The role of cognitive fusion and experiential avoidance in anxiety and depression

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Abstract

This research used the theory underpinning Acceptance and Commitment Therapy (ACT) to develop our understanding of anxiety and depression. ACT proposes that cognitive fusion (CF) and experiential avoidance (EA) are two processes fundamental to psychological distress. However, CF and EA's role in anxiety and depression in the context of one another has not been established. This study aimed to test the hypotheses that CF and EA would make both unique and interrelated contributions to explaining a) variance in symptoms of anxiety and depression, and b) the effect of three different internal (worry and rumination) and external (stressful life-events) vulnerabilities to anxiety and depression.

A correlational cross-sectional design was conducted in a student (n=106) and clinical sample (n=57). The hypotheses were additionally tested longitudinally in the student sample (n=97). Cross-sectionally, in students, only CF explained unique variance in anxiety and depression and mediated the relationships between vulnerabilities to and indicators of symptomology. These results were not replicated longitudinally. In the clinical sample, CF acted in concert with EA in explaining variance in symptomology and in mediating the effect of all three vulnerability factors.

These results partially supported CF and EA as core transdiagnostic processes in anxiety and depression, and therefore key targets for prevention and treatment. CF appeared particularly relevant to students' mental health, with CF's serial

effect through EA increasingly important where clinically significant symptoms were present. Further longitudinal and experimental research is needed to verify the causal assumptions inherent in this study.

Abbreviations

AAQ	Acceptance and Action Questionnaire
ACT	Acceptance and Commitment Therapy
BEAQ	Brief Experiential Avoidance Questionnaire
CBT	Cognitive Behavioural Therapy
CF	Cognitive Fusion
CFQ	Cognitive Fusion Questionnaire
CI	Confidence Interval
DASS	Depression Anxiety Stress Scale
DSM	Diagnostic and Statistical Manual of Mental Disorders
DV	Dependent Variable
EA	Experiential Avoidance
GAD	Generalised Anxiety Disorder
GAD-7	Generalised Anxiety Disorder Assessment
IAPT	Improving Access to Psychological Therapies
IV	Independent Variable
LESS	Life Events Scale for Students
MEAQ	Multidimensional Experiential Avoidance Questionnaire
MH	Mental Health
NICE	National Institute for Health and Clinical Excellence
PHQ-9	Patient Health Questionnaire
PSWQ	Penn State Worry Questionnaire
PTSD	Post-Traumatic Stress Disorder
R&D	Research and Development
REC	Research Ethics Committee
RRS	Ruminative Response Scale
SD	Standard Deviation
SE	Standard Error
SPSS	Statistical Package for Social Sciences
SRRS	Social Readjustment Rating Scale
T1/T1	Time 1/ Time 2
VIF	Variance Inflation Factors

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CHAPTER 1: INTRODUCTION

1.1 Chapter Overview

Anxiety and depression affect nearly one in five adults in the United Kingdom at any one time (Office for National Statistics, 2016); however, treatment remains far from fully effective. Thus, advancing our understanding of the key factors involved in their development and maintenance is of paramount importance. This helps identify core targets for intervention, thereby reducing the unrelenting impact of anxiety and depression on society.

Traditionally, theory and research has focused on the content of an individual's internal and external world in understanding anxiety and depression. For example, negative thinking patterns (worry and rumination) and stressful life-events are formulated as key factors in the development of these mental health (MH) disorders. However, more recently, the theory underpinning Acceptance and Commitment Therapy (ACT, S. C. Hayes, Strosahl, & Wilson, 1999) has provided an exciting shift in the way it conceptualises psychological distress. ACT proposes that difficult thoughts, life-experiences and associated feelings are ubiquitous and not problematic per se. Rather, how we *relate* to our internal and external world is the key determinant of MH. Cognitive fusion (CF) and experiential avoidance (EA) describe two unhelpful ways in which we relate to our experiences, implicated as core processes in the development and maintenance of MH difficulties (S. C. Hayes, Luoma, Bond, Masuda, & Lillis, 2006). However, CF and EA's contribution to explaining variance in anxiety and

depression in the context of one another is largely unknown. Furthermore, the ACT prediction that the impact of negative thinking (worry, rumination) and stressful life-events on anxiety and depression occurs indirectly through CF and EA has not been directly tested. This study aimed to address these gaps in knowledge.

This chapter will first define anxiety and depression and their impact on society, before discussing the role of negative thinking patterns (worry and rumination) and stressful life-events in their development. Next, the theory and model of ACT, and its supporting evidence, will be presented as a framework within which to advance this current understanding. Firstly, the relationships of EA and CF with anxiety and depression will be reviewed. Secondly, the role of EA and CF as core mediating variables in the relationships between vulnerabilities to, and indices of, MH problems will be discussed. This will specifically focus on exploring EA and CF's role in explaining the impact of worry, rumination and stressful life-events on anxiety and depression. Finally, having highlighted gaps in our current understanding, the research aims and hypotheses of this study will be presented.

1.2   Anxiety and Depression

MH problems affect approximately one in four British adults in any one-year (McManus, Meltzer, Brugha, Bebbington, & Jenkins, 2009). This constitutes an immense personal cost to individuals and their wider systems, as well as a considerable economic and social cost to society (Davies, 2013; Insel, 2011).

Anxiety and depression are the most common of these MH disorders (National Institute for Health and Clinical Excellence [NICE], 2011a).

Anxiety describes a normal response to threat or danger that has evolved to keep us safe. However, when anxiety is exaggerated, enduring and significantly impacts daily functioning, it can be characterised as a MH problem. Generalised anxiety disorder (GAD) is the most common anxiety disorder (Singleton, Bumpstead, O'Brien, Lee, & Meltzer, 2001). Other anxiety disorders include social phobia, obsessive-compulsive disorder, specific phobias, health anxiety, panic disorder and post-traumatic stress disorder (PTSD). While specific anxiety disorders have a different constellation of symptoms, all have a common theme of fear, tension, worry, and physical changes in the body (Davey, 2008). Anxiety disorders have a high prevalence, with a yearly rate of 18% and lifetime prevalence of almost 30% reported in adults (Kessler, Ruscio, Shear, & Wittchen, 2010; Kessler et al., 2005).

Depression is characterised by low mood and/or loss of interest or pleasure. Clinical features also include changes in appetite and sleep, feeling restless or slowed down, fatigue, excessive feelings of worthlessness and guilt, concentration difficulties and recurrent thoughts of death (American Psychiatric Association, 2013). Major depression is one of the leading causes of disability worldwide and a major contributor to suicide (Vos et al., 2015; Whiteford et al., 2013). It affects approximately 8-12% of the UK population in any year

(Singleton et al., 2001), with a reported lifetime prevalence of mood disorders at just over 20% (Kessler et al., 2005).

Anxiety and depression are often discussed as categorical entities, with prevalence studies determining their 'presence' versus 'absence'. This dates back to the work of Kraepelin (1899) and is reflected in psychiatric classification systems, such as the *Diagnostic and Statistical Manual of Mental Disorders* (DSM, American Psychiatric Association, 2013) or *International Classification of Diseases* (World Health Organization, 2004). However, there has been a growing movement away from this diagnostic conceptualisation, instead viewing MH on a continuum (e.g. Angst & Merikangas, 1997; Angst, Merikangas, & Preisig, 1997; Lilienfeld, 1998; Marzillier, 2004; Verdoux & van Os, 2002). From this perspective, the experiences of those with severe and debilitating anxiety or depression are quantitatively, but not qualitatively, different from those with milder forms.

Following on from this, researchers use both clinical and non-clinical samples to clarify whether the same processes are relevant across the MH continuum. This enhances our understanding of anxiety and depression in different populations and where similar processes operate, allows the use of non-clinical analogue samples to test theoretical models (Abramowitz et al., 2014). As a result, literature from both clinical and non-clinical populations will be reviewed during this chapter. A particularly well-researched non-clinical population are university students. Of note, students exhibit higher levels of anxiety and

depression compared to the general population (Eisenberg, Gollust, Golberstein, & Hefner, 2007; Roberts & Zelenyanski, 2002; Stewart-Brown et al., 2000), likely reflecting the pressures of university life (e.g. financial, academic and relationship difficulties; Grant, 2002).

In summary, anxiety and depression have a high prevalence and considerable impact on society. However, treatments, such as Cognitive Behavioural Therapy (CBT), fall short of optimal effectiveness (Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012; Olatunji, Cisler, & Deacon, 2010). This emphasises a pressing need to further understand the processes underpinning anxiety and depression to identify the best targets for intervention. The next section will highlight core vulnerabilities to anxiety and depression that theory and research has traditionally focussed on, before introducing the theory underpinning ACT to help advance this understanding.

1.3 Predictors of Anxiety and Depression

CBT is a common framework in which MH disorders are formulated and the recommended treatment for anxiety and depression (NICE, 2009; NICE, 2011b). Within CBT, the content and nature (e.g. intensity, frequency, negative valence) of our thoughts and thinking styles are emphasised (Beck, Rush, & Shaw, 1979). Worry and rumination are two repetitive, negatively-toned categories of thinking particularly associated with anxiety and depression respectively. Worry describes apprehensive expectation of possible negative outcomes of future events (Borkovec, Robinson, Pruzinsky, & DePree, 1983). Rumination describes

recurrent thinking about the self, past upsetting events, unresolved concerns and depressive symptoms, their causes and consequences (Nolen-Hoeksema, 1998). They are conceptualised as distinct constructs with worry typically future-oriented and rumination past-focused (Watkins, Moulds, & Mackintosh, 2005).

As well as being emphasised in the diagnostic criteria of GAD (American Psychiatric Association, 2013), worry is a common factor across all anxiety disorders (Barlow, 1988; Borkovec et al., 1983; Papageorgiou, 2006). Furthermore, worry is considered causally related to anxiety, not just part of its phenomenology (Purdon & Harrington, 2006). A path analysis demonstrated that while worry predicted anxious arousal, anxiety did not predict worry (Gana, Martin, & Canouet, 2001).

The association between rumination and depression has long been proposed. Beck (1967) discussed the tendency of depressed patients to ruminate on perceived defects and other negative cognitions. In the response styles theory (Nolen-Hoeksema, 1991), the tendency to ruminate in response to feelings of sadness is a risk factor for depression. This has been empirically supported. Rumination predicts the onset, length and severity of depressive episodes (Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, 2000). Experimental inductions of ruminative thinking also impact depressive mood (e.g. Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998; Trask & Sigmon, 1999).

As well as theory and research focussing on one's internal world, external life-experiences have also been implicated as key precipitating factors in psychological distress. For example, the stress-vulnerability model (Zubin & Spring, 1977) proposed that a genetic predisposition to mental illness is not sufficient to manifest the disorder, but requires interaction with psychosocial stressors or stressful life-events. A substantial body of evidence has supported the link between difficult life-events and depression and anxiety (e.g. Barrett, 1979; Kendler, Karkowski, & Prescott, 1999; Paykel, 1978; Spinhoven et al., 2011a; Spinhoven et al., 2011b; Surtees et al., 1986).

However, much is still unknown about how our thoughts and life-experiences impact wellbeing. As well as being pervasive in psychopathology, worry and rumination are common everyday phenomena (Davey & Wells, 2006; Harvey, Watkins, Mansell, & Shafran, 2004; Tallis, Davey, & Capuzzo, 1994; Wells & Morrison, 1994). This raises the question as to what turns these thinking patterns inherent in us all into clinical symptoms of anxiety and depression. Furthermore, stressful life-events are neither necessary nor sufficient for symptom development (Wardenaar, van Veen, Giltay, Zitman, & Penninx, 2014), suggesting other mediating and moderating factors are at play. The theory and model of ACT (S. C. Hayes et al., 1999) has shown promise in providing a new framework within which to develop this understanding. Rather than focusing on the content or frequency of different 'problematic' patterns of thought or 'distressing' life-events, ACT proposes our *relationship* to these internal and

external experiences is more important. The ACT model will be discussed in more detail next.

1.4   Acceptance and Commitment Therapy (ACT)

ACT (S. C. Hayes et al., 1999) is a psychological treatment that has gained substantial clinical and research interest over the last two decades. It broadly sits within the Cognitive Behavioural Therapies; however, it differs from traditional CBT and Cognitive Therapy, which have focussed on the *content* of our thoughts (Beck et al., 1979), and is rather consistent with mindfulness-based ideas emphasising our *relationship* to our experiences (Kabat-Zinn, 1990).

ACT proposes that psychological pain is universal. To take action towards valued goals, contact with the full spectrum of emotions is inevitable. Rather than difficult experiences being problematic in themselves, an individual's *relationship* to these experiences is considered the key determinant of MH (S. C. Hayes et al., 2006). Within ACT, and herein this thesis, 'experiences' refers to the full spectrum of internal (i.e. thoughts, memories, emotions, bodily sensations, behavioural dispositions) and external (i.e. life-events) experiences one can have.

These ideas are based on a philosophy of science called Functional Contextualism (Biglan & Hayes, 1996) and a theory of language and cognition called Relational Frame Theory (S. C. Hayes, Barnes-Holmes, & Roche, 2001). These philosophical and theoretical frameworks suggest that no thought, feeling,

or behaviour is inherently problematic. Rather, this depends on the context in which these experiences occur. Within ACT, 'context' principally refers to the way in which an individual *relates* to their experiences and the extent to which this inhibits or supports a life that is consistent with their core values.

The ACT model highlights six transdiagnostic processes characterising different unhelpful relationships to our experiences. While all six processes are interrelated, each is more linked with one process than the others (S. C. Hayes, Strosahl, & Wilson, 2011). The first linked pair includes 'experiential avoidance' (EA) and 'cognitive fusion' (CF). EA describes all attempts to get rid of, avoid or escape from unwanted thoughts, feelings, memories, bodily sensations and behavioural dispositions and the situations that elicit them (S. C. Hayes et al., 1999). CF describes a state in which one is excessively entangled with aversive thoughts, viewing them as a reflection of literal facts about the content to which they refer (i.e., the world, oneself, the past, future, others etc.; S. C. Hayes et al., 1999). As well as unique processes directly leading to psychological distress, ACT suggests that the more fused an individual gets with their difficult thoughts, the more motivated they will be to avoid them and associated aversive experiences (i.e. higher CF leads to greater EA, S. C. Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). The second pair includes 'attachment to the conceptualised self' and 'lack of contact with the present moment'. As an individual gets more entangled with their thoughts (CF) and avoidant of aversive experiences (EA), they cannot access a continuous conceptualisation of themselves that is separate from their thoughts and feelings and contact with the

present moment is lost. The third and final pair includes 'lack of values clarity' and 'unworkable action', whereby people lose sight of what they desire in life, beyond liberation from psychological pain (S. C. Hayes et al., 2006).

