (F(1,39)=.01, P=.93, P=.001).

Change in fear of emotion over treatment

To determine whether there was a difference in fear of emotion over time between those who received CBT only and those who received CBT and pharmacotherapy, 2 x 3 mixed design ANOVAs were conducted with medication status at six-month follow-up as the between subjects variable with two levels (medicated and not medicated). The within subjects variable was time with three levels (pre-treatment,

post-treatment and six-month follow-up). The dependent variables were the total scale scores on the ASI and the ACS. The means and standard deviations are presented in Table 26.

Table 26

Fear of Emotion Scores at Pre-treatment, Post-treatment and Six-month Follow-up.

Measure	Medicated	N	Mean	Standard
				deviation
ASI-total				
Pre-tx	Yes	28	31.61	12.96
	No	12	30.75	13.56
Post-tx	Yes	28	21.46	11.76
	No	12	23.83	11.58
Follow-up	Yes	28	20.04	13.28
	No	12	20.67	16.08
ACS-total				
Pre-tx	Yes	25	4.10	.65
	No	12	4.37	.64
Post-tx	Yes	25	3.72	.69
	No	12	4.13	.77
Follow-up	Yes	25	3.51	.87
	No	12	4.00	.80

For the ASI-total, there was a significant main effect for time, Wilks' Lambda=.574, F(2,37)=13.75, p<.05. This indicates that there was a change in fear of emotion scores over time. Partial eta squared had a value of .43 indicating a large effect size according to Cohen's (1988) criteria. The main effect for medication was not

significant, F(1,38)=.03, p=.856, $\eta^2=.001$, nor was the interaction between medication and time, F(2,37)=.33, p=.720, $\eta^2=.018$. To better understand the main effect of time, a post-hoc 2x2 mixed design ANOVA was conducted. The between subjects variable was medication status at the six-month follow-up (medicated, not medicated) and the within subjects variable was time (post-treatment, six-month follow-up). The main effect for time was not significant (F(1,39)=2.12, p=.15, $\eta^2=.05$), neither were the main effects for medication (F(1,39)=.22, p=.64, $\eta^2=.01$), nor the interaction between medication status and time (F(1,39)=.22, p=.64, $\eta^2=.01$).

For the ACS-total, there was a significant main effect for time, Wilks' Lambda=.763, F(2,34)=5.27, p<.05. This indicates that there was a change in fear of emotion scores over time. Partial eta squared had a value of .24 indicating a large effect size. The main effect for medication was not significant, F(1,35)=3.10, p=.087, η^2 =.08, nor was the interaction between medication and time, F(2,34)=.28, p=.76, η^2 =.016. As with ASI data, a post-hoc 2x2 mixed design ANOVA was conducted with the ACS as the dependent variable. The between subjects variable was medication status at the sixmonth follow-up (medicated, not medicated) and the within subjects variable was time (post-treatment, six-month follow-up). The main effect for time was not significant (F(1,35)=3.27, p=.08, η^2 =.08), neither were the main effects for medication (F(1,35)=3.00, p=.09, η^2 =.08), nor the interaction between medication status and time (F(1,35)=.20, p=.66, η^2 =.01).

Predicting symptom severity

To determine if fear of emotion predicted severity of anxiety and depression symptoms six months after the completion of CBT treatment, two sets of multiple regression analyses were performed.

Firstly, multiple regression analyses were performed with depression and anxiety symptom scores (BDI and BAI) at follow-up as the dependent variables and fear

of emotion scores on the ASI and ACS at post-treatment as predictor variables. In replicating the analysis by Arch and Craske (2007), pre-treatment symptom scores were also entered into the regression as the first step to control for pre-treatment symptom severity. Table 27 shows the standardised regression coefficients (β), the variance and the change in variance accounted for by the model. For BDI scores at follow-up, R for regression was significant, F(3,33)=19.18, p<.05 in the final model. After controlling for pre-treatment depression symptom severity, fear of emotion scores at post-treatment accounted for 25.3% of the variance in follow-up depression scores. Fear of emotion as measured by the ACS was a significant predictor. For BAI scores at follow-up, R for regression was significant, F(3,33)=23.59, p<.05 in the final model. Controlling for pre-treatment anxiety symptom severity, fear of emotion scores at post-treatment accounted for 21.5% of the variance in follow-up anxiety scores. As with the depression scores, fear of emotion as measured by ACS was a significant predictor, but not as measured by ASI.

Table 27
Summary of Multiple Regression Predicting BDI and BAI Follow-up Scores from ASI-total and ACS-total Post-treatment Scores Controlling for Pre-treatment Scores.