These six processes make up the overall state of 'psychological inflexibility' (see Figure 1), where action is overly driven by a 'rigid dominance of psychological reactions, over chosen values and contingencies' (Bond et al., 2011, p. 678). While those experiencing mental ill health would be expected to demonstrate a more psychologically inflexible relationship to their experiences, ACT takes a dimensional approach to MH (S. C. Hayes et al., 2011) with these processes relevant and present, all be it to a lesser extent, in non-clinical as well as clinical populations (Levin, Hildebrandt, Lillis, & Hayes, 2012).
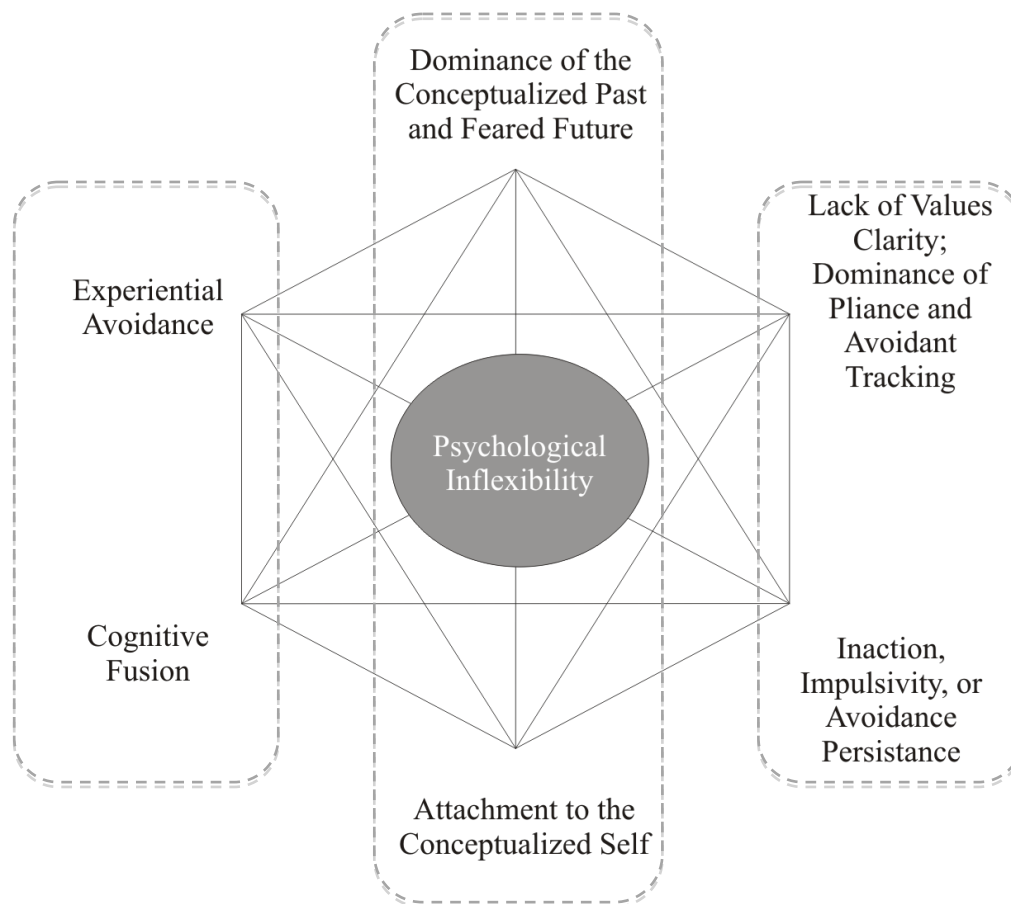
Figure 1: An ACT Model of Psychopathology, Adapted from S. C. Hayes et al. (2006)

ACT therapy reflects the underpinning theory and aims to change one's unhelpful relationships to difficult experiences, in a way that supports valued living (Ciarrochi, Bilich, & Godsell, 2010). It has shown considerable promise in randomised controlled trials across wide-ranging problems, including anxiety disorders and depression (Avdagic, Morrissey, & Boschen, 2014; Forman, Herbert, Moitra, Yeomans, & Geller, 2007; Roemer & Orsillo, 2007; Tamannaeifar, Gharraee, Birashk, & Habibi, 2014; Zettle, Rains, & Hayes, 2011).

To summarise, ACT moves from focussing on the content or nature (e.g. intensity, frequency, negative valence) of internal and external experiences towards thinking further about common unhelpful *relationships* one may have with these experiences (S. C. Hayes et al., 1999). Six processes constitute the overall state of 'psychological inflexibility', which underpins psychological distress. Of these, EA and CF are hypothesised to be the cornerstone of psychopathology (S. C. Hayes et al., 2011). They cut across different diagnoses and are at the heart of the other processes in the ACT model (S. C. Hayes et al., 2011). As a result, this study focussed on EA and CF to help advance our understanding of the key factors involved in anxiety and depression. The following section will discuss EA and CF in more detail and review the literature supporting their role in MH. EA has been discussed first, as this is where the majority of previous research has focussed (Chawla & Ostafin, 2007).

1.5 Experiential Avoidance and Cognitive Fusion

1.5.1 Experiential Avoidance (EA)

The motivation of animals to avoid situations associated with negative affect, driven by the evolutionary advantage this brings, has long been established (e.g. Blanchard & Blanchard, 1968). According to the theory underpinning ACT, as verbal-beings, humans avoid private events in a similar way to the actual external threat (S. C. Hayes et al., 2001). For example, the recollection of a traumatic event can lead to re-experiencing the associated pain and hence will

be avoided in the same way as the event itself. In this way, language greatly increases the cues for danger to be avoided (S. C. Hayes et al., 2001). Thus, EA encompasses the motivation to evade *all* aversive private experiences, as well as the situations that elicit them, even when doing so is futile or interferes with valued action (S. C. Hayes et al., 1996). The latter part of this definition highlights the inflexible and indiscriminate context in which EA occurs.

Specific avoidance strategies people may use include cognitive avoidance (e.g. thought control, suppression and reappraisal; Lazarus, 1991; Wenzlaff & Wegner, 2000); affective avoidance (e.g. emotional suppression; Gross & Levenson, 1993); and behavioural avoidance (e.g. avoiding certain places or situations, excessive drinking/drug-use and parasuicidal behaviour; Baker, Piper, McCarthy, Majeskie & Fiore, 2004; Chapman, Gratz & Brown, 2006). EA is the psychological process that unites these topographically distinct avoidance strategies by their common function (Chawla & Ostafin, 2007; S. C. Hayes et al., 2004).

EA is considered detrimental to wellbeing (S. C. Hayes et al., 1999). Paradoxically, suppressing a thought or emotion triggers a subsequent increase in its frequency (Wenzlaff & Wegner, 2000). This could be because suppression attempts inherently reference the item to be suppressed (e.g. 'Don't think of *a dog',* contains the word '*dog'*) and increase the salience of cues related to the suppression item (S. C. Hayes et al., 2011). Furthermore, directly conditioned private events are not readily eliminated by verbal rules, with attempts at

verbally regulating internal experiences rendered futile (S. C. Hayes et al., 1996). Finally, avoiding situations that might elicit difficult internal experiences usually comes with high costs and inhibits value-congruent action (Kashdan, Barrios, Forsyth, & Steger, 2006).

The Acceptance and Action Questionnaire (AAQ-I, S. C. Hayes et al., 2004; revised AAQ-II, Bond et al., 2011) has almost exclusively been used to measure EA and empirically establish its involvement in psychopathology (Chawla & Ostafin, 2007). S. C. Hayes et al. (2006) published the first exhaustive review of ACT evidence, finding EA accounted for 28% of variance in health-related outcomes. Since then, a growing literature has found EA to be reliably associated with psychological distress, including a range of anxiety disorders and depression (Blakey, Jacoby, Reuman, & Abramowitz, 2015; Kashdan et al., 2013; Kumpula, Orcutt, Bardeen, & Varkovitzky, 2011; Roemer, Salters, Raffa, & Orsillo, 2005; Ruiz, 2010). Furthermore, EA's prospective role in predicting change in anxiety and depression symptomology over two-years was supported in a mixed sample of 2,316 adults (Spinhoven, Drost, de Rooij, van Hemert, & Penninx, 2014).

Although research has supported EA's relationship with psychological distress, poor psychometrics of the AAQ has threatened the validity of these findings (Chawla & Ostafin, 2007). The questionnaire has limited discriminant validity (Gámez, Chmielewski, Kotov, Ruggero, & Watson, 2011), especially when discriminating between levels of EA (i.e. the process) and negative affect (i.e. the outcome of the process). AAQ-II items load onto the same factor as items

measuring distress, rather than EA (Wolgast, 2014), risking overestimations of EA's relationship with psychopathology. Discriminant validity with neuroticism is also poor (Gámez et al., 2011). Additionally, given EA encompasses a wide variety of behaviours, its measurement is further complicated (McMullen, Taylor, & Hunter, 2015). The internal consistency of the AAQ-I has not been particularly high, with alpha coefficients of .70 or lower (S. C. Hayes et al., 2004). This could reflect the scale's heterogeneous nature without the different parts forming a coherent representation of EA. The revised AAQ-II demonstrated higher internal consistency (Bond et al., 2011); however, its ability to incorporate all aspects of EA comprehensively is still questioned (Gámez et al., 2011). Finally, as well as assessing EA, the AAQ is often used to assess psychological inflexibility more generally (S. C. Hayes et al., 2006). This highlights a lack of clarity around what the questionnaire was actually designed to measure.

Researchers have since developed the Multidimensional Experiential Avoidance Questionnaire (MEAQ, Gámez et al., 2011), with the aim of addressing the limitations of the AAQ and comprehensively assessing the different facets of EA. This included behavioural avoidance, distress aversion, procrastination (i.e. delaying activities that may cause distress), attempts to ignore, suppress and distance from distress as well as distress endurance. The MEAQ showed excellent internal consistency (α=.91-.95), as well as convergent and discriminant validity across clinical and non-clinical samples (Gámez et al., 2011). However, the MEAQ's length (62-items, 12 minute average completion time) practically constrained its use. Therefore, a shorter version, the 15-item

Brief Experiential Avoidance Questionnaire (BEAQ, Gámez et al., 2014), was developed. The BEAQ exhibited content from each of the MEAQ subscales and good psychometric properties, with an internal consistency of .80-.86 (Gámez et al., 2014). Construct validity was shown by its convergence with a range of well-validated avoidance-related measures, as well as measures of negative emotionality and psychopathology (including anxiety and depression). Unlike the AAQ-II, which showed stronger associations with neuroticism, negative affect and psychopathology than with avoidance (Gámez et al., 2011), the BEAQ was more strongly associated (but not multicollinear) with measures of avoidance (Gámez et al., 2014).

The BEAQ's psychometrics show considerable promise; however, given how recently it was developed, to our knowledge only three studies have used it to date. The BEAQ was used to demonstrate that attentional control moderated the relationship between EA and PTSD symptoms (Bardeen & Fergus, 2015), and to clarify the relationship between CF and health anxiety was independent of EA (Fergus, 2015). The test-retest reliability has also been recorded in a sample of cancer patients (r=.85, Carr, 2014).

To summarise, ACT has implicated EA as a core mechanism in anxiety and depression. However, this understanding is limited by the almost exclusive use of the widely criticised AAQ in supporting research. As well as measuring EA, the AAQ has been found to tap into negative affect as well as the broader construct of

psychological inflexibility. The development of the BEAQ (Gámez et al., 2014) has provided a more robust instrument with which to clarify EA's unique role in MH.

1.5.2   Cognitive Fusion (CF)

In addition to EA, ACT proposes CF is another core process negatively impacting MH (S. C. Hayes et al., 1999). CF refers to excessive entanglement with difficult thoughts, such that they are treated as if they illustrate reality. For example, we may react to the thought, 'I am useless' or 'I am going to have a panic attack' as if this were an inevitable conclusion. In this way, we respond to our mental construction as though we are responding to a physical situation and verbal events dominate emotional and behavioural regulation to the exclusion of other contextual variables (S. C. Hayes et al., 2011). By taking a more detached relationship to difficult thoughts, the illusion of language can be dissolved (Ciarrochi et al., 2010). This shifts one's perspective away from, 'My thoughts tell me how things really are and what I need to do', towards, 'My thoughts are just one way to think about things – what I do next is my choice and based on what works' (S. C. Hayes et al., 2011). This later position is called cognitive defusion (i.e. the opposite to CF). Therefore, 'negative' thoughts are not problematic in themselves, but over-identification with verbal processes is what leads to suffering (S. C. Hayes et al., 1999). As well as CF directly impacting MH, ACT proposes that higher CF also motivates greater avoidance of all aversive experiences (i.e. EA). In this way, CF and EA are unique but interrelated processes (S. C. Hayes et al., 2011).

CF shows some similarities with other constructs in the literature (Gillanders et al., 2014), in particular, 'decentering' or 'metacognitive awareness' (Fresco et al., 2007; Safran & Segal, 1990; Teasdale et al., 2002; Wells, 2008). Decentering and metacognitive awareness both describe taking a detached, present-focused and accepting view of one's thoughts and emotions. This includes elements of cognitive defusion, as well as other processes in the ACT model, such as acceptance, self-as-context (i.e. the opposite of 'attachment to the conceptualised self'), present-moment awareness and self-compassion. While related, CF is more narrowly defined and behaviourally operationalised, particularly focused on the impact of fusion with thoughts on valued workable action. This distinction has been empirically supported, with decentering (measured using the Experiences Questionnaire, Fresco et al., 2007) containing two sub-factors, namely cognitive defusion and self-as-context (McCracken, Barker, & Chilcot, 2014).

Empirical evidence of CF's relationship with psychological distress has been stalled by a lack of measures adequately operationalising it (Gillanders et al., 2014). Indeed, McCraken et al. (2014) described CF as the neglected facet of the ACT model. The limited research has largely used experimental (e.g. S. C. Hayes et al., 1999; Takahashi, Muto, Tada, & Sugiyama, 2002), component (Levin et al., 2012) and clinical outcome designs (Zettle et al., 2011) to investigate the impact of cognitive defusion techniques (using measures of 'believability' of thoughts) on distress. Masuda, Hayes, Sackett and Twohig (2004) used an alternating treatment design in a small sample of undergraduate students (n=8) to find the

believability of, and discomfort associated with, self-relevant thoughts reduced in a defusion condition as compared to distraction and control conditions. Furthermore, Zettle et al. (2011) found a greater reduction in self-reported depression in a clinical sample following an ACT (n=13) compared to Cognitive Therapy (n=12) intervention, with believability of thoughts mediating this effect.

However, measures of 'believability of thoughts' fail to fully capture the construct of CF (Gillanders et al., 2014). CF measures should more broadly encompass the dominance of cognition in a person's experience, including the inability to view thoughts from a different perspective, reacting emotionally to thoughts, and using thoughts as the predominant guide for action (Gillanders et al., 2014). Additionally, assessments of believability of thoughts, such as one-item rating scales (e.g. Masuda et al. 2004) are psychometrically questionable (Gillanders et al., 2014).

The Drexel Defusion Scale (Forman et al., 2012) has also been used to measure cognitive defusion, demonstrating a negative relationship with psychopathology (Bernstein et al., 2015; Forman et al., 2012). However, this questionnaire has been criticised for its use of vignettes looking at defusion in constrained hypothetical scenarios, and for describing what is meant by the term defusion in the extended instructions, possibly priming defused responding (Gillanders et al., 2014).

The Cognitive Fusion Questionnaire (CFQ, Gillanders et al., 2014) was recently developed to address the previous failure to adequately operationalise CF. It has demonstrated good reliability as well as convergent, divergent and incremental validity in a series of studies involving over 1,800 people across diverse clinical and non-clinical samples (Gillanders et al., 2014). A good internal consistency (α=.88-.90) and test-retest reliability (r=.80) was documented, as well as predicted correlations with related constructs and outcomes, including mindfulness, thought control, distress, anxiety, depression and life satisfaction (Gillanders et al., 2014). The promising psychometrics of the CFQ have given opportunity to more robustly assess CF's role in MH. Indeed, a recent study used the CFQ to find a positive relationship between CF and health anxiety, independent of EA, in community adults (n=371, Fergus, 2015). Furthermore, Gillanders, Sinclair, MacLean and Jardine (2015) found in a sample of adults with cancer (n=105) that the CFQ was the strongest predictor of anxiety (however not depression) over and above illness-related cognitions, avoidant coping and self-compassion. Controlling for EA and related constructs when assessing CF's *unique* role in MH was a strength of these two studies over previous research.

It should be noted that the CFQ has demonstrated very high correlations with the AAQ-II (r=.72-.87, Gillanders et al., 2014) possibly indicating CF and EA are so interdependent that they represent the same underlying construct. Alternatively, the high correlation could reflect the fact that the AAQ-II is often used to assess the overall construct of 'psychological inflexibility', and not just EA (S. C. Hayes et al., 2006). This implies that the questionnaire was also designed to measure

other constructs within the ACT model, with a number of AAQ items related to CF (Gillanders et al., 2014).

While CF and EA are intrinsically linked in the ACT model, they have traditionally been researched separately, with their relative contributions to MH in the context of each other largely neglected. More recently, Bardeen and Fergus (in press) used a cross-sectional design in a large sample of adults from the general population (n=955) to find that the relationship between CF (measured using CFQ) and outcome variables (anxiety, depression, stress, PTSD) became stronger as EA (measured using AAQ) increased. They concluded that the aggregate effect of high EA and CF was particularly detrimental to MH. The authors highlighted a need to use more robust measures of EA, citing the BEAQ, to verify their results, and for researchers to continue to examine theoretically grounded and ecologically valid models that include both CF and EA to help clarify the complex interrelations among potential risk factors to MH.

The above study was interested in the aggregate effect of CF and EA, rather than their sequential relationship. Dinis, Carvalho, Gouveia and Estanqueiro (2015) are the only researchers to have examined whether EA at least partially explains CF's effect on MH. They used a cross-sectional design in a general population sample (n=181) to find a significant proportion of CF's (measured using CFQ) effect on depression occurred indirectly through EA (measured using AAQ). While their correlational cross-sectional design prevented causality from being empirically determined, their results were consistent with ACT theory (S. C.

Hayes et al., 1999), whereby high CF leads to greater avoidance of all difficult experiences (EA), which in turn increases suffering.

To summarise, ACT theory and a small body of research has implicated CF as another fundamental mechanism in MH, and a key context in which EA manifests. The development of the CFQ has allowed researchers to more robustly assess CF and further research in this area is now required.

1.5.3   Summary

The theory and research underpinning ACT has been discussed, particularly highlighting EA and CF's role in the development and maintenance of anxiety and depression. However, shortcomings of the measures used to assess these processes have impacted this understanding. The emergence of better-validated questionnaires provides potential to address this in the future. Additionally, with CF and EA mainly studied separately, their relationships with anxiety and depression in the context of each other has yet to be substantively established. Preliminary research has supported ACT's conceptualisation of CF and EA as both unique and interrelated processes related to psychological distress (Bardeen & Fergus, in press; Dinis et al., 2015). Finally, cross-sectional designs have dominated with limited research considering CF and EA's temporal associations with future outcomes.

The next section will extend our understanding of EA and CF's role in MH beyond just their simple associations with symptomology, to consider EA and CF as core

mechanisms explaining the effect of more traditionally held vulnerabilities to mental ill health.

1.6   EA and CF as Mediating Processes

Mediational models allow a more refined theoretical understanding of MH by determining the important psychological processes underpinning the relationships between predictor and outcome variables. The central tenet of ACT is that it is not the content, nature or frequency of difficult thoughts, emotions or life-experiences that directly impacts anxiety and depression, but rather how we *relate* to these experiences (S. C. Hayes et al., 1999). This implicates EA and CF as core mediating processes that might explain the impact of more traditional predictors of psychological distress.

EA's mediational characteristics were first highlighted when S. C. Hayes et al. (1996) conceptualised EA as a core transdiagnostic functional domain explaining the effect of psychological and situational vulnerabilities to MH difficulties. This has been empirically supported. Kashdan et al. (2006) used a cross-sectional design to find EA accounted for the effects of emotion inhibition, rumination and perceived anxiety uncontrollability on anxiety-related distress in undergraduates (n=382). EA further mediated the relationships between two emotion regulation strategies (emotion suppression and cognitive reappraisal) and negative and positive daily experiences in a 21-day experience-sampling methodology in 97 students. This suggested emotion-focused coping strategies only become problematic when inflexibly applied with the resolve to avoid or

minimise difficult experiences (EA). The authors concluded EA is 'a core toxic diathesis underlying several other psychological vulnerabilities' (Kashdan et al., 2006, p. 1302).

Furthermore, EA has been found to mediate other relationships in MH. These include the relationships between both childhood trauma and negative affect intensity, and the tendency to engage in problem behaviours (J. Kingston, Clarke & Remington, 2010); life hassles and distressing delusional experiences (Goldstone, Farhall, & Ong, 2011); self-critical perfectionism and depressive symptoms (Moroz & Dunkley, 2015); coping strategies and psychopathology (Costa & Pinto-Gouveia, 2011; Fledderus, Bohlmeijer, & Pieterse, 2010); and anxiety sensitivity and depression (Tull & Gratz, 2008). This suggests EA provides a more streamlined explanation for the impact of many disparate external and internal predictors of psychopathology (Boulanger, Hayes, & Pistorello, 2010). However, these studies have relied on the AAQ, which not only assesses EA, but also psychological inflexibility more broadly (as previously discussed). Therefore, it remains unclear what is the key mediator(s) in these relationships. Furthermore, the majority of these studies have used cross-sectional designs, with limited longitudinal research investigating temporal associations.

Given CF is considered another maladaptive way of relating to one's experiences (S. C. Hayes et al., 1999), it may also play a mediating role in psychological distress. While few studies have researched this, Trindale and Ferreira (2014)

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found CF mediated the negative effect of body-related cognitions on eating psychopathology in female students (n=342). Furthermore, Gillanders et al. (2015) investigated the roles of CF and avoidant coping in mediating the relationships of threatening illness-related appraisals with anxiety and depression in cancer patients (n=105). CF (measured using CFQ) mediated the effect of threatening appraisals on anxiety. In contrast, avoidant coping (measured using the Emotional Avoidance Coping subscale of the Brief-COPE inventory; Carver, 1997) mediated the relationship between threatening appraisals and depression.

Based on CF and EA's proposed interrelationship, whereby CF at least partially underpins the manifestation of EA (S. C. Hayes et al., 2011), one would further predict a serial mediation effect. In other words, vulnerabilities to poor MH lead to increased CF, which motivates greater EA, in turn causing increased psychological distress. Only one study has explicitly researched this. In the aforementioned research by Dinis et al. (2015), the authors also examined the mediating role of CF and EA in the relationship between memories of early-life shame experiences and current depressive symptoms. Where shame memories presented with traumatic-like characteristics, both CF and EA exhibited *unique* mediational effects, such that they independently explained a significant proportion of the relationship between perceived impact of shame experiences and depressive symptoms. Furthermore, an additional double mediation effect was found, suggesting fusion with difficult cognitions around traumatic shame experiences also increased avoidance of aversive internal experiences, leading to

greater depression. Note, the cross-sectional, correlational design could not establish causality and the mediational model was rather grounded in theory.

In summary, growing evidence has developed S. C. Hayes et al.'s (1996) initial ideas to consider the mediating role of EA, and more recently CF, in the relationships between predictors and indices of MH. Furthermore, as well as having unique effects, initial support for a serial mediation effect of CF and EA has been found (Dinis et al., 2015), reflective of their interrelationship outlined in ACT (S. C. Hayes et al., 1999). Shifting our attention away from the content of disparate predictors of MH problems, towards common relationships one may have with them, may provide a more helpful explanation of psychological distress and common targets for treatment (Kashdan et al., 2006).

Having developed the rationale for EA and CF as core mediating processes in MH in general, their role in explaining the impact of well-established internal and external vulnerabilities of anxiety and depression more specifically will now be considered. The literature supporting a possible mediating role of EA and CF in the relationships between worry and anxiety, rumination and depression and stressful life-events and anxiety and depression will be reviewed in turn.

1.7 The Relationship between Worry and Anxiety

1.7.1 The Mediating Role of EA

The link between worry and avoidance is not novel. For example, Borkovec, Alcaine and Behar (2004) proposed worry leads the individual to believe they avoided a low-probability catastrophic event and distracts them from highly aversive images and associated autonomic activation (Borkovec & Hu, 1990). Additionally, Well's (1997) metacognitive model of GAD features avoidance techniques as a means of averting the hypothesised dangers of worrying. Roemer and Orsillo (2002, 2005) developed these ideas within an ACT framework. They proposed that GAD is maintained by an individual's reactive, judgmental and 'fused' relationship to their cognitions and associated feelings, thereby perceiving them as overwhelming and dangerous and motivating high EA. EA's rebound effect leads to an increase in symptomatology and inhibits engagement in value-oriented living.

Positive relationships found between worry, EA and GAD symptomology have initially supported the proposition that worry leads to increased anxiety through the negative consequences of EA (Buhr & Dugas, 2012). Roemer et al. (2005) used a cross-sectional study in a non-clinical sample (n=140) to find EA was associated with chronic-worry and further predicted GAD-severity over and above worry. In a clinical sample with GAD (n=19), EA was higher than the non-clinical sample, and significantly associated with stress and anxiety symptomology. It is worth noting that the generalisability of this research was limited by a female-only non-clinical sample and small clinical sample.

Additionally, the cross-sectional design neglected temporal associations. Lee, Orsillo, Roemer and Allen (2010) also used a similarly designed study with a slightly larger clinical sample (n=33) to find analogous results.

The aforementioned research has particularly focused on GAD-samples. EA, a transdiagnostic process (S. C. Hayes et al., 1996), may also be relevant to worry in other anxiety disorders more generally. Furthermore, EA was exclusively measured using the AAQ. 'Worry' explicitly features in two of the ten AAQ questions (e.g. 'Worries get in the way of my success'). This possibly reduces the capacity to discriminate between cognitive content (worry) and one's relationship to such cognitions (Wolgast, 2014), overestimating EA and worry's relationship. Additionally, whether EA *mediates* the relationship between worry and anxiety has not been directly tested, either alone or in the context of CF, as discussed next.

1.7.2 The Mediating Role of CF

Given the verbal-linguistic nature of worry, the theory underpinning ACT would predict that if the thinker becomes cognitively fused with their worrisome thoughts, the same amount of anxiety would be elicited as if the worries were the present reality (S. C. Hayes et al., 2001). This implicates CF as another core process explaining the impact of worry on anxiety. Furthermore, considering high CF is proposed to at least partially underpin the manifestation of EA, CF may be a central component supporting the relationship between worry and EA (S. C. Hayes et al., 1999; Roemer and Orsillo, 2002, 2005). Where an individual has a

more cognitively defused relationship to their worries, a more accepting and functional, rather than experientially avoidant, stance may instead prevail.

Research indicating individuals with GAD view their worrisome thoughts as more dangerous and uncontrollable (Wells & Carter, 1999) may provide initial support for CF's role in worry and anxiety. Furthermore, research has shown cognitive defusion helps explain the impact of therapy on recovery from GAD (Arch, Wolitzky-Taylor, Eifert, & Craske, 2012). However, whether CF mediates the relationship between worry and anxiety, both uniquely and in tandem with EA, requires investigation. This could help explain how the everyday phenomenon of worry can become so problematic (Harvey et al., 2004; Tallis et al., 1994).

1.8 The Relationship between Rumination and Depression

1.8.1 The Mediating Role of EA

While rumination is topographically distinct from worry, it may impact MH through common processes. Like worry, researchers have formulated a relationship between rumination and avoidance in various ways. Ruminators have been proposed to dwell upon past losses or failures in a passive, vague manner to avoid their specific troubles (Dickson, Ciesla, & Reilly, 2012; Watkins & Moulds, 2005) and to deal with aversive thoughts and feelings by behaviourally withdrawing (Moulds, Kandris, Starr, & Wong, 2007; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Nolen-Hoeksema et al. (2008)

discussed the tendency of ruminators to turn to thought suppression and 'escapist' behaviours (e.g. binge eating, drinking, self-harm) to temporarily quell their self-directed ruminative thoughts. Uniting these ideas, Kashdan et al. (2006) proposed rumination is detrimental to MH through the toxic influence of EA.

These theoretical positions have been supported by preliminary research documenting positive relationships between rumination, depression and cognitive, behavioural and experiential avoidance in non-clinical samples (Cribb, Moulds, & Carter, 2006; Dickson et al., 2012; Moulds et al., 2007). Bjornsson et al. (2010) further found in a cross-sectional design in two large student samples (N=748, 887) that rumination was only associated with depression symptoms when EA (measured using AAQ) was high. However, surprisingly, in a second longitudinal component to their study in 72 female students, neither rumination nor EA predicted depressive symptoms 8-12 weeks later, having controlled for baseline symptoms. It is important to note that their sample only exhibited mild symptoms of depression. Furthermore, the AAQ confounds the measurement of EA and negative affect (Gámez et al., 2011). In light of this, controlling for baseline depression may have led to a Type II error (i.e. a "false negative" result). Task-based and experimental studies have further supported the idea that rumination leads to increased avoidance behaviours (e.g. E. F. Kingston, Watkins, & Nolen-Hoeksema, 2014; Thomas, Raynor, & Ribott, 2015).

As previously discussed, Kashdan et al. (2006) more explicitly conceptualised EA as a mediator in their research, finding EA (measured using AAQ) partially accounted for the effect of rumination on anxiety-related outcomes. The authors cautioned that their study needs replicating in clinical samples using other measures of EA. Eisma et al. (2013) also found EA mediated the longitudinal relationship between grief rumination and symptoms of complicated grief following a bereavement and depression (having controlled for baseline) 12-months later.

Only a few studies in this area have used clinical samples. Brockmeyer et al. (2015) examined the role of behavioural avoidance and motive satisfaction (i.e., high agreement between personal motives and actual experiences) in the relationship between rumination and depression in individuals with clinical depression (n=160). Behavioural avoidance mediated this relationship in series with reduced motive satisfaction. The authors concluded rumination defends against engaging in possibly distressing activities, however this reduces motive satisfaction and thereby intensifies depressive symptoms. Note, the authors focussed on behavioural avoidance rather than the broader theoretical construct of EA. Additionally, their cross-sectional correlational design precluded the establishment of causality.

The first study to directly examine the mediating role of EA in the relationship between rumination and depression in adults with and without a historical or present diagnosis of depression (n=2513) was recently published (Spinhoven,

Drost, de Rooij, van Hemert, & Penninx, 2016). A longitudinal design over four-years found that EA predicted onset, relapse and maintenance of depression, as expected. However, these relationships fell insignificant when rumination, worry and neuroticism were controlled for. Furthermore, change in EA did not mediate the prospective effect of rumination, nor worry, on future depressive symptoms, controlling for baseline. While this research demonstrated great strengths, including a fully longitudinal design, EA was measured using the AAQ, which has shown limited ability to discriminate between EA and neuroticism (Gámez et al., 2011) or negative affect (Wolgast, 2014). Therefore, controlling for neuroticism and/or baseline depression may have inadvertently controlled for variance in the AAQ, attenuating the AAQ's relationship with other variables. This could explain these non-significant findings and future research would benefit from more robust measurement of EA.

Furthermore, while researchers have drawn upon the ACT model when theoretically and empirically exploring EA's role in rumination and depression, they have neglected to consider the ACT proposition that CF is a key context in which EA manifests (S. C. Hayes et al., 1999). A more cognitively defused relationship to ruminative thoughts may foster a more reflective approach, disrupting the avoidant processes otherwise triggered. Similar to the relationship between worry and anxiety, looking at EA's mediating role in the context of CF may advance thinking in this area to determine how rumination turns toxic as opposed to remaining reflective (Treynor, Gonzalez, & Nolen-Hoeksema, 2003).

1.8.2 The Mediating Role of CF

While CF is considered a core mechanism by which our cognitions impact MH (S. C. Hayes et al., 1999), its role in the relationship between rumination and depression has not been investigated, other than in a sample of dementia caregivers (n=176; Romero-Moreno, Márquez-González, Losada, Fernández-Fernández, & Nogales-González, 2015). The authors divided participants into four groups: high rumination and high CF; high rumination and low CF; low rumination and high CF; and low rumination and low CF. CF was measured using a Spanish version of the CFQ (Romero-Moreno, Márquez-González, Losada, Gillanders, & Fernández-Fernández, 2014). Those high in both rumination *and* CF demonstrated the greatest levels of psychological distress, as compared to the three other groups. The authors concluded that considering rumination and CF *simultaneously* might develop understanding in this area.