	β	T	R^2 (adjusted R^2)	ΔR^2	\overline{F}
BDI					
Step 1			.38 (.36)	.38	21.67*
BDI pre-tx	.62	4.66*			
Step 2			.64 (.60)	.25	11.46*
ASI-total	.13	.86			
ACS-total	.44	2.71*			
BAI					
Step 1			.47 (.45)	.47	30.61*
BAI pre-tx	.68	5.53*			
Step 2			.68 (.65)	.22	11.18*
ASI-total	.23	1.53			
ACS-total	.34	2.32*			

^{*}*p*<.05

A second pair of hierarchical multiple regression analyses were performed with symptom scores at follow-up (BDI and BAI) as the dependent variables and fear of emotion scores (ASI and ACS) as post-treatment as predictor variables. In contrast to Arch and Craske (2007), post-treatment symptom scores were entered as the first step to control for symptom severity at the completion of CBT treatment. Table 28 shows the standardised regression coefficients (β), the variance and the change in variance accounted for by the model.

Table 28

Summary of Multiple Regression Predicting BDI and BAI Follow-up Scores from ASItotal and ACS-total Post-treatment Scores Controlling for Post-treatment Scores.

	β	T	R^2 (adjusted R^2)	ΔR^2	F
BDI					
Step 1			.71 (.70)	.71	86.86*
BDI post-tx	.84	9.32*			
Step 2			.75 (.72)	.04	2.25
ASI-total	.11	.85			
ACS-total	.15	1.00			
BAI					
Step 1			.61 (.60)	.61	55.14*
BAI post-tx	.78	7.43*			
Step 2			.67 (.64)	.06	2.78
ASI-total	.17	1.02			
ACS-total	.21	1.35			

^{*}*p*<.05

For BDI scores at follow-up, R for regression was significant, F(3,33)=32.53, p<.05 in the final model. After controlling for post-treatment depression symptom severity, however, fear of emotion scores did not account significantly for additional variance, and neither fear of emotion measure was a significant predictor of symptom scores at follow-up. Similarly, for BAI scores at follow-up, R for regression was significant, F(3,33)=22.10, p<.05 in the final model. After controlling for post-treatment anxiety symptom severity, fear of emotion scores did not account for significant

additional variance. As with depression scores, neither of the fear of emotion scores was a significant predictor of symptom scores at follow-up.

To determine if change in fear of emotion scores from pre-treatment to post-treatment predicted severity of anxiety and depression symptoms six months after the completion of CBT treatment, the regression analyses were repeated. The variables and steps were as for the previous analyses other than the predictor variables, which were defined as the change in fear of emotion scores over CBT treatment. The same pattern of results was found.

These results show that fear of emotion at completion of treatment and change in fear of emotion over treatment are weak to moderate predictors of depression and anxiety symptoms at six-month follow-up when controlling for pre-treatment symptom severity, but not when controlling for post-treatment symptom severity.

Chapter 9 Discussion for Study 2

This study was carried out to investigate if fear of emotion was associated with symptoms of anxiety and depression six months after completing CBT treatment and if there were any differences in fear of emotion scores between medicated and unmedicated patients who attended a community mental health centre.

Fear of emotion and symptom severity

To determine if fear of emotion at post-treatment was associated with symptom severity at follow-up, analyses were performed taking into account depression and anxiety symptoms at pre-treatment and post-treatment.

Results of regression analyses showed that fear of emotion scores at posttreatment were a weak to moderate predictor of symptoms of anxiety and depression, accounting for 21.5% and 25.3% of variance in follow-up scores while controlling for pre-treatment symptom scores. This finding is consistent with the study published by Taylor, Jakubovski and Bloch (2015) who reported that fear of emotion at posttreatment was a significant predictor of symptoms and relapse in patients with anxiety disorders at six-month follow-up, although that study did not control for pre-treatment symptom scores. Controlling for pre-treatment symptom scores is important because pre-treatment severity of symptoms has been shown to be a reliable predictor of treatment outcome, with patients recording higher scores at pre-treatment also recording higher scores at post-treatment. This holds for patients presenting with depression (Hamilton & Dobson, 2002) and anxiety (Kampman, Keijsers, Hoodguin & Hendriks, 2008; McEvoy, Nathan, Rapee & Campbell, 2012). The finding of the current study is also consistent with Boswell et al. (2013) who found that change in fear of emotion scores predicted symptom scores at follow-up while controlling for pre-treatment scores.

The findings of the current study differ from those of Mavissakalan and Guo (2004), who reported that fear of emotion scores at post-treatment did not predict symptom intensity at follow-up. The regression analyses in that study controlled for additional variables including the rate of change across the follow-up period, and the number of months between the follow-up assessment and the penultimate assessment. Relapse was also calculated as a dichotomous variable rather than as scores on a continuous measure of symptoms. However, their results show that despite no differences in fear of emotion scores at pre-treatment, those who relapsed had significantly higher scores of fear of emotion at their last assessment session than those who did not relapse.