It is worth noting, a strong correlation (r=.84; Gillanders et al., 2014) has been found between CF (measured using CFQ) and rumination (measured using the Ruminative Response Scale; Nolen-Hoeksema & Morrow, 1991), possibly questioning whether these are distinct constructs. However, Gillanders et al. (2014) demonstrated that rumination and CF loaded onto separate latent factors. Consistent with this, McCraken et al. (2014) found decentering (similar to cognitive defusion) and rumination represented independent factors that did not reflect related parts of a wider unifying psychological process. Furthermore, CF explained variance in depressive symptoms over and above that explained by

rumination and metacognition, thereby demonstrating incremental validity (Gillanders et al., 2014). Gillanders et al. (2014) concluded that while related, CF and rumination are distinct constructs. Rumination describes a particular category of repetitive negative thinking typically associated with depression. CF is not attached to particular content or frequency of thoughts, but more generally describes a transdiagnostic context in which an individual over-identifies with verbal processes such that thoughts become impenetrable and disproportionately influence emotion and action. Gillanders et al. (2014) highlighted the need to further investigate how CF relates to rumination and other variables, and their influence on mood. This would include the determination of CF and EA's unique and interrelated mediational roles in the relationship between rumination and depression.

1.9 The Relationship between Stressful Life-Events and Anxiety/Depression

1.9.1 The Mediating Role of EA

While stressful life-events are implicated in the onset and course of MH problems, they alone account for a limited amount of its variance (e.g. Kuyken & Brewin, 1994). This suggests other mediating variables are at play. The role of EA in the relationship between traumatic life-events and PTSD has received particular attention. Traumatic events can prompt painful internal experiences including re-experiencing memories of the event, heightened physiological reactivity and increased fear and anxiety. Post-trauma processing theories highlight that exposure to these difficult experiences is necessary to process and

integrate all trauma-related information into a coherent model of the self (Batten, Orsillo, & Walser, 2005; Foa & Kozak, 1986). High EA may impact upon this, impinging healthy adjustment. This is corroborated by research (Chawla & Ostafin, 2007). A large effect size was observed between EA and PTSD symptoms in a recent meta-analysis (Seligowski, Lee, Bardeen, & Orcutt, 2015) and research has found EA to be a risk factor for PTSD (Kumpula et al., 2011; Orcutt, Pickett, & Pope, 2005). Additionally, EA has mediated the relationships between childhood maltreatment and negative adult outcomes (e.g. Marx & Sloan, 2002; Polusny, Rosenthal, Aban, & Follette, 2004; Reddy, Pickett, & Orcutt, 2006; Shenk, Putnam, Rausch, Peugh, & Noll, 2014).

While most studies have concentrated on the role of EA following traumatic life-events on PTSD symptoms, a few researchers have measured anxiety and depression too. For example, EA explained significant variance in anxiety and depression in women who had been exposed to potentially traumatic events, over and above PTSD symptom severity (Tull, Gratz, Salters, & Roemer, 2004). A few studies have also used more diverse samples of individuals who have experienced a wider spectrum of stressful life-events, as opposed to the narrower categorisation of 'traumatic' events. Plumb, Orsillo and Luterek (2004) used a longitudinal design in female undergraduates to find EA predicted psychological distress following stressful life-events (e.g. academic failure, financial problems, injury/illness of a close family member) experienced during the testing period, beyond baseline distress. Additionally, Shallcross, Troy, Boland and Mauss (2010) used a prospective design with 55 female adults to

find only participants with high EA exhibited increased depressive symptoms in response to high versus low cumulative stress over 4 months.

To summarise, the literature suggests an experientially avoidant relationship with internal experiences triggered by difficult life-events leads to subsequent maladjustment. However, the majority of research has focussed on PTSD symptoms in response to narrowly defined traumatic events. Anxiety and depression are also particularly relevant outcomes in view of their association with a broader spectrum of stressful life-events. A mediating role of EA is supported by preliminary research using the AAQ, but has yet to be adequately established.

1.9.2   The Mediating Role of CF

Research investigating CF's role in the relationship between stressful life-events and psychological distress is minimal, despite being implicated in the ACT model (S. C. Hayes et al., 1999). CF with thoughts and memories triggered by difficult life-events is understood to elicit the same emotional reaction as re-experiencing the actual event itself (S. C. Hayes et al., 2001). Furthermore, CF motivates EA, which as previously discussed, may impact healthy adjustment (S. C. Hayes et al., 1999).

The aforementioned research of Dinis et al. (2015) has provided initial support for these ideas, finding unique mediating effects of both CF and EA in the relationship between traumatic memories of early-life shame experiences and

later depressive symptoms, as well as a double mediation effect (i.e. perceived impact of shame experiences→CF→EA→depressive symptoms). This preliminary research demands further attention to advance our understanding of why some people may develop symptoms of anxiety and depression in response to a broader categorisation of stressful life-events.

1.10 Summarising the Gaps in, and Limitations of, the Existing Literature

ACT is showing considerable promise in providing a helpful framework within which to understand anxiety and depression; however, there are some limitations and gaps within the literature. Our current understanding, and its limits, will next be summarised before introducing the current study's aims in attempting to address these gaps in knowledge.

ACT theory highlights the importance of one's relationship to difficult experiences in MH (S. C. Hayes et al., 1999). In support of this, EA, and to a lesser extent CF, have been associated with anxiety and depression (e.g. S. C. Hayes et al., 2006; Ruiz, 2010; Spinhoven et al., 2014; Zettle et al., 2011). ACT further suggests that part of CF's effect on MH occurs indirectly through EA (i.e. CF leads to greater EA). However, research investigating CF and EA in the context of each other is lacking, with the assumption that these two processes have both unique as well as interrelated roles in anxiety and depression not adequately explored. Some pioneering research has supported an additive effect of CF and EA in explaining psychological distress (Bardeen & Fergus, in press), as well as CF and EA operating in series (Dinis et al., 2015).

Having explored EA and CF's association with anxiety and depression, these processes have been further drawn upon to develop our understanding of how more traditional internal (worry, rumination) and external (stressful life-events) predictors of these MH conditions might operate. This shifts the focus from the content, frequency and nature of disparate vulnerabilities to psychological distress, towards considering common dysfunctional relationships one may have with these vulnerability factors (S. C. Hayes et al., 1996; Kashdan et al., 2006). Research has preliminarily supported associations between relevant constructs. However, hypotheses of mediation have not been directly tested or where they have, examined related but not identical constructs/models and neglected EA and CF's mediating role in the context of each other (e.g. Brockmeyer et al., 2015; Dinis et al., 2015; Kashdan et al., 2006; Spinhoven et al., 2016). Research investigating EA and CF within the same design would help illuminate whether one process is more important than the other, they both have unique effects, and/or they work in conjunction. Dinis et al. (2015) provided preliminary support for the ACT-based prediction that EA and CF display both unique as well as interrelated mediating properties.

It has been noted that, in the absence of any other well-validated measures, the predominant use of the poorly validated AAQ has compromised research into EA. The more recent development of the BEAQ has shown potential in addressing the AAQ's limitations (Gámez et al., 2014). Furthermore, measures of CF have been limited and those available criticised, stalling research in this area

(McCracken et al., 2014). The CFQ has provided the most promising operationalisation of CF to date (Gillanders et al., 2014). Additionally, much of the research has been restricted to non-clinical, female-only, homogenous and small samples, limiting the generalisability of results. Furthermore, cross-sectional designs have dominated. Having first established a cross-sectional relationship between variables, longitudinal designs should extend this understanding to determine temporal associations.

1.11 The Present Study

This study aimed to address the limitations of the current literature and develop our understanding of CF and EA's role in anxiety and depression in the context of one another. Firstly, this study aimed to determine whether CF and EA explained variance in anxiety and depression symptoms. Based on ACT theory (S. C. Hayes et al., 1999) and preliminary research investigating CF and EA together (Bardeen & Fergus, in press; Dinis et al., 2015), it was predicted that they would make unique contributions to explaining symptomology such that higher CF *and* higher EA would be independently related to greater anxiety and depression. Additionally, EA was predicted to partially explain a significant proportion of CF's effect. Secondly, if CF and/or EA proved to be important variables in explaining variance in anxiety and depression, this study aimed to further determine whether increased CF and EA mediated the positive relationships between worry and anxiety, rumination and depression, and stressful life-events and anxiety and depression. Again, based on ACT theory and previous research, CF and EA were predicted to uniquely mediate these relationships, independent

of one another, as well as to act in series (i.e. worry/rumination/life-events→CF→EA→anxiety/depression). This is shown in Figure 2.
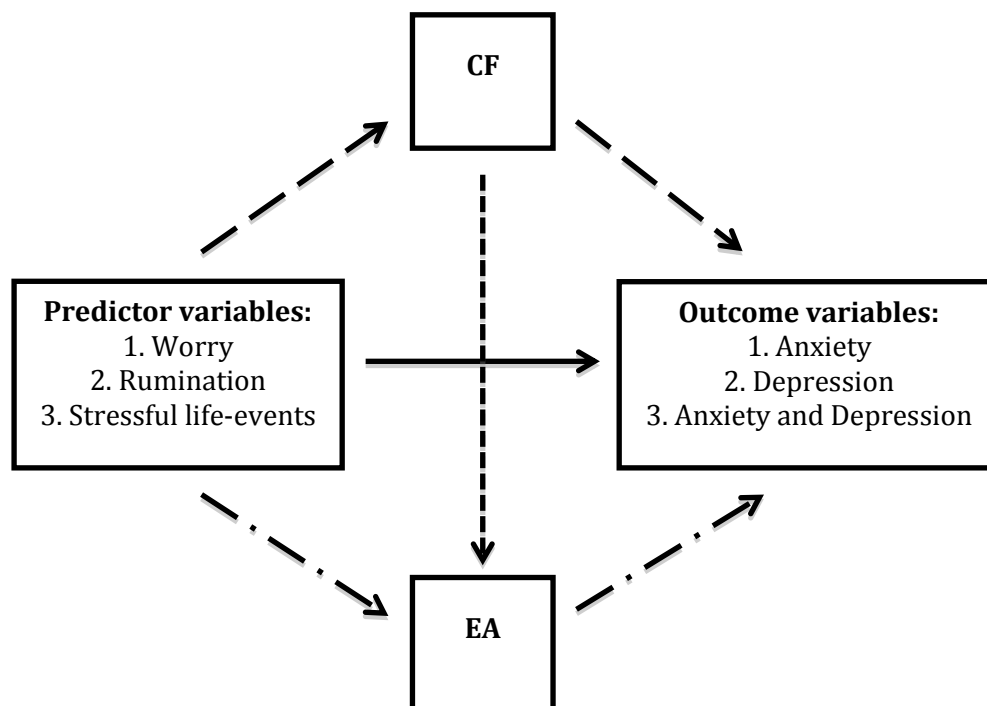
Figure 2: A Diagrammatic Representation of the Mediational Models to be Tested in Hypothesis 2.

With MH viewed on a continuum and CF and EA based on normal psychological processes (S. C. Hayes et al., 2011), CF and EA were expected to be relevant across the spectrum of experience. This study therefore investigated the research questions in two different samples likely to exhibit a diverse range of symptomology: a non-clinical student sample and a clinical sample of adults experiencing anxiety and/or depression. It should be noted that university students were recruited as a convenient means of obtaining a non-clinical

sample. As a result, caution was taken not to prematurely extend the results to non-clinical populations from the wider general population.

The study aimed to first test the research questions cross-sectionally in each sample. Next, the study aimed to test the research questions longitudinally, in other words establishing CF and EA's contribution to understanding change in anxiety and depression over time. In practice, regrettably, sufficient longitudinal data was only available in the student, and not clinical, sample as discussed in the next chapter. The research hypotheses were addressed using questionnaire methodology, with measures of EA (BEAQ, Gámez et al., 2014) and CF (CFQ, Gillanders et al., 2014) chosen due to their promising psychometrics and ability to address the shortcomings of previous measures.

To conclude this chapter, the research hypotheses (tested in each sample first cross-sectionally, and then, where sufficient data allowed, longitudinally) are summarised below.

Hypothesis 1: CF and EA will explain variance in anxiety and depression.

Three specific predictions were made:

 a) CF will explain unique variance in anxiety and depression, controlling for EA.

 b) EA will explain unique variance in anxiety and depression, controlling for CF.

 c) CF will have an additional effect on anxiety and depression, indirectly through EA.

Hypothesis 2: CF and EA will mediate the relationships between predictors

and symptoms of anxiety and depression.

Three specific predictions were made:

a) CF and EA will mediate the relationship between worry and anxiety, individually as well as in series.

b) CF and EA will mediate the relationship between rumination and depression, individually as well as in series.

c) CF and EA will mediate the relationships between stressful life-events and anxiety and depression, individually as well as in series.

CHAPTER 2: METHOD

2.1 Design

The first element of this study used a non-experimental cross-sectional design with questionnaire methodology to test the proposed hypotheses in a non-clinical sample of university students and a clinical sample of adults experiencing depression and/or anxiety disorders. Predictors of anxiety and depression (worry, rumination and stressful life-events), ACT processes (cognitive fusion, CF and experiential avoidance, EA) and anxiety and depression symptoms (DVs) were all measured at Time 1 (T1). Additionally, where possible, Time 2 (T2) measures of anxiety and depression were obtained to enable the second element of this study, which tested the hypotheses longitudinally using a repeated-measures design. Here, T2 anxiety and depression formed the DVs, with T1 symptoms controlled for. The design is summarised in Figure 3.

T1 Measures:
Worry, Rumination and Stressful Life-Events
CF and EA
Anxiety and Depression

T2 Measures:
Anxiety and Depression

Student sample - minumum of 6-weeks after T1.
Clinical sample - minumum of 2-weeks after T1.
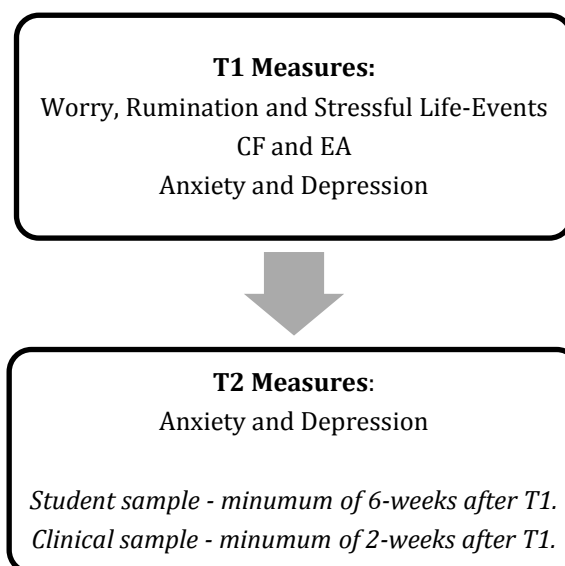
Figure 3: A Diagrammatic Representation of the Research Design

2.2   Power Calculation

Power calculations were based on the mediational analyses used to test Hypothesis 2, as these required larger samples than Hypothesis 1 due to the inclusion of an additional variable. Consensus on required sample sizes for mediational analysis is not yet established, and the best guidelines currently available are based on simple mediation models (Fritz & Mackinnon, 2007). Therefore, in the absence of clear guidance on power analyses in more complex multiple mediations, this study was constrained by the use of Fritz and Mackinnon's (2007) recommended sample sizes (power=.80, p=.05), based on estimated effect sizes (Cohen, 1992) of relationships within a simple mediation model with one mediator. Cohen's sample size recommendations for multiple regressions (Cohen, 1992) were then consulted to estimate how many extra participants would be needed to account for the additional mediating variable in this study.

Estimations of effect sizes of component relationships within our mediational models needed for the power analysis are shown in Table 1. Where directly comparable research had not been conducted, the best estimates available from the closest designed studies were used. The lack of research on CF prohibited sensible estimations of effect sizes, and so estimates were rather based on relationships with EA. This was felt appropriate given the initial validation paper of the CFQ (Gillanders et al., 2014) showed CF exhibited similar, if not larger, relationships to indices of MH as found with the BEAQ (Gámez et al., 2014). Estimations of EA's relationship with anxiety/depression were based on

research using the BEAQ, given the aforementioned limitations of the AAQ. Other estimations had to rely on data using the AAQ.

Where estimates could be made, medium to large effect sizes were found in both non-clinical and clinical samples (see Table 1). Taking a conservative estimate of medium effect sizes (given the more complex relationships in our multiple mediation models being tested), for a power of .80, bias-corrected bootstrapping mediation analyses required a sample of 71 (Fritz & Mackinnon, 2007). To further account for the additional mediating variable in this study, Cohen's (1992) sample size recommendations for multiple regressions were cross-referenced. Going from two to three predictors, Cohen (1992) recommends an extra nine participants (for a medium effect size, .80 power and p=.05). Therefore, a total sample size of 80 was recommended in both samples.

Table 1: Estimations of Effect Sizes Necessary to Calculate Sample Size Requirements.

Component relationships	Non-Clinical		Clinical *(Anxiety/Depression)*	
	Effect size	*Reference*	*Effect size*	*Reference*
EA mediates the relationship between worry and anxiety.				
Worry→EA	Large	Roemer et al. (2005)	Medium-Large	Lee et al. (2010)
EA→ Anxiety	Medium-Large	Gámez et al., (2014)	Medium-Large	Gámez et al., (2014)
EA mediates the relationship between rumination and depression.				
Rumination→EA	Medium	Kashdan et al. (2006)	Large	Spinhoven et al. (2016)
EA→ Depression	Medium - Large	Gámez et al., (2014)	Large	Gámez et al., (2014)
EA mediates the relationship between stressful life-events and anxiety and depression.				
Life-events→ EA	Medium	Shallcross et al. (2010)	*No prior research*	-
EA→Depression/Anxiety	*As above*	-	*As above*	-

2.3   Participants

2.3.1   Student Sample

One hundred and six students (92 [87%] female, 14 [13%] male) were recruited from Royal Holloway, University of London, between October 2015 and January 2016, of which 97 additionally completed longitudinal T2 data. All English-speaking students (due to unavailability of translated questionnaires) could participate. No other exclusion criteria were used. The mean sample age was

19.3 years (Standard Deviation [SD]: 2.7). Further demographic information is provided in Chapter 3, 'Results'.

2.3.2 Clinical Sample

Fifty-seven participants (42 [74%] female, 15 [26%] male) were recruited from the Centre for Psychology, Improving Access to Psychological Therapies (IAPT) service. The service sees adults, over 18 years old, experiencing symptoms of anxiety and/or depression. Individuals can be referred to the service by a health professional (usually their GP) or self-referred. The mean sample age was 42.0 years (SD=15.6). Further demographic information is provided in Chapter 3, 'Results'.

The inclusion criteria were all individuals referred to the service and on the waiting list for psychological therapy during the recruitment period of July 2015 to March 2016. Clients already receiving therapy (as this could have been a confounding factor) and clients that did not speak English were not eligible to participate. Additionally, the study was not introduced to clients if they were experiencing significant distress, and/or imminent risk issues emerged at the time when consent-to-contact would have been obtained (see Section 2.4., 'Recruitment'). This decision was based on the clinicians' clinical judgement as to the appropriateness to introduce the research at that time and capacity to consent.

T2 data was collected from questionnaires completed as part of standard practice at participants' first therapy appointment; therefore, the researcher did not have control over the timing of this. As a result, only eight of the 57 participants completed valid T2 data. Four participants disengaged from the service and 11 had not had their first appointment by the end of the data collection period. Thirty-four participants completed T2 questionnaires less than two-weeks after T1 questionnaires. This time-delay was considered too short for use (discussed in Section 2.7, 'Procedures'). As a result, regrettably the T2 sample was too small for the data to be used longitudinally.

2.4 Recruitment

2.4.1 Student Sample

Students were recruited using two methods. First-year psychology undergraduates signed up to the research in exchange for course credits. The rest of the sample signed up via an online system advertising studies to all university students. These participants were entered into a prize draw (prizes of £50, £20 and two £10). Having signed up to the research, participants accessed an online information sheet (Appendix 1) and consent form (Appendix 3) to read and complete in their own time.

2.4.2 Clinical Sample

Following a referral to the Centre for Psychology, it is standard practice that clients are offered a telephone triage (assessment) appointment with a

Psychological Needs Assessor (typically Counselling Psychology Doctorate students) to determine their care pathway. At the end of this triage assessment, if judged as appropriate by the assessor, clients were informed that a research project was running at the service and asked whether they consented to being contacted by the researcher for further information. Where time constraints prohibited 'consent-to-contact' from being discussed in this call, it was raised in a similar manner when a member of the Centre for Psychology team rang clients at a later date to give them their first appointment time.

Those consenting to be contacted by the researcher were telephoned on a prearranged date, fully informed about the study and given the opportunity to ask questions. Those still interested then received a written information sheet (Appendix 2) and consent form (Appendix 4), via post or email (as preferred), to read in their own time before documenting their consent.

Consenting participants' GPs were informed of their participation by letter (Appendix 5). Participants were entered into a prize draw (prizes of £50, £20 and two £10).

The recruitment process and procedure for both samples is summarised in Figure 4.
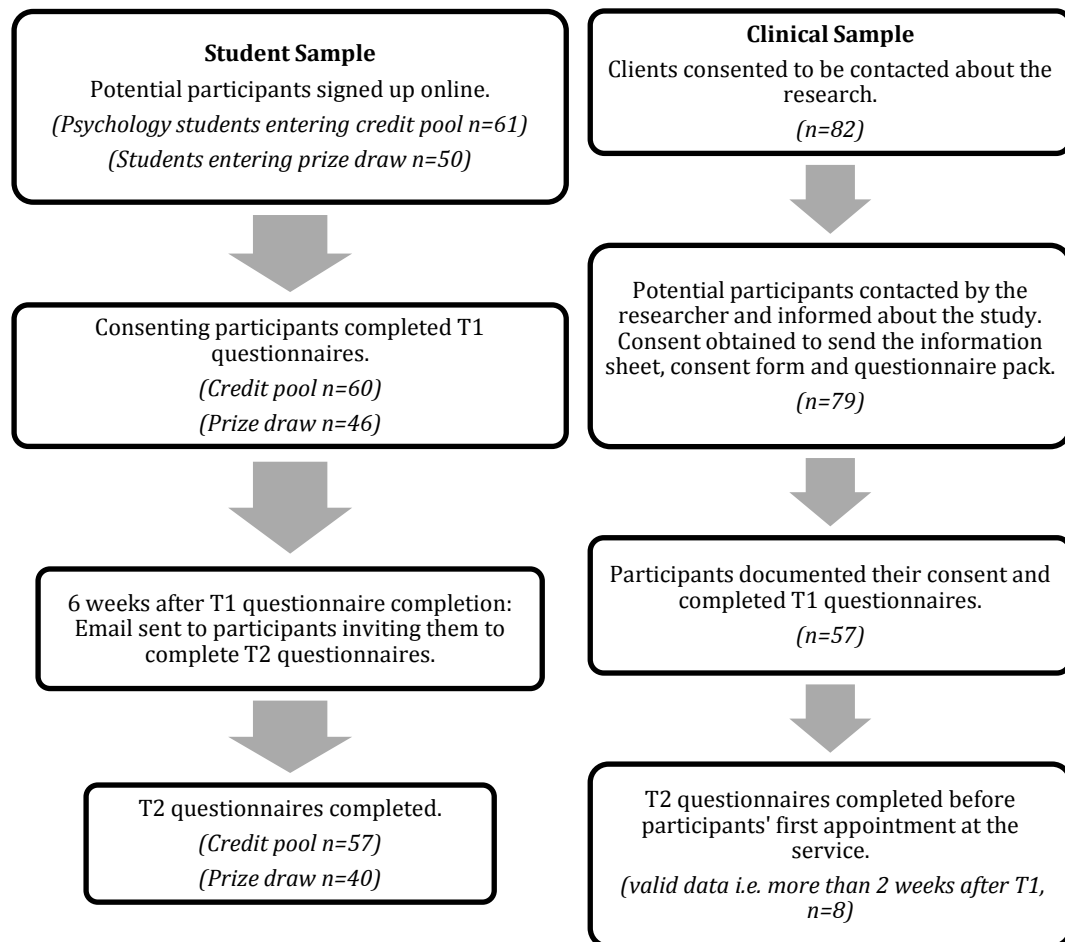
Figure 4: Recruitment Process and Procedure

2.5 Measures

Similar measures were used in the student and clinical samples, with the following exceptions. A different measure of stressful life-events was used with students to better reflect the life-events they may experience. Anxiety and depression measures used in the clinical sample were questionnaires already utilised by the Centre for Psychology, which have cut-off scores to indicate the presence and severity of clinical anxiety and depression. In the student sample, an alternative, more appropriate measure was selected that takes a dimensional

approach to anxiety and depression and has been well-validated in students. Questionnaires were designed to provide demographic information specific to each sample. The measures used are described below.

Demographic Questionnaire

Student Sample (Appendix 6)

Participants were asked their gender, age, ethnicity, year of study, subject studied and whether they had a history of MH problems.

Clinical Sample (Appendix 7)

Participants were asked their gender, age, ethnicity, highest level of education achieved and current employment status.

Penn State Worry Questionnaire *(PSWQ, Meyer, Miller, Metzger, & Borkovec, 1990); Appendix 8.*

The PSWQ is a well-validated and widely used, unidimensional trait measure of worry. Sixteen items are rated on a 5-point Likert scale, ranging from "1" (not at all typical of me) to "5" (very typical of me). The PSWQ has evidenced good psychometric properties. A good internal consistency (α=.93-.95) and test–retest reliability (r=.93) has been found in student and clinical samples (Brown, Antony, & Barlow, 1992; Meyer et al., 1990). Cronbach's alpha in the present study was .94 (student sample) and .87 (clinical sample). The questionnaire has also demonstrated good construct validity, correlating with psychological measures related to worry and not measures distinct from the construct, and

successfully discriminating between college samples that met none, some or all of the diagnostic criteria for GAD (Meyer et al., 1990).

Ruminative Response Scale of the Response Styles Questionnaire *(RRS, Nolen-Hoeksema & Morrow, 1991, 1993); Appendix 9.*

The RRS is a 22-item questionnaire commonly used to assess the tendency to engage in ruminative thinking, using the 4-point scale of "1" (almost never) to "4" (almost always). The RRS has shown good internal consistency (α=.90) and test re-test reliability (r=.67) in a community sample (Treynor et al., 2003) as well as convergent and predictive validity, predicting prospective episodes of major depression (Nolen-Hoeksema, 2000; Nolen-Hoeksema & Morrow, 1991, 1993). Internal consistency in the present study was also good (α=.93, student sample; α=.88, clinical sample).

Life Events Scale for Students *(LESS, Clements & Turpin, 1996; Linden, 1984); Student Sample, Appendix 10.*

The LESS is an adapted version of the life-events measure used in the clinical sample (see below), for use with university students. It contains 36 stressful life-events students may experience (e.g. death of a parent, failing a course, break-up with boy/girlfriend). The scale was originally developed in Canadian populations (Linden, 1984) and subsequently validated for use with British students (Clements & Turpin, 1996). Life-event ratings capturing the relative amount of change required following each life-event have been derived (maximum item rating=100; Clements & Turpin, 1996). Participants indicate whether they have

experienced any of the listed events in the last year. Values for each event indicated are summated to obtain an overall score (maximum score: 1849). The scale has demonstrated acceptable test-retest reliability and good construct validity, with greater levels of stressful life-events associated with greater psychological distress (Clements & Turpin, 1996).

Social Readjustment Rating Scale *(SRRS, Holmes & Rahe, 1967); Clinical Sample, Appendix 11.*

The SRRS (Holmes & Rahe, 1967), used in the clinical sample, consists of 43 commonly reported stressful life-events requiring change to on-going life (e.g., death of a spouse, marital separation, change in work). Holmes and Rahe (1967) used a convenience sample to rate the relative degree of readjustment necessary for each life-event (maximum item rating=100). The relative weightings have been subsequently re-evaluated (Scully, Tosi, & Banning, 2000) and these more recent ratings were used in this study. Participants indicate whether they have experienced any of the listed events in the last year. Values for selected events are summated to obtain an overall score (maximum score: 1214). The SRRS is one of the most widely cited questionnaires in stress literature (Scully et al., 2000) and, in support of the scale's construct validity, is significantly associated with psychological and physiological symptoms of stress (Scully et al., 2000).

Cognitive Fusion Questionnaire *(CFQ, Gillanders et al., 2014); Appendix 12.*

The CFQ is a seven-item measure of CF. The respondent indicates how true they believe items are on a seven-point scale, ranging from "1" (never true) to "7"

(always true). The CFQ's psychometric properties have been tested across non-clinical and clinical samples (Gillanders et al., 2014), with good internal consistency (α=.88-.90) and test-retest reliability (r=.80). Internal consistency in the present study was good (α=.91, student sample; α=.89, clinical sample). In support of its construct validity, Gillanders et al. (2014) found the CFQ was significantly associated with related constructs, including measures related to the ACT model (e.g. AAQ-II), constructs related to CF (e.g. trait mindfulness and thought control strategies) and outcomes including distress, anxiety, depression and quality of life. Incremental validity was also demonstrated by the CFQ adding to variance explained by well-established predictors of key outcomes (Gillanders et al., 2014).

Brief Experiential Avoidance Questionnaire *(BEAQ, Gámez et al., 2014);*
Appendix 13.

The BEAQ is a 15-item measure of EA. Participants indicate the extent to which they agree with statements using a six-point scale, ranging from "1" (strongly disagree) to "6" (strongly agree). It has shown good internal consistency (α=.83 [clinical sample], .80-.86 [student sample]; Gámez et al., 2014). In the current sample, Cronbach's alpha was .81 (student sample) and .66 (clinical sample), demonstrating a 'good' and 'acceptable' (P. Kline, 2013) internal consistency, respectively. Construct validity has also been exhibited. The BEAQ showed expected associations with a range of well-validated measures of avoidance, psychopathology and quality of life and was distinguishable from negative affectivity and neuroticism (Gámez et al., 2014).

Depression Anxiety Stress Scale *(DASS-42, S. Lovibond & Lovibond, 1996);*

Student Sample, Appendix 14.

The DASS is a 42-item questionnaire assessing symptoms of depression, anxiety and stress over the past week. Each sub-scale includes 14 items, rated on a 4-point scale ranging from "0" (did not apply to me) to "3" (applied to me very much/most of the time). Only the depression and anxiety scales were used in this study's analysis. The depression and anxiety scales have shown good internal consistency in a non-clinical sample (α=.95, .90 respectively; Crawford & Henry, 2003) and acceptable test-retest reliability in a clinical sample (r=.71, .79 respectively; Brown, Chorpita, Korotitsch, & Barlow, 1997). Cronbach's alphas in the present study were .96 (Depression Scale, T1), .97 (Depression Scale, T2) and .89 (Anxiety Scale, T1 and T2). Convergent and discriminant validity have been demonstrated in student samples (P. F. Lovibond & Lovibond, 1995), with high correlations between the DASS Anxiety Scale and Beck Anxiety Inventory (Beck & Steer, 1990; r=.81), and between the DASS Depression Scale and Beck Depression Inventory (Beck & Steer, 1987; r=.74). Cross correlations were substantially lower, as expected.

Generalised Anxiety Disorder Assessment *(GAD-7, Spitzer, Kroenke, Williams, & Löwe, 2006); Clinical Sample, Appendix 15.*

The GAD-7 is a brief self-report measure originally designed to assess GAD. However, it is used as the standard means of assessing anxiety more generally in IAPT services, including the Centre for Psychology, given it also captures other common anxiety disorders, including panic disorder, social anxiety disorder, and

PTSD (IAPT National Programme Team, 2011). Its use as a general measure of anxiety is supported by its strong correlations with other well-validated anxiety questionnaires (Spitzer et al., 2006).