In the second regression analysis, symptom scores at follow-up were not predicted by post-treatment fear of emotion scores when controlling for post-treatment symptom scores. This finding is at odds with the study published by Schmidt and Bates (2003) who found that post-treatment fear of emotion scores predicted anxiety symptom scores at follow-up. In the current study, anxiety and depression symptom scores remained low between post-treatment and six-month follow-up. Consequently, there was very little remaining variance to explain in the six-month follow-up symptom scores. Assuming the CBT intervention was effective in reducing patients' fear of emotion, then the follow-up symptom scores would likely remain low. To further understand the relationship between post-treatment fear of emotion and anxiety and depression symptoms at six-month follow-up, bivariate correlations were performed as supplementary analyses. These showed that fear of emotion at post-treatment, as measured by ASI, correlated strongly with anxiety symptom scores (r=.69, p<.05) and depression symptom scores (r=.57, p<.05) at six-month follow-up. Strong correlations were also observed between fear of emotion at post-treatment, as measured by ACS, and anxiety symptoms (r=.69, p<.05) and depression symptoms (r=.69, p<.05) at sixmonth follow-up. Thus, the variance in follow-up symptom scores explained by post-treatment fear of emotion scores is between 32.5% and 47.6% which is greater than the variance explained when controlling for pre-treatment scores.

These findings suggest that if a person is fearful of emotions at post-treatment, then they are likely to report more symptoms at follow-up.

Change in fear of emotion over treatment and follow-up and medication status

The second question addressed by this study was whether fear of emotion scores changed across treatment and at a six-month follow-up period, and if that change differed between those who received CBT treatment and those who received combined therapy with CBT and naturalistic pharmacotherapy.

Results from the current study showed that fear of emotion decreased over CBT treatment and remained low at six-month follow-up. This pattern of results was consistent across both fear of emotion measures. These results were expected and consistent with the results discussed in Study 1.

The secondary hypothesis was not supported in that those patients receiving combined CBT with pharmacotherapy did not fare worse than those patients who received CBT alone. Overall, results from the current study were consistent with published findings reviewed in Chapter 6, with fear of emotion scores decreasing over treatment. Additionally, in this study, the decreased fear of emotion scores were maintained at six-month follow-up. No differences were found between those who were medicated and those who were not, a finding shared with the studies reported by Telch et al. (1993) and Marchand, Roberge, Primiano and Germain (2009). In both of those studies, the participants had a primary diagnosis of panic disorder with or without agoraphobia, the CBT treatment was administered in group format, and the most common type of medications used was anxiolytics. The current study extended those findings in two ways: Firstly, the predominant medications used were antidepressants.

Antidepressants combined with CBT treatment have been shown to be more effective than medication alone in treating panic disorder (Roy-Byrne et al., 2005). Secondly, in addition to one person diagnosed with panic disorder and agoraphobia, the remaining patients were diagnosed with other anxiety or depressive disorders.

The results from the current study differ from those reported by Arch and Craske (2007). In their study, patients being treated with CBT for panic disorder had lower scores on fear of emotion at six-month follow-up compared with patients who were medicated with SSRI antidepressants and received CBT. In the current study, there were insufficient participants to allow analyses to be conducted by medication class. Arch and Craske (2007) also reported than when a broader group of antidepressant medication was considered (all classes of antidepressants as opposed to just SSRI and SNRI antidepressants), the difference between the non-medicated group and the medicated group failed to reach significance. Thus the comparable analysis in the current study shares this finding. A further replication study with more patients taking SSRI/SNRI medication would be helpful to further explore the possible differences between conditions.

Limitations

This study sought to advance understanding of the relationship between fear of emotion and symptoms of anxiety and depression in patients undergoing CBT treatment with and without medications. The conclusions drawn from this study are subject to limitations of the design.

As for the previous study, the two treatments provided cannot be considered to be optimised; indeed the purpose of the study was to determine how treatment delivered in a manner consistent with routine clinical practice would impact on the relationship between fear of emotion and symptoms of anxiety and depression. The CBT component of treatment was delivered in a community mental health centre and was facilitated and

supervised by staff with many years of experience in delivering CBT interventions. This transdiagnostic treatment was manualised and has been shown to be effective in the treatment of depression and anxiety disorders (McEvoy & Nathan, 2007). This may mean that the CBT component was delivered in a manner closer to protocols established in efficacy studies than the medication condition where prescribing was not subject to any attempts to standardise or regulate. While the question remains about the optimisation of the pharmacotherapy, the situation tested in the current study would be typical of clinical practice where a clinician administering a CBT intervention to medicated clients would have little or no opportunity to influence the medication prescribed to a patient. Therefore, to further improve generalisability of results, it would be interesting to see whether the same pattern of results emerged with 'average' CBT from a range of, say, community providers.

Like in the previous study, participants were not randomly assigned to the CBT alone or combined therapy groups. It is possible that selection biases existed for the reasons previously discussed (e.g., patients with more severe symptoms may be more likely to be medicated). However, there were no differences between the two groups on any of the measured variables at pre-treatment. Other design limitations included no control group and no blinding to treatment condition, although these limitations are consistent with effectiveness studies. Again as for the previous study, a number of statistical analyses were performed on the same data set. Except where stated, alpha protection was not used to reduce the risk of Type 1 'family-wise' errors. Therefore it possible that the significant findings reported were spurious, although, once again, most of the analyses were not 'family-wise' as they were testing theoretically distinct hypotheses (O'Keefe, 2003).