Respondents indicate how much seven items have "bothered" them over the last two-weeks on a four-point scale, ranging from "0" (not at all) to "4" (nearly every day). This provides a continuous measure of symptom severity. Cut-off scores can give categorical descriptions of severity, however these were not used for this research. The GAD-7 is a well-validated and reliable tool (Spitzer et al., 2006), demonstrating excellent internal consistency (α=.92) and good test-retest reliability (r=.83) in a sample recruited from primary care services. A good internal consistency was also found in the present sample (α=.87). Construct validity has also been documented, with strong associations between increasing GAD-7 scores and other anxiety measures, worsening functional impairment, self-reported disability days, clinic visits and symptom-related difficulty (Spitzer et al., 2006).

Patient Health Questionnaire *(PHQ-9, Kroenke, Spitzer, & Williams, 2001);*

Clinical Sample, Appendix 16.

The PHQ-9 is a nine-item measure of depression, based on the DSM-IV diagnostic criteria (American Psychiatric Association, 2013). It is the standard means of assessing depression in IAPT services and routinely used in the Centre for Psychology. Respondents rate how much each item has "bothered" them over the last two-weeks on a four-point scale, ranging from "0" (not at all) to "3" (nearly

everyday). Like the GAD-7, this questionnaire provides a continuous measure of symptom severity as well as categorical descriptions (not used for this research). The PHQ-9 has been well-validated (Kroenke et al., 2001), showing good internal consistency (α=.89) and test-retest reliability (r=.84) in a sample recruited from primary care services. A good internal consistency was also found in the present sample (α=.85). Construct validity has been demonstrated by associations with functional impairment, symptom-related difficulty, sick days, and health care utilisation (Kroenke et al., 2001). Likelihood ratios exhibited a substantial association between increasing PHQ-9 scores and likelihood of major depression (Kroenke et al., 2001).

2.6 Service-User Consultation

Face-to-face interviews with three service-users who had recently completed treatment from the Centre for Psychology provided consultation on the study design and procedures in the clinical sample. The student study was based on the clinical design to maintain consistency, with procedural adaptions made where necessary. Feedback was obtained in the following areas:

Recruitment of the Clinical Sample

Service-users highlighted that at the initial contact with the service (the triage call), individuals are likely to feel distressed and overloaded with information. Therefore, a full discussion about what was involved in the research would not have been appropriate. Rather, they preferred the method of first obtaining consent-to-contact followed by a later telephone conversation with the

researcher giving the opportunity to ask questions. They felt the written information sheet was then helpful to read in one's own time, before documenting written consent.

Questionnaire Completion

Service-users highlighted that completing questionnaires can sometimes be daunting and therefore thought offering telephone assistance would be valued. As the BEAQ and CFQ are relatively new compared to the other questionnaires in our study, these were shown to service-users to comment on how they would feel completing them. Service-users did not feel that the questions would be particularly distressing or intrusive to complete.

Participant Documents

Service-users provided feedback on the content and clarity of the information sheet, consent form and debrief form.

Additional Consultation

The completion of the questionnaires (online and on paper) was additionally piloted by a convenience sample of community adults to comment on their length and delivery. Participants from the clinical sample also provided consultation on the writing of the final report before it was circulated.

2.7 Procedures

2.7.1 Student Sample

Students signed up to complete the study online. This provided them with a link to a secure survey website, where they read and completed the information and consent form, followed by the T1 questionnaires (taking approximately 15-minutes). Questionnaire order was informed by recommendations from within mediational analysis. Kenny (2014) suggests the temporal order of questionnaires should measure the mediators after the IV, and the DV after the mediators, even when using cross-sectional designs. Therefore, questionnaires were presented in the following order: questionnaires assessing IVs (worry, stressful life-events and rumination), followed by those assessing mediators (CF and EA) and finally the anxiety and depression questionnaire (DVs).

Six-weeks after completing T1 questionnaires, participants received an email inviting them to complete the T2 questionnaire online, meaning six-weeks formed the minimum time elapsed post T1 (average: 48 days; range=42-97). This time-delay was selected to replicate previous longitudinal research in this area (e.g. Bardeen, Fergus, & Orcutt, 2014; Bjornsson et al., 2010) and to reflect the hypothesised time-delay in the clinical sample, which was dictated by the expected length of the waiting list for therapy.

Upon completion of the study, participants could read the debrief form (Appendix 17) and were invited to receive a copy of the final report (Appendix 19).

2.7.2 Clinical Sample

Participants were sent the questionnaires at the same time as the information sheet and consent form (T1). Questionnaires were sent and returned via post (in a prepaid envelope) or completed on a secure online survey website, as preferred. Questionnaires took approximately 20-minutes to complete. The questionnaire order was as specified in the student sample. Additionally, the GAD-7 was completed last, as opposed to the PHQ-9, so that participants did not end with the potentially more distressing question assessing suicidal and self-harm thoughts. Participants were offered additional telephone assistance by the chief researcher when completing the questionnaires. In view of the longitudinal component of the design, if participants had not completed the questionnaires within a week, a reminder email/letter was sent. All T1 questionnaires were completed prior to a participant's first session at the service.

Some time after their triage call, clients attend their first appointment at the service and complete GAD-7 and PHQ-9 questionnaires in the waiting room beforehand as part of standard practice. These questionnaires are therefore *not* additional measures just for the research but completed by all clients at the service. Participants gave their consent for their responses to be used for the research, forming the T2 data. The time-delay between T1 and T2 questionnaire completion was estimated to be 1-3 months, based on the average service waiting time between clients' triage and first appointment audited when the study was designed. However, in practice, waiting times were much shorter and

varied considerably during the running of the study (time-delay range: 0-51 days), due to unforeseen staff and service changes. Where less than two-weeks had elapsed between T1 and T2 questionnaire completion, T2 data was not used. The GAD-7 and PHQ-9 ask about symptoms over the past *two*-weeks and therefore would not have been sensitive to change over shorter periods. This was especially relevant when waiting-list times were short and where consent-to-contact had to be obtained when clients were given their first appointment time, and not the initial triage call. In practice, insufficient T2 data was obtained for analysis.

Following participation, participants were sent a debrief letter (Appendix 18) and invited to receive the final report of the results (Appendix 19).

The recruitment process and procedure for both samples is summarised in Figure 4, presented earlier in this chapter.

2.8 Ethical Considerations

2.8.1 Student Sample

We did not anticipate that completing the questionnaires posed significant risk or harm to consenting participants; however, the questionnaires did enquire about potentially distressing topics. Additionally, questionnaires were completed anonymously (using a unique identification code) and remotely for participants' convenience, not requiring them to travel. This meant the

researcher was not privy to participants' feelings and reactions to questions (as discussed in the internet-mediated research guidelines by the British Psychological Society, 2013). Therefore, it was clearly stated in the information sheet and debrief form where students could access support should they be concerned about their mood. The internet-mediated research guidelines (British Psychological Society, 2013) further informed the process of obtaining valid informed consent, maintaining confidentiality, debriefing and the right to withdraw. Participants were informed how data was securely stored.

Ethical approval was granted by the RHUL Departmental Ethics Committee (reference: 2015/113; Appendix 20).

2.8.2   Clinical Sample

This study recruited treatment-seeking adults experiencing symptoms of anxiety and/or depression, who by nature are a potentially vulnerable group. Furthermore, clients were first approached about the study during their triage session (a 20-minute discussion of current difficulties), a potentially vulnerable time. This might have lead participants to feel coerced into opting in. It was therefore made clear at that point that participation was voluntary and would not impact their treatment at the service. In addition, there were a number of opportunities for clients to think about their participation before making a decision. Only clients opting to hear more about the study were contacted. The researcher then explained the study in detail, allowing the opportunity for questions. This information was reiterated in an information sheet, prior to

documenting consent. Participants' right to withdraw from the study at any time was made explicit.

The PHQ-9 could potentially reveal information regarding risk, asking about thoughts of suicide and self-harm. Participants were aware that clinicians had access to information from the PHQ-9 and GAD-7 completed as part of standard practice (i.e. upon initial referral, assessment and each treatment session) and would act upon any risk concerns themselves following standard service protocol. However, T1 questionnaires were not given to clinical staff as these were only completed for the research and this would have compromised anonymity and confidentiality. Additionally, as with the student sample, T1 questionnaires were completed remotely for participants' convenience. We therefore requested in the information sheet and debrief form that participants contact the service if any of the questionnaires caused them distress or concern about their wellbeing. If this occurred, standard service procedures were employed, including giving information about where to gain additional 24-hour support if in a crisis. Furthermore, all participants were, by virtue of the invitation, shortly receiving therapy from the service.

Additionally, internet-mediated research guidelines (British Psychological Society, 2013) were consulted, with the same considerations made as the student sample. All participants had the choice of completing questionnaires by post as well as online and were offered telephone assistance.

Full ethical approval was granted by the Brent London Research Ethics Committee (REC, reference 15/LO/0707; Appendix 21) and the RHUL Departmental Ethics Committee (reference: 2015/113; Appendix 20). As the clinical research site was a private social enterprise service commissioned by GPs, while the clients accessing the service were NHS clients, the site itself was not. The service is self-insured and not covered by NHS insurance policies. Therefore, upon consultation with the service manager, Surrey & Borders Research and Development (R&D) Department, Health Research Authority Queries line and Surrey & Borders local REC department, it was advised that NHS R&D approval was not applicable or needed. This was made explicit and approved by the Brent REC.

CHAPTER 3: RESULTS

3.1 Chapter Overview

This chapter will first outline the data analysis plan, detailing the main statistical methods used to test each hypothesis as well as the general conventions adhered to. Next, the data screening process will be described and the socio-demographic and clinical characteristics of each sample presented. Finally, the main cross-sectional statistical analyses will be reported for the student and clinical samples in turn, followed by the longitudinal analysis conducted in the student sample.

3.2 Data Analysis Plan

The data was analysed using the Statistical Package for Social Sciences (SPSS), version 21. Statistics are reported to two decimal places, other than for the bootstrapping analysis coefficients, which are reported to three decimal places as recommended by A. F. Hayes (2013), as these required a greater degree of precision to interpret. P-values are reported to two decimal places throughout, with $p \leq .05$ indicating statistical significance. While multiple analyses can increase Type I errors (i.e. detecting an effect that is not there), adjusting for this (for example, using Bonferroni corrections) can have a reverse effect and place the risk of Type II errors (i.e. failing to detect an effect that exists) at unacceptable levels (Nakagawa, 2004). This is especially true in underpowered studies. Rather, Nakagawa (2004) suggested reporting confidence intervals (CIs) for all effects, as done in the present study.

All hypotheses were tested by mediational analyses using multiple ordinary least squares regressions with bootstrapping (Preacher & Hayes, 2004, 2008). Bootstrapping is a non-parametric method, using repeated random resampling. Confidence intervals (CIs) are calculated and if zero is not in the 95% CI, the effect is significantly different from zero at p=.05. An indirect effect with a CI not crossing zero signifies mediation has occurred. Five thousand bootstrapping resamples are recommended by Preacher and Hayes (2008) and presently used, as well as bias-corrections of the CIs (Efron & Tibshirani, 1993). Mediation analyses were conducted using PROCESS (A. F. Hayes, 2013), a computational procedure for SPSS. A. F. Hayes' (2013) conventions for reporting mediations are used. Mediation effects are reported in unstandardised form, as is the norm and recommendation (A. F. Hayes, 2013). Some studies additionally report the proportion of the effect of the IV on the DV that operates indirectly through the mediator. However, this is not recommended (and therefore not presently used) in sample sizes less than 500 given this statistic has large sampling variance and is very unstable (A. F. Hayes, 2013).

This analytic approach is preferable to traditional tests of mediation, such as Baron and Kenny's (1986) causal steps approach and Sobel's test (Sobel, 1982). Bootstrapping makes no assumptions about the sampling distribution and obtaining a CI better accounts for irregularities (A. F. Hayes, 2013). Bootstrapping mediation is also a higher-powered test, with reduced chance of Type I and II errors (A. F. Hayes, 2013).

While mediation inherently tests causal explanations, A. F. Hayes (2013) advocates that mediation analysis is still useful even when causality cannot be established due to the research design. In these situations, a strong theoretical argument and acknowledgement of the difficulties of inferring causality must be clarified (A. F. Hayes, 2013). Such precautions were taken in the present study. While reporting mediation analyses uses language with causal inferences, it is with awareness that these results can only be supportive of, and are *not* able to conclude, causality without true experimental designs.

Next, the specifics of this analysis plan will be outlined for each hypothesis, to be tested cross-sectionally in both samples. After this, the longitudinal analysis plan will be described.

Hypothesis 1: CF and EA will explain variance in anxiety and depression.

Three specific predictions were made:

a) CF will explain unique variance in anxiety and depression, controlling for EA.

b) EA will explain unique variance in anxiety and depression, controlling for CF.

c) CF will have an additional effect on anxiety and depression, indirectly through EA.

This was analysed using two simple mediation analyses (PROCESS model 4, A. F. Hayes, 2013; Preacher & Hayes, 2004); with CF as the IV, EA as the mediator, and 1) anxiety and 2) depression, as the DVs (see Figure 5). All variables were

measured at T1. The first prediction (CF's unique effect on anxiety/depression) was represented in path c' which calculates the direct relationship between CF and anxiety/depression, *controlling for EA*. The second prediction (EA's unique effect on anxiety/depression) was represented in path b, which calculates the relationship between EA and anxiety/depression, *controlling for CF*. The third prediction (EA mediating the relationship between CF and anxiety/depression) was represented in the indirect effect (ab), which is calculated from the product of path a (the relationship between CF and EA) and path b.
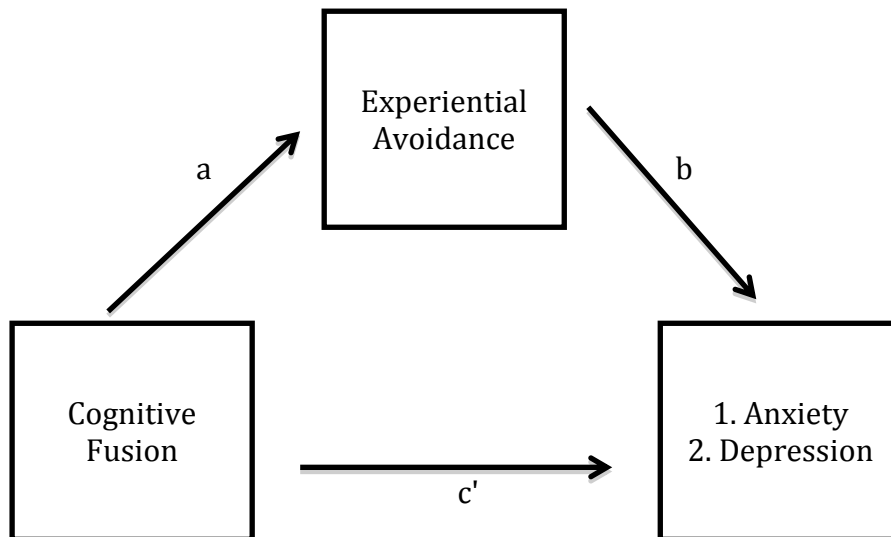
Figure 5: The Simple Mediation Models Used to Test Hypothesis 1

Hypothesis 2: CF and EA will mediate the relationships between predictors and symptoms of anxiety and depression.

Three predictions were made:

a) CF and EA will mediate the relationship between worry and anxiety, individually as well as in series.

b) CF and EA will mediate the relationship between rumination and depression, individually as well as in series.

c) CF and EA will mediate the relationships between stressful life-events and anxiety and depression, individually as well as in series.

These predictions were tested using four serial multiple mediation analyses (PROCESS model 6, A. F. Hayes, 2013) with 1) worry, 2) rumination and 3&4) life-events as the IV; CF and EA as the mediators; and 1&3) anxiety and 2&4) depression as the DVs. All variables were measured at T1. Serial multiple mediation was currently favoured over the more traditionally used parallel multiple mediation as it does not assume that the mediators are not causally related (A. F. Hayes, 2013). This was important given that ACT theory predicts a causal relationship between CF and EA. The analysis calculates the indirect effects of the IV on the DV through CF and EA separately, *controlling for the other*, as well as the indirect effect through CF and EA in series (i.e. double mediation; IV→CF→EA→DV). The paths analysed in these models are shown in Figure 6. As in the simple mediation analysis, the unique indirect effects are calculated from the product of the corresponding paths a (the relationship between IV and mediator) and b (the relationship between mediator and DV, controlling for IV),

with both paths controlling for the influence of the other mediator. The double mediation computes the serial path where the IV affects CF, which in turn affects EA, which in turn affects the DV (path $a_1a_3b_2$). The direct effect of the IV on DV, holding constant the two mediators, is also calculated (c').
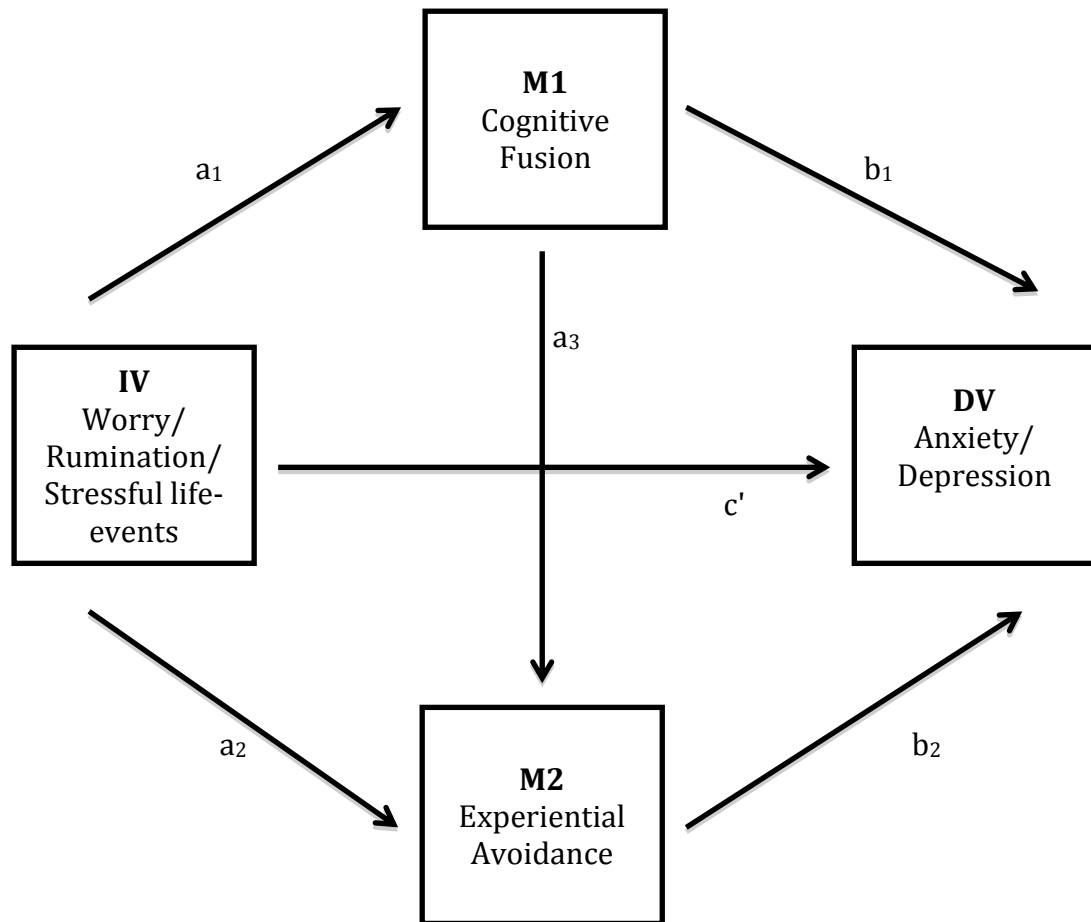
Figure 6: Model Paths Tested in the Serial Multiple Mediation Models.

Longitudinal Analyses

Where data was available to test the hypotheses longitudinally, the same analyses were conducted as detailed in the cross-sectional analysis plan above,

however the DV was instead T2 anxiety/depression, with T1 symptoms controlled for.

3.3   Data Screening

Prior to carrying out the main analyses, all data were checked for input errors and missing values. More than 95% of both samples responded on all items and participants missed a small number of items (<10%) for any one questionnaire. The distribution of missing responses was also observed to be random (using Little's Missing Completely at Random test; R. J. Little, 1988). Therefore, the sample mean could be used in place of missing values (Tabachnick & Fidell, 2007). The only exception to this was one participant in the clinical sample that did not complete the RRS and so was not included in relevant analyses.

The sample was checked for extreme univariate scores (more than three SDs from the mean, Field, 2013) and multivariate scores (using Mahalanobis Distance calculations, Tabachnick & Fidell, 2007). In both samples, no extreme scores were observed to represent outliers in the population from which the sample was obtained. The normality of the distributions of variable scores was evaluated by inspecting histograms with normal curves and using standard indices of skewness and kurtosis. These were calculated using the following formulae:

$$Z_{skewness} = \frac{Skew}{SE^*_{skewness}} \qquad\qquad Z_{kurtosis} = \sqrt{\frac{Kurtosis}{SE^*_{kurtosis}}}$$

SE=Standard Error

A distribution was considered normal if z-scores for both skewness and kurtosis were less than 2.58 (p>.01, Field, 2013). In the student sample, variable distributions were normal other than the LESS (Z_{skew}=9.23, $Z_{kurtosis}$=4.45), DASS Depression at T1 (Z_{skew}=5.32) and T2 (Z_{skew}=4.32), and DASS Anxiety at T1 (Z_{skew}=3.74) and T2 (Z_{skew}=3.67). These were all positively skewed. Considering this was a non-clinical sample, it was unsurprising that most people fell within the lower end of the anxiety and depression scales with a few people representing higher scores. Square root transformations (Tabachnick & Fidell, 2007) resulted in normal distributions with acceptable levels of skew and kurtosis. Transformed variables were only used when examining correlations between variables, as bootstrapping analyses do not assume normality (Preacher & Hayes, 2004). In the clinical sample, all variables were normally distributed.

3.4 Demographic Information and Descriptive Statistics

3.4.1 Student Sample

One hundred and six participants completed T1 data. Of these, 97 completed T2 longitudinal data. The majority of the sample were female (86.8%) and of white ethnicity (69.0%), with a mean age of 19.3 years (Standard Deviation [SD]: 2.7). Further demographic information is displayed in Table 2.

Table 2: Demographic Information for the Student and Clinical Samples

	Student *N*=106	Clinical *N*=57
Gender: N (%)		
Female	92 (86.8)	42 (73.7)
Male	14 (13.2)	15 (26.3)
Age in Years: Mean (SD)	19.3 (2.7)	42.0 (15.6)
Ethnicity: N (%)		
White	73 (69.0)	53 (93.0)
Asian/Asian British	17 (16.0)	2 (3.5)
Mixed/Multiple Ethnic Groups	9 (8.5)	0 (-)
Black/ African/ Caribbean/ Black British	5 (4.7)	0 (-)
Other	1 (0.9)	1 (1.8)
Blank	1 (0.9)	1 (1.8)
Employment Status: N (%)		
Employed Full-Time	-	26 (45.6)
Employed Part-Time	-	10 (17.5)
Retired	-	7 (12.3)
Unemployed	-	4 (7.0)
Student	-	2 (3.5)
Other	-	4 (7.0)
Blank	-	4 (7.0)
Education: N (%)		
No Academic Qualifications	-	6 (10.5)
GCSEs or Equivalent	-	19 (33.3)
A-levels or Equivalent	-	11 (19.3)
Undergraduate Degree	-	6 (10.5)
Postgraduate Certificate or Diploma	-	5 (8.8)
Master's Degree	-	4 (7.0)
PhD or Doctoral Level	-	1 (1.8)
Other	-	3 (5.3)
Blank	-	2 (3.5)

Year of Study: N (%)		
First	81 (76.4)	-
Second	17 (16.0)	-
Third	5 (4.7)	-
Masters	2 (1.9)	-
Blank	1 (0.9)	-
Course Studied: N (%)		
Psychology	77 (72.6)	-
Sciences	7 (6.6)	-
Geography	4 (3.8)	-
Drama/Theatre	3 (2.8)	-
English	2 (1.9)	-
History	2 (1.9)	-
Languages	1 (0.9)	-
Other	9 (8.5)	-
Blank	1 (0.9)	-
History of MH Difficulties **N (%)**		
Yes	17 (16.0)	-
No	86 (81.1)	-
Prefer Not to Answer	3 (2.8)	-

Table 3 reports means, SDs and SEs for all variables in the student sample. Table 3 also reports Pearson's correlations, supporting significant positive relationships between predictors of anxiety (worry, stressful life-events) and anxiety symptomology and between predictors of depression (rumination, stressful life-events) and depressive symptomology. Additionally, while not the focus of this study, significant relationships were also found between worry and depression, and rumination and anxiety. As expected, increasing CF and EA were also significantly related to each other, heightened anxiety and depression, and

predictors of anxiety and depression. The only relationship expected to be significant that was in fact not, was between stressful life-events and EA (r=.17, p=.08); however, this still demonstrated a trend towards significance.

Tabachnick and Fidell (2007) advised caution before including two variables with a correlation greater than .70 in the same analysis. Where this was the case between predictor variables, Variance Inflation Factors (VIF; Montgomery & Peck, 1992) were calculated. VIFs quantify the severity of multicollinearity between predictor variables in regression analyses. Given some degree of multicollinearity is unavoidable in mediation (Kenny, 2014), a VIF lower than 10 is deemed acceptable (Myers, 1990). A particularly high correlation was found between RRS and CFQ (r=.75); however, the VIF (2.31) indicated multicollinearity was not a large concern. Additionally, a high correlation between RRS and T1 DASS Depression (r=.71) possibly indicated overlap between these measures. Therefore, to err on the side of caution, a subset of items from the RRS was used in a secondary exploratory analysis, having removed confounding content with items measuring depression (Treynor et al., 2003); discussed in more detail in Section 3.5, 'Main Findings: Cross-Sectional Hypotheses Testing'.

Table 3: Means, SDs, SEs of Mean and Correlations for Measures in the Student Sample.

	Mean	SD	SE	1	2	3	4	5	6	7	8	9
							Correlations					
1. PSWQ	55.03	13.96	1.36	-								
2. RRS	48.12	13.39	1.30	.45***	-							
3. LESS	291.81	179.94	17.48	.07	.39***	-						
4. BEAQ	50.48	11.24	1.09	.25*	.51***	.17	-					
5. CFQ	28.03	9.25	0.90	.58***	.75***	.28**	.52***	-				
6. T1 DASS Depression	9.82	10.36	1.01	.28**	.71***	.41***	.48***	.64***	-			
7. T2 DASS Depression	11.21	11.44	1.16	.29**	.58***	.27**	.32***	.54***	.67***	-		
8. T1 DASS Anxiety	8.46	7.15	0.69	.40***	.53***	.27**	.44***	.58***	.59***	.46***	-	
9. T2 DASS Anxiety	8.30	7.37	0.75	.33***	.45***	.25*	.27**	.45***	.44***	.68***	.62***	-

* p<.05, **p<.01, ***p<.001.

3.4.2 Clinical Sample

Fifty-seven participants completed T1 data and eight completed valid T2 data. Given the minimal T2 data, and therefore sub-optimal power, longitudinal analyses were not viable. Demographic information is displayed in Table 2. The majority of the sample were female (73.7%) and of white ethnicity (93.0%). The mean age was 42.0 years (SD=15.6).

Table 4 reports means, SDs, SEs and Pearson's correlations for all variables. Fifty-two participants (91.2%) had clinically significant symptoms of anxiety and/or depression (IAPT National Programme Team, 2011). The other five participants displayed 'mild' symptoms (Kroenke et al., 2001; Spitzer et al., 2006). Statistically significant positive relationships existed between predictors of anxiety (worry, stressful life-events) and anxiety symptomology, and between predictors of depression (rumination, stressful life-events) and depressive symptomology. While not the focus of this research, rumination and anxiety were also significantly related. As expected, increasing CF and EA was significantly related to each other and to heightened anxiety and depression. While CF was significantly associated with all predictors of anxiety and depression, EA was only significantly associated with rumination. None of the correlations were greater than .70, indicating multicollinearity was not of great concern.

Table 4: Means, SDs, SEs of Mean and Correlations for Measures in the Clinical Sample.

| | Mean | SD | SE | Correlations | | | | | | |
				1	2	3	4	5	6	7
1. PSWQ	61.70	10.20	1.35	-	-	-	-	-	-	-
2. RRS	57.28	10.79	1.44	.19	-	-	-	-	-	-
3. SRRS	173.09	98.44	13.03	.20	.23	-	-	-	-	-
4. BEAQ	56.78	9.44	1.25	.11	.51***	.23	-	-	-	-
5. CFQ	35.25	7.08	0.94	.31*	.53***	.36**	.58***	-	-	-
6. PHQ-9	14.09	5.95	0.79	.07	.66***	.43***	.58***	.44***	-	-
7. GAD-7	12.47	5.17	0.69	.36**	.38**	.45***	.49***	.51***	.65***	-

* $p<.05$, ** $p<.01$, *** $p<.001$.

3.5 Main Findings: Cross Sectional Hypotheses Testing

3.5.1 Student Sample

Hypothesis 1: CF and EA will explain variance in anxiety and depression.

Two mediation analyses were performed (IV: CF, Mediator: EA and DV: Anxiety/Depression). All variables were measured at T1.

As expected, higher levels of CF was significantly associated with higher levels of EA (a=.629, p<.001). More importantly and contrary to predictions, EA was not uniquely related to anxiety (b=.071, p=.24), or depression (b=.159, p=.06) when controlling for CF. EA also did not mediate the relationships between CF and anxiety (ab=.044, CI -.020 to .137) or depression (ab=.100, CI -.017 to .246), with bias-corrected bootstrap CIs crossing zero. However, as predicted, CF showed a unique direct effect on anxiety (c'=.397, p<.001) and depression (c'=.573, p<.001) that was independent of EA. Model summary information showed that CF and EA together explained 33% and 38% of variance in anxiety and depression respectively.

This partially supported Hypothesis 1. Higher CF was uniquely predictive of higher levels of anxiety and depression. However, contrary to expectations, EA did not significantly mediate this relationship, nor explain unique variance in anxiety and depression.

Hypothesis 2: CF and EA will mediate the relationships between predictors

and symptoms of anxiety and depression.

Serial multiple mediations tested whether CF and EA mediated the relationships between a) worry and anxiety, b) rumination and depression, c) stressful life-events and anxiety/depression. All variables were measured at T1. The output is presented in Table 5 and discussed below. Table 6 provides further model summary information for each of the mediational models.

Hypothesis 2a) CF and EA will mediate the relationship between worry and anxiety, individually as well as in series.

Results (see Table 5) indicated that only CF emerged as a significant mediator of the relationship between worry and anxiety, as demonstrated by the bias-corrected bootstrap CI for the indirect effect (a_1b_1=.133) that was entirely above zero (.075 to .215). Neither the indirect effect of EA, nor the serial mediation path through CF and EA were significant. The direct effect of worry on anxiety was also not significant. These results suggested that the relationship between increasing worry and heightened anxiety occurred indirectly through increased CF (but not EA).

Hypothesis 2b) CF and EA will mediate the relationship between rumination and depression, individually as well as in series.

Results (see Table 5) indicated that neither CF, EA, nor the serial mediation path through CF and EA, significantly mediated the relationship between rumination and depression. A total indirect effect (ab_{total}=.122, CI .001 to .268) was however

found as well as a significant direct effect of rumination on depression ($c'=.417$, $p<.001$). This suggested that as well as a direct relationship between increasing rumination and heightened depression independent of CF and EA, a significant proportion of this relationship was also mediated by CF and EA when summating all the indirect paths together. However, the individual indirect effects were not large enough to reach significance alone.