In addition to these factors, there are further limitations relating to the current study. This study did not require patients to abstain from therapeutic interventions

between post-CBT treatment and the follow-up survey. The survey questions asked patients to disclose any other treatments they had undertaken in the six-month follow-up period. Fifty per cent reported that they had attended a psychologist, psychiatrist, counsellor/therapist, general practitioner or group program for an average of 8.45 visits. For the medicated patients, 50% also changed their medication by increasing the dosage, decreasing the dosage or changing to different medication. To remove the potential influence of these different treatments on the results, a larger number of participants who remained stable on their medication and did not engage in other treatments between post-treatment and follow-up would have to be recruited.

With regard to medication classes, a considerable body of literature has investigated the impact of benzodiazepine drugs on various measures of treatment outcome and mechanisms of change. Interestingly, in this study only three patients were medicated with benzodiazepines. Thus, separate analyses were not conducted for these participants and comparisons were not made with other studies.

It is possible that fear of emotion may be confounded with severity of emotion, such that those participants with higher levels of emotion may therefore experience more fear. This potential confound was explored with post hoc multiple regression analyses with depression and anxiety symptom scores (BDI and BAI) at post-treatment as the dependent variables and fear of emotion scores on the ASI and ACS at pretreatment as predictor variables. Symptom change scores, calculated as the post-CBT treatment scores on the BDI and BAI subtracted from the corresponding pre-treatment scores were also entered into the regression as the first step to control for symptom change. For BDI scores at post-CBT treatment, R for regression was significant, F(2, 37)=17.24, p<.05 in the final model. After controlling for change in depression symptom scores, fear of emotion scores at pre-treatment accounted for 13.9% of the variance in post-treatment depression scores. Fear of emotion as measured by both the

ASI and ACS was a significant predictor. For BAI scores at post-CBT treatment, R for regression was significant, F(2, 37)=29.83, p<.05 in the final model. Controlling for change in anxiety symptom scores, fear of emotion scores at pre-treatment accounted for 40.5% of the variance in post-treatment scores. As with the depression scores, fear of emotion as measured by both the ASI and ACS was a significant predictor. These results suggest that fear of emotion predicts depression and anxiety symptoms after controlling for emotion severity.

The follow-up scores on both fear of emotion measures were similar in magnitude to post-treatment scores and both exhibited large standard deviations. As a result, the current study was underpowered to detect significant small differences. Of the 77 patients who completed the CBT treatment, only 53% responded to requests to complete follow-up measures; had this proportion been higher then the study may have had sufficient participants to detect differences between groups.

Finally, it is possible that the participants who responded to the follow-up survey were different in some way to the 47% of participants who did not respond to the survey. However, the participants who returned the six-month follow-up questionnaires did not differ from those who did not in terms of age, gender, marital status, education, co-morbidity, chronicity, or number of CBT treatment sessions attended. There were also no differences between those who returned questionnaires and those who did not on pre-CBT treatment and post-CBT treatment measures of depression and anxiety symptoms or fear of emotion.

Implications

This study is one of very few to investigate the impact of medication on CBT treatment of fear of emotion in patients with depression and/or anxiety disorders.

Fear of emotion appears to be an important treatment target, but traditional CBT is effective in reducing this whether concurrent medication is given or not. It is possible

that CBT protocols could be further enhanced to decrease patients' fear of emotion by adding treatment components to the standard protocols. A likely candidate for this is to add interoceptive exposure exercises, such as those described by Barlow and Craske (1994). These exercises directly target the fear of experiencing and responding to physical cues of emotional reactions by mimicking those feared symptoms and learning to tolerate them. This treatment approach has been an important component of CBT treatments for panic disorder. Thus, elements of interoceptive exposure could be included in transdiagnostic protocols to further reduce fear of emotion, and ultimately the reporting of symptoms of anxiety and depression. This approach has been incorporated in Barlow et al.'s (2011) so called 'Unified Protocol" and preliminary data show that it is effective in decreasing fear of emotion (Sauer-Zavala, Boswell, Gallagher, Bentley, Ametaj & Barlow, 2012). In a targeted intervention, a single session consisting of psychoeducation and interoceptive exposure was effective in reducing fear of emotion scores in non-treatment seeking individuals with elevated anxiety sensitivity scores (Keough & Schmidt, 2012). Future studies could look at the impact of incorporating interoceptive exposure into standard CBT protocols to either further reemphasise not responding to emotions fearfully or to augment existing strategies.

Fear of emotion is not the only target of treatment – other variables explain the majority (approximately 75%) of variance in anxiety and depression symptom scores – but attempting to help patients remain remitted following treatment, being less fearful of their emotions appears useful.

Despite the limitations, there are a number of strengths in this study. The impact of a broad range of medications was investigated, not just benzodiazepines and tricyclic antidepressants that were the focus of many previous studies. Importantly, the construct of fear of emotion was examined across a range of disorders, not just panic disorder as in the majority of previous studies. These two factors improve the generalisability of the

results to other community healthcare settings, and help to clarify the transdiagnostic nature of fear of emotion and how it responds to treatment.

Conclusions

Following CBT treatment, fear of emotion scores predicted severity of symptoms of anxiety and depression at follow-up. That is, patients who remained fearful of their emotions following treatment were likely to report higher levels of symptoms of anxiety and depression six months later. While this relationship has been shown with panic disorder, this study suggests that the relationship holds for depression and other anxiety disorders as well. Patients undergoing treatment for psychological disorders with CBT had improved outcomes on measures of fear of emotion at the completion of treatment, which was maintained at six-month follow-up. These outcomes were similar in patients concurrently taking psychotropic medications and in unmedicated patients.

Chapter 10 General Discussion

The purpose of this research project was to further the understanding of the construct of fear of emotion, its measurement, its relationship to psychological disorders, and how it changes over treatment and follow-up with CBT alone or combined therapy in a community mental health centre.

In the first study, two measures for measuring fear of emotion were compared. Consistent with previously published psychometric studies, the subscales of the two measures were strongly correlated with the total scale scores, and the subscale scores of each measure were moderately correlated. Overall, the total scale scores of the Anxiety Sensitivity Index (ASI) and the Affective Control Scale (ACS) were moderately correlated.

Secondly, fear of emotion, as measured by these two instruments, predicted scores on symptom measures of anxiety and depression in patients diagnosed with a depressive or anxiety disorder, with each measure's subscale scores explaining a larger proportion of the variance than total scale scores. There was support for the specificity of the subscales of the ACS, with the depression subscale uniquely predicting depression symptoms and the anxiety subscale uniquely predicting anxiety symptom scores. There was some support for the specificity of the ASI subscales with the Physical Concerns subscale predicting anxiety symptoms.

Thirdly, fear of emotion scores decreased from pre-treatment to post-treatment in both treatment conditions; CBT alone or CBT combined with naturalistic pharmacotherapy. The data also suggested that those patients who received CBT alone had lower scores than those who received CBT and medication (both before and after treatment). This pattern of results was also observed in the analyses of the subscales, which also showed an effect for medication in the ACS depression and anxiety subscales.

Fourthly, when investigating the type of medication used in the combined therapy condition, fear of emotion scores decreased over CBT treatment regardless of medication class, with those taking the combination of antidepressant and anxiolytic medication reporting higher scores before and after treatment compared with those taking antidepressant medication alone or not taking psychotropic medication at all. In the analyses of subscales, medication use was significantly associated with ASI Mental Concerns.

Fifthly, tests for clinical significance showed a trend toward patients undergoing CBT without medication to achieve more reliable improvement and clinically significant change in fear of emotion scores than medicated patients. However, these results did not achieve statistical significance.

In the second study, fear of emotion at post-treatment predicted symptom severity at follow-up when symptom severity was controlled at pre-treatment, but did not predict symptom severity at follow-up when symptom severity was controlled at post-treatment. Secondly, the reductions in fear of emotion scores observed over CBT treatment were maintained over the follow-up period. Thirdly, there was no difference at follow-up in fear of emotion scores between those receiving CBT alone to those receiving combined CBT and pharmacotherapy.

Overall, these findings indicated that fear of emotion is a cognitive construct that is elevated in individuals diagnosed with depression or an anxiety disorder. Fear of emotion responds to treatment with either CBT alone or combined with pharmacotherapy. Fear of emotion following successful CBT treatment also predicted severity of symptoms of anxiety and depression at six-month follow-up even when fear of emotion remained stable across this period. The addition of pharmacotherapy to CBT did not affect fear of emotion scores at six-month follow-up.

Implications for theory and clinical practice.

The findings of the current studies inform the theory and practice of the assessment and management of anxiety and depressive disorders in clinical practice.

From a theoretical perspective, three key themes emerge: fear of emotion is a transdiagnostic factor in anxiety and depression; it changes with treatment; and decreased fear of emotion is associated with the maintenance of therapeutic gains six months after completing psychological treatment.

The current findings support the view that fear of emotion is a primary maintaining factor of panic disorder and agoraphobia. Since the introduction of this construct, subsequent studies have shown that fear of emotion is also a feature of anxiety disorders and depression (as reviewed in Chapter 2). The current study extends these findings to a more general clinical population. This finding highlights the transdiagnostic nature of fear of emotion in that awareness and fearful responding to the sensations of emotion is a feature of people with anxiety and depression.

The findings also show that fear of emotion is a malleable characteristic in that it responds to treatment. The current studies showed that fear of emotion decreased over treatment either with CBT alone or when combined with naturalistic pharmacotherapy, along with symptoms of anxiety and depression in individuals with a diagnosed mood or anxiety disorder. It was interesting to note that combining CBT with pharmacotherapy did not lead to improved outcomes, nor did it lead to adverse outcomes. That is, CBT was effective in reducing fear of emotion regardless of the patient's medication status. Fear of emotion has been shown to be responsive to pharmacotherapy (as detailed in Chapter 2) and it is possible that medicated patients presenting for CBT treatment had already experienced some decrease in their fear of emotion scores. However, upon presenting for psychological treatment, pre-treatment scores on the ASI were of the same magnitude as in clients who were not medicated.

This suggests that CBT can provide at least an additional benefit in managing fear of emotion. Clinicians can also be confident that, with the exception of benzodiazepines (Westra et al., 2002), the patient's medication status need not affect the potential gains a patient can make from CBT.

Fear of emotion also predicted symptoms over time. It is conceivable that patients who were less fearful of emotion at the end of treatment were more confident in maintaining the gains across the ensuing months and less sensitive to relapse compared with those who were more fearful of emotions.

Thus, not only does fear of emotion decrease over treatment, it appears that it is important that it does so, in order to maintain symptom reduction. The current study suggested that CBT was an effective intervention in reducing fear of emotion; however, it is likely that other therapeutic approaches would achieve similar outcomes. As proposed by Boswell et al. (2013), a reduction in the fearful response to emotions could be achieved through biological, cognitive, behavioural or acceptance-based approaches.

As reviewed in Chapter 2, biological therapy (i.e., the use of medication) has been shown as one way of maintaining decreased fear of emotion (Romano, van Beek, Cucchi, & Perna, 2004; Simon et al., 2004), and the current study suggests that this can be improved upon as further reductions were seen when medication was augmented with CBT, specifically by challenging unhelpful thoughts or predictions about experiencing emotion. Albeit these positive outcomes, further research could look at improving treatment techniques to target fear of emotion in order to produce lasting effects.

From a clinical perspective, the findings of this study inform clinicians about the need for fear of emotion to be addressed in the treatment of anxiety and affective disorders, and the importance of assessing and measuring this important construct. This

is especially important as clinically significant change findings were modest, suggesting there is opportunity for improved treatment.

As the studies reviewed in Chapter 2 show, fear of emotion is amenable to treatment whether targeted directly or indirectly as part of treatment for depression and anxiety disorders. In the current studies, a specific treatment component involved directly challenging patients' beliefs about experiencing strong emotion (the example in the treatment manual is waking up feeling depressed) or sensations of emotion (e.g., noticing one's heart beating rapidly or feeling unusually hot). Patients are then guided through a typical CBT thought discovery and challenging exercise in which they are encouraged to identify distressing thoughts about these feelings, look for confirming or more importantly disconfirming evidence, and arrive at a more balanced way of thinking; e.g., "Just because I have woken up feeling depressed doesn't mean that I'll be depressed forever", or "Even though I'm feeling hot, it is just a symptom of anxiety and will soon pass".

One treatment strategy that has been shown to be highly effective in directly targeting fear of emotion is interoceptive exposure (Taylor, 2003). This is a series of tasks where sensations of emotional experiences are deliberately evoked in order for the patient to learn that the sensations are not harmful (i.e., a cognitive rationale) and habituate to the anxious arousal elicited (i.e., a behavioural rationale). Future studies could investigate adding interoceptive components to existing therapies to further reduce fear of emotion. This approach has been suggested with particular regard for somatic symptom disorder and illness anxiety disorder (Norton & Sears Edwards, 2017).

Just as there are transdiagnostic maintaining factors of emotional disorders (see Harvey, Watkins, Mansell & Shafran, 2004), trans-therapy change processes have been proposed (McEvoy & Erceg-Hurn, 2016) to manage these. That is, there may be

therapeutic techniques in different therapy 'brands' that achieve the same outcome. Acceptance-based approaches from ACT have also been shown to reduce fear of emotion (e.g., Roemer & Orsillo, 2007). It is entirely possible that merely talking about distressing internal experiences results in a reduction of an individual's fear of emotion such that any therapeutic approach where emotional expression is managed in a non-fearful way could be helpful.

If reducing fear of emotion is to be at least a secondary focus of treatment in patients with anxiety and depressive disorders, then appropriate measurement of the construct is important. Both of the measures used in current study were developed specifically to assess fear of emotion; the ASI with regard to panic and the ACS for a broader range of emotions (depression, anxiety, positive emotions, anger). As the two measures were at least moderately correlated, as expected, it would appear clinically prudent to use the simplest measure to make administering and scoring convenient for patients and clinicians. The ASI offers the benefit of fewer items and the most recent version (ASI-3) shows improved psychometric properties, particularly of the three subscales. However, the current studies showed some evidence of specificity of the subscales on the ACS and future research could ascertain if reducing the items in the 42 item full-scale could yield similar information and provide a more thorough assessment of fear of emotion.

As the ASI appears to be a theoretically and clinically useful measure of fear of emotion, it may be more accurate for it to be considered as an 'Emotion Sensitivity Index' to reflect the broader application of the measure from anxiety sensitivity to a more general sensitivity of emotion.

Limitations

The major limitations of these studies reflect the difficulties of clinically applied research. Effectiveness studies such as these increase generalisability of findings and are

important to inform about likely outcomes in routine clinical contexts. However, they come at the cost of a range of uncontrolled variables which prevent definitive conclusions being drawn, particularly with regard to factors such as the temporal sequencing of change, and the contribution of various treatment components.

In addition to the limitations described in the discussions of the two previous studies (see chapter 5 and chapter 9), other common limitations are acknowledged. As described earlier, fear of emotion at post-treatment accounted for just over 20% of the variance in depression and anxiety symptoms six months after completing CBT treatment. Other factors that account for the remaining variance may also interact with fear of emotion. For example, a patient's self-efficacy has been shown to be a factor with regard to treatment response such that patients who are confident about being able to manage their emotional distress report greater symptom improvement (Brown et al., 2014). It is possible that there is interaction between patients' fear of emotion and their coping self-efficacy such that, as fear of emotion decreases, coping self-efficacy increases, which then leads to a reduction in emotional distress.

Future studies

These limitations serve as impetus for further studies. The findings of Study 2 show that a reduction in fear of emotion is associated with lower scores on anxiety and depression symptom measures at six month follow-up. It is plausible that being less fearful of emotions leads to the reporting of fewer symptoms or a reduction in their intensity. It is also plausible that a reduction in symptoms of anxiety and depression could lead to reduction in fear of these emotions. Future studies could be designed to specifically address this, such as a time lag design where measures of fear of emotion and symptoms are taken regularly throughout treatment - not just at pre- and post-treatment - to help elucidate the temporal sequencing of these changes.

As suggested in the clinical implications, specifically testing therapeutic techniques designed to reduce fear of emotions could also be investigated. As many treatments appear to offer the prospect of reducing fear of emotion, including fear of emotion measures in studies would be informative; as opposed to extracting fear of emotion techniques and examining as stand-alone interventions.

The current studies focused on patients diagnosed with an anxiety or depressive disorder. If fear of emotion truly is a transdiagnostic construct then it should be evident across all emotions. Preliminary research has shown that fear of positive emotions may be a feature of people diagnosed with bipolar disorders (Gilbert, Nolen-Hoeksema & Gruber, 2013). Including patients with these diagnoses in future studies would provide evidence for this.

Conclusion

Anxiety and depressive disorders are common in the Australian population and despite successful treatments can persist for many years (Bruce et al., 2005). Fear of emotion has been identified as a transdiagnostic factor which is associated with these conditions. Further, a reduction in fear of emotion appears to be associated with maintenance of symptom reduction at six month follow-up. The current study suggests that it is important to continue focusing on this construct to determine more effective treatments to assist patients to overcome anxiety and depression. CBT has been shown to be effective in reducing fear of emotion, whether delivered alone or in combination with naturalistic pharmacotherapy. Researchers and clinicians should continue to further study this construct to assist in our understanding of the maintenance of these common psychological disorders and to develop increasingly effective treatments.

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Appendix A

Information Sheet

Emotion Sensitivity

Thank you for completing the assessment measures marking the end of your treatment at CCI. My name is Bruce Campbell, I am a clinical psychologist (registrar) at CCI and I am also conducting a research project under the supervision of Professor Peter Drummond at Murdoch University.

As part of the CCI process you have previously given consent for us to use and analyse your data for specific purposes. I am now seeking your further consent for two things:

- The first is to report the analysed data as part of my research project. The
 data is entirely de-identified and I will not be reporting any personal details.
 I will be reporting only aggregate or summarised results.
- The second is to follow up with you on your treatment and assess its ongoing effectiveness. If you agree, I will contact you in about six months time for this.

As part of this evaluation, I am investigating the longer-term effectiveness of our treatments and changes to a person's emotion sensitivity over time. Emotion sensitivity can be defined as sensitivity to, and beliefs about inability to cope with strong emotions. I hope to understand the relationship between effective treatments for anxiety and depression and the role of sensitivity to emotions in the outcome of treatment. At CCI this is routinely measured by asking you to complete a number of guestionnaires.

I will invite you to complete four additional questionnaires approximately six months after you finish your treatment at CCI. These questionnaires along with a form to record any more recent treatment you have received in the time since you finished therapy at CCI will be mailed to the address you have recorded with us. I am also able to contact you by telephone to record your responses if this is more convenient for you.

You can decide at any time to withdraw your consent to participate in this research. If you decide to withdraw, any material you have given me will be destroyed. Withdrawal from the research will have no consequences on you being able to access services from this clinic in the future.

All questionnaires completed will be kept completely confidential. Individual answer sheets will be stored separately from identifying information and no individual results will be utilised from this study.

If you are willing to participate in this project, please complete the questionnaires and return them in the reply paid envelope. My supervisor and I are happy to discuss with you any concerns you may have on how this study has been conducted. If you wish to talk to an independent person about your concerns you can contact Murdoch University's Human Research Ethics Committee on 9360 6677 or email ethics@murdoch.edu.au.

This study has been approved by the Murdoch University Human Research Ethics Committee (approval No. 2008/190)

Appendix B

Emotion Sensitivity Study

ı	. Current	Med	ications
	. Current	1 160	เนสเมษาร

Please list any medications that you are currently taking for your psychiatric problem

Name of medication	Dose taken each day	Length of time taking this medication	Length of time taking this dose

Has this medication changed since you were coming to CCI? Yes / No If yes, in what way (has the dose increased decreased, have you stated taking something new or stopped taking a medication)?	
	-
	_
	-

2. Other treatments

Please indicate whether you have attended any of the following for your psychiatric problem since your treatment at CCI.

	Yes	Number of	When seen	Focus of treatment
		appointments		
An outpatient mental				
health clinic				
Psychologist				
Psychiatrist				
Counsellor/therapist				
GP				
Group program				

Appendix C

31st December 2xxx

Ms Jane Citizen 23 Smith St SMITHSVILLE WA 6xxx

Emotion Sensitivity Study

Dear Ms Citizen,

It is now approximately six months since you completed your treatment at the Centre for Clinical Interventions (CCI).

You may recall that you agreed to be contacted at this time to participate in a research study I am undertaking looking at emotion sensitivity. I have attached a copy of the information sheet.

Please find enclosed the four questionnaires that we would like you to complete; two are measures of anxiety and depression symptoms and two are measures of emotion sensitivity. You will have previously completed these questionnaires on at least two occasions while you were coming to CCI.

To help me determine what factors affect emotional sensitivity is it also important for us to know of any changes to medication you are taking or any other therapy or treatment you may have received for the problems for which you presented to CCI. I have also enclosed a sheet with a number of questions for your to record this information if it is relevant to you.

I would be grateful if you could complete the questionnaires and response sheet and return it to me in the envelope provided.

Once I have received your responses I will provide feedback as to how your scores may have changed over time.

I wish to reassure you that the information we receive from you will be kept completely confidential and no information identifying you will be used in the research reports.

If you have any questions, please call me on 9227 4399, otherwise I look forward to receiving the completed questionnaires from you soon. Thank you again for your help.

Yours sincerely

Bruce Campbell Clinical Psychologist (Registrar)

Appendix D

20th February 2xxx

Ms Jane Citizen 99 Address St ADDRESS WA 6100

Emotion Sensitivity Study

Dear Jane,

We recently sent you a letter to see if you would like to receive some feedback about how you are going since you finished the Mood Management Course and to participate in some further research at CCI. As yet we haven't heard back from you, and so I am writing to you again just in case the first package went astray.

I have enclosed another copy of the questionnaires and a reply paid envelope to hopefully make it more convenient for you to return. However, *if it easier for you we can complete the measures over the phone*. If you would prefer to do this, please call me on 9227 4399 and we can make a time that suits you.

Of course, once we have the information I will write to you again and provide feedback on any changes in the measures that have occurred since the end of the Course.

Once again, I wish to reassure you that the information we receive from you will be kept completely confidential and no information identifying you will be used in the research.

If you have any questions, please call me, otherwise I hope to receive the questionnaires from you soon.

Thank you again for your help.

Yours sincerely

Bruce Campbell Clinical Psychologist

Appendix E

27th February 2xxx

Ms Jane Citizen 23 Smith St SMITHSVILLE WA 6xxx

Emotion Sensitivity Study

Dear Jane

Thank you for completing the questionnaires we sent you recently. It was good to hear back from you!

I am now writing to provide you with some feedback on the questionnaires to see if here have been any changes in the six months since you finished the Mood Management Course at CCI.

The table below shows your scores on the questionnaires before you started therapy (**Pretreatment**), at the end of the Mood Management Course (**Post-treatment**) and on the most recent set (**6months follow-up**).

Your scores on all the questionnaires indicate that the gains you made were maintained and even improved n in the six-month period after completing the course. For the measures, the downward arrow next to a score shows that you have improved. Here are your scores:

	Pre- treatment	Post -treatment	6months follow-up
Depressive Symptoms	38 (extremely severe)	I7 (moderate) ↓	I0 (minimal) ↓
Anxiety Symptoms		9 (normal) ↓	
Sensitivity to Anxiety			63% ↑ (improved)
Reactions to Emotions			

We hope you find this feedback useful. If you have any questions about these scores please call me on 9227 4399.

You have done very well to maintain the gains you made over the last six months. We hope the strategies and principles you learned during the program are continuing to be helpful as you continue on life's journey.

Again, thank you for your help in completing the forms, and I wish you all the very best for the future.

With kind regards,

Bruce Campbell Clinical Psychologist