Hypothesis 2c) CF and EA will mediate the relationships between stressful life-events and anxiety and depression, individually as well as in series.
Results (see Table 5) indicated that only CF emerged as a significant mediator of the relationships between stressful life-events and anxiety ($a_1b_1= .005$, CI .003 to .010) and depression ($a_1b_1=.008$, CI .003 to .015), as demonstrated by the bias-corrected bootstrap CIs entirely above zero. Neither the indirect effects of EA nor the serial mediation pathways through CF and EA were significant. Significant direct effects of stressful life-events on anxiety ($c'=.008$, $p=.02$) and depression ($c'=.014$, $p=.003$) were also found. This suggested that as well as stressful life-events being uniquely predictive of heightened anxiety and depression, increased CF (but not EA) also mediated a significant proportion of these relationships.

Table 5: Bootstrapping Output for Cross-Sectional Mediational Models Testing Hypothesis 2 (Student Sample)

Model paths	B	SE	Indirect effects	B	SE	95% CI	Significant
The mediating role of CF and EA in the relationship between worry and anxiety							
a_1 [worry to CF]	.383***	.053	a_1b_1	.133	.035	.075 to .215	Yes
a_2 [worry to EA]	-.063	.083	a_2b_2	-.005	.008	-.031 to .005	No
a_3 [CF to EA]	.683***	.125	$a_1a_3b_2$	.020	.017	-.008 to .061	No
b_1 [CF to anxiety]	.347***	.086	**Total indirect**	.147	.036	.089 to .231	Yes
b_2 [EA to anxiety]	.075	.060					
c' [worry to anxiety]	.054	.051					
The mediating role of CF and EA in the relationship between rumination and depression							
a_1 [rumination to CF]	.520***	.045	a_1b_1	.089	.065	-.035 to .220	No
a_2 [rumination to EA]	.235*	.105	a_2b_2	.018	.024	-.013 to .085	No
a_3 [CF to EA]	.372*	.152	$a_1a_3b_2$	.015	.021	-.012 to .075	No
b_1 [CF to depression]	.170	.077	**Total indirect**	.122	.068	.001 to .268	Yes
b_2 [EA to depression]	.077	.077					
c' [rumination to depression]	.417***	.084					

Model paths	B	SE	Indirect effects	B	SE	95% CI	Significant
The mediating role of CF and EA in the relationship between stressful life-events and anxiety							
a_1 [life-events to CF]	.015**	.005	a_1b_1	.005	.002	.003 to .010	Yes
a_2 [life-events to EA]	.003	.006	a_2b_2	.002	.001	-.001 to .002	No
a_3 [CF to EA]	.611***	.107	$a_1a_3b_2$	.001	.001	-.001 to .003	No
b_1 [CF to anxiety]	.357***	.073	**Total indirect**	.006	.002	.003 to .011	Yes
b_2 [EA to anxiety]	.063	.059					
c' [life-events to anxiety]	.008*	.003					
The mediating role of CF and EA in the relationship between stressful life-events and depression							
a_1 [life-events to CF]	.015***	.005	a_1b_1	.008	.003	.003 to .015	Yes
a_2 [life-events to EA]	.003	.006	a_2b_2	.001	.001	-.001 to .003	No
a_3 [CF to EA]	.611***	.107	$a_1a_3b_2$	.001	.001	-.001 to .005	No
b_1 [CF to depression]	.506***	.100	**Total indirect**	.009	.003	.004 to .017	Yes
b_2 [EA to depression]	.146	.080					
c' [life-events to depression]	.014**	.005					

*p<.05, **p<.01, ***p<.001

Table 6: Model Summary Information for the Mediational Models (Student Sample)

Mediational Model	Consequent		
	CF	EA	DV (Anxiety/Depression)
IV=Worry, DV=Anxiety	R^2=.33	R^2=.27	R^2=.34
	F(1,104)=52.05, p<.001	F(2,103)=19.20, p<.001	F(3,102)=17.64, p<.001
IV=Rumination, DV=Depression	R^2=.57	R^2=.30	R^2=.50
	F(1,104)=136.24, p<.001	F(2,103)=22.23, p<.001	F(3,102)=34.32, p<.001
IV=Life-events, DV=Anxiety	R^2=.08	R^2=.27	R^2=.37
	F(1,104)=9.40, p=.01	F(2,103)=19.04, p<.001	F(3,102)=20.05, p<.001
DV=Depression	-	-	R^2=.43
	-	-	F(3,102)=25.98, p<.001

Exploratory Analysis with the Reduced RRS

The high correlation between RRS and T1 DASS Depression (r=.71) suggested possible overlap between measures. Treynor et al. (2003) highlighted 12 RRS items with overlapping content to depression. Therefore, the mediational analysis with rumination was rerun using a reduced 10-item RRS (internal consistency, α=.83) having removed this confounding content. As expected, the reduced RRS was less highly correlated with T1 Depression (r=.57) compared to the full scale.

Only CF emerged as a significant mediator of the relationship between the reduced RRS and depression (a_1b_1=.397; CI .153 to .673). Neither the indirect effect of EA (a_2b_2=.026, CI -.025 to .160), nor the serial mediation pathway through CF and EA ($a_1a_3b_2$=.084, CI -.016 to .238) were significant. A significant direct effect of the reduced RRS on depression was also found (c'=.463, p=.01). This suggested that, as well as rumination having a direct effect on depression, a significant proportion of this relationship was mediated by CF (but not EA).

Summary of Hypothesis 2 Results

Hypothesis 2 was partially supported. As predicted, CF uniquely mediated the relationships between worry and anxiety, and stressful life-events and anxiety and depression. However, contrary to expectations, neither EA, nor the serial indirect path through CF and EA, significantly mediated these relationships. In the relationship between rumination and depression, none of the indirect mediational effects were large enough to reach significance alone. However, in

the exploratory analysis with the reduced RRS, a similar pattern of results was found as in the analysis of worry and life-events.

3.5.2 Clinical Sample

Hypothesis 1: CF and EA will explain variance in anxiety and depression

Two mediation analyses were performed (IV: CF, Mediator: EA and DV: Anxiety/Depression). All variables were measured at T1*.*

Higher levels of CF was significantly associated with higher levels of EA (a=.773, p<.001). More importantly, and as predicted, EA was uniquely related to anxiety (b=.164, p=.03), and depression (b=.304, p<.001), when controlling for CF. EA also mediated the relationships between CF and anxiety (ab=.127, CI .028 to .291) and depression (ab=.235, CI .102 to .439), as evidenced by bias-corrected bootstrap CIs above zero. Finally, CF showed a unique direct effect, independent of EA, on anxiety (c'=.244, p=.02), however not on depression (c'= .136, p=.24). Model summary information showed that CF and EA together explained 32% and 35% of variance in anxiety and depression respectively.

The results were largely supportive of Hypothesis 1. Higher EA uniquely predicted greater anxiety and depression, and higher CF uniquely predicted greater anxiety (but not depression). CF had an additional indirect effect on anxiety and depression through increased EA.

Hypothesis 2: CF and EA will mediate the relationships between predictors and symptoms of anxiety and depression.

Serial multiple mediations tested whether CF and EA mediated the relationships between a) worry and anxiety, b) rumination and depression, c) stressful life-events and anxiety/depression. All variables were measured at T1. The output is presented in Table 7 and discussed below. Table 8 provides further model summary information for each of the mediational models.

Hypothesis 2a) CF and EA will mediate the relationship between worry and anxiety, individually as well as in series.

Results (see Table 7) showed that CF and EA did not individually mediate the relationship between worry and anxiety. However, a double mediation effect (worry→CF→EA→anxiety) was found ($a_1a_3b_2$=.031, CI .005 to .100). Increased worrying was associated with higher CF. This was in turn associated with increased EA and finally heightened anxiety. Additionally, worry had a significant direct effect on anxiety (c'=.125, p=.04), independent of CF and EA.

Hypothesis 2b) CF and EA will mediate the relationship between rumination and depression, individually as well as in series.

Results (see Table 7) found that CF did not individually mediate the relationship between rumination and depression. However, EA showed a significant indirect effect independent of CF (a_2b_2=.046; CI .001 to .138). Additionally, a double mediation effect (rumination→CF→EA→depression) was found ($a_1a_3b_2$=.032, CI

.007 to .106). This suggested that rumination led to increased EA, both directly and indirectly through increased CF. This was in turn associated with greater depression. Additionally, rumination had a significant direct effect on depression ($c'=.290$, $p<.001$) that was independent of CF and EA.

Hypothesis 2c) CF and EA will mediate the relationships between stressful life-events and anxiety and depression, individually as well as in series.

Results (see Table 7) showed that CF and EA did not individually mediate the relationships between stressful life-events and anxiety or depression. However, CF and EA in series demonstrated a double mediation effect in the relationships between stressful life-events and anxiety ($a_1a_3b_2=.003$, CI .001 to .009), and depression ($a_1a_3b_2=.006$, CI .002 to .013). Increased CF following stressful life-events was associated with increased EA and this was in turn associated with heightened anxiety and depression. Additionally, stressful life-events had a significant direct effect on anxiety ($c'=.016$, $p=.01$) and depression ($c'=.018$, $p=.01$), independent of CF and EA.

Table 7: Bootstrapping Output for Cross-Sectional Mediational Models Testing Hypothesis 2 (Clinical Sample)

Model paths	B	SE	Indirect effects	B	SE	95% CI	Significant
The mediating role of CF and EA in the relationship between worry and anxiety							
a_1 [worry to CF]	.213***	.089	a_1b_1	.038	.028	-.002 to .115	No
a_2 [worry to EA]	-.073	.107	a_2b_2	-.013	.018	-.059 to .017	No
a_3 [CF to EA]	.805***	.155	$a_1a_3b_2$	.031	.022	.005 to .100	Yes
b_1 [CF to anxiety]	.178	.102	**Total indirect**	.055	.040	-.007 to .157	No
b_2 [EA to anxiety]	.178*	.074					
c' [worry to anxiety]	.125*	.058					
The mediating role of CF and EA in the relationship between rumination and depression							
a_1 [rumination to CF]	.344***	.074	a_1b_1	-.011	.046	-.125 to .062	No
a_2 [rumination to EA]	.248*	.108	a_2b_2	.046	.034	.001 to .138	Yes
a_3 [CF to EA]	.511**	.168	$a_1a_3b_2$	.032	.024	.007 to .106	Yes
b_1 [CF to depression]	-.033	.105	**Total indirect**	.066	.048	-.024 to .170	No
b_2 [EA to depression]	.183*	.079					
c' [rumination to depression]	.290***	.065					

Model paths	B	SE	Indirect effects	B	SE	95% CI	Significant
The mediating role of CF and EA in the relationship between stressful life-events and anxiety							
a_1 [life-events to CF]	.026**	.009	a_1b_1	.004	.003	-.001 to .013	No
a_2 [life-events to EA]	.002	.011	a_2b_2	.001	.002	-.003 to .005	No
a_3 [CF to EA]	.762***	.159	$a_1a_3b_2$	.003	.002	.001 to .009	Yes
b_1 [CF to anxiety]	.170	.072	**Total indirect**	.008	.004	.002 to .018	Yes
b_2 [EA to anxiety]	.159*	.072					
c' [life-events to anxiety]	.016*	.006					
The mediating role of CF and EA in the relationship between stressful life-events and depression							
a_1 [life-events to CF]	.026**	.009	a_1b_1	.001	.003	-.004 to .010	No
a_2 [life-events to EA]	.002	.011	a_2b_2	.001	.003	-.005 to .008	No
a_3 [CF to EA]	.762***	.159	$a_1a_3b_2$	.006	.003	.002 to .013	Yes
b_1 [CF to depression]	.050	.112	**Total indirect**	.008	.005	-.001 to .018	No
b_2 [EA to depression]	.299***	.081					
c' [life-events to depression]	.018*	.007					

*p<.05, **p<.01, ***p<.001

Table 8: Model Summary Information for the Mediational Models (Clinical Sample)

Mediational Model	CF	EA	DV (Anxiety/Depression)
		Consequent	
IV=Worry, DV=Anxiety	R^2=.09	R^2=.34	R^2=.37
	$F(1,55)$=5.71, p=.02	$F(2,54)$=14.02, p<.001	$F(3,53)$=10.46, p<.001
IV=Rumination, DV=Depression	R^2=.28	R^2=.37	R^2=.50
	$F(1,54)$=21.37, p<.001	$F(2,53)$=15.28, p<.001	$F(3,52)$=17.14, p<.001
IV=Life-events, DV=Anxiety	R^2=.13	R^2=.34	R^2=.39
	$F(1,55)$=8.29, p=.01	$F(2,54)$=13.70 p<.001	$F(3,)53$=11.50, p<.001
DV=Depression	-	-	R^2=.43
	-	-	$F(3,53)$=13.09, p<.001

Exploratory Analysis with the Reduced RRS

As with the student sample, the mediational model looking at rumination was rerun with a reduced RRS (internal consistency, α=.76) having removed confounding depression content (Treynor et al., 2003). The reduced RRS was less highly correlated with depression (r=.52), compared to the full scale.

Results showed that CF (a_1b_1=-.023, CI -.268 to .191) and EA (a_2b_2=.090, CI -.015 to .285) did not individually mediate the relationship between the reduced RRS and depression. However, a double mediation effect (rumination→CF→EA→depression) was found ($a_1a_3b_2$=.117, CI .029 to .320). Additionally, the reduced RRS had a significant direct effect (c'=.411, p=.01) on depression that was independent of CF and EA.

Summary of Hypothesis 2 Results

To summarise, while direct relationships existed between all predictors and symptoms of anxiety and depression, CF and EA also mediated a significant proportion of these relationships when considered in series, as predicted. However, contrary to expectations, unique mediation effects of CF and EA, independent of one another, were not found, other than in the relationship between rumination (full scale RRS) and depression where EA showed a unique indirect effect.

3.6 Main Findings: Longitudinal Hypotheses Testing

3.6.1 Student Sample

Hypothesis 1: CF and EA will explain variance in anxiety and depression

The longitudinal analyses were run in the same way as the cross-sectional analyses but instead using T2 anxiety and depression as the outcome variables. T1 anxiety and depression were controlled for in the respective models.

In both analyses, none of the model paths were significant apart from the relationship between CF and EA (a=.538, p<.001 in the anxiety model; a=.470, p<.001 in the depression model). Support for the three predictions made in Hypothesis 1 was therefore not found, namely a) CF did not explain unique variance in T2 anxiety (c'=.108, p=.21) or depression (c'=.209, p=.08) controlling for EA and baseline symptoms; b) EA did not explain unique variance in T2 anxiety (b=-.052, p=.40) or depression (b=-.095, p=.26), controlling for CF and baseline symptoms; and c) CF did not have an indirect effect on T2 anxiety (ab= -.028, CI -.119 to .034) or depression (ab=-.045, CI -.144 to .019) through EA, controlling for baseline symptoms. In summary, contrary to the hypothesis, neither CF nor EA significantly predicted change in anxiety or depression.

Hypothesis 2: CF and EA will mediate the relationships between predictors and symptoms of anxiety and depression.

As CF and EA made no unique contributions to predicting change in anxiety and depression over time, it was not felt appropriate or necessary to proceed to look at their mediating properties in the longitudinal analysis.

3.6.2   Clinical Sample

Insufficient longitudinal data was available to run these analyses.

3.7   Summary of Main Findings

The cross-sectional results demonstrated partial support for our hypotheses. However, the two samples showed differences in CF and EA's roles in explaining variance in anxiety and depression and the impact of worry, rumination and stressful life-events. In the student sample, CF displayed the most explanatory value. In the clinical sample, the interrelationship between CF and EA working in series showed greatest importance. These results did not translate into the longitudinal analyses, with no effects reaching significance in students, and insufficient T2 data to conduct longitudinal analyses in the clinical sample. The results will be discussed in more detail in the next chapter.

CHAPTER 4: DISCUSSION

4.1 Chapter Overview

This study aimed to further understand the role of CF and EA in anxiety and depression. Based on ACT theory (S. C. Hayes et al., 1999) and previous research, the following hypotheses were investigated cross-sectionally and longitudinally: 1) CF and EA will explain variance in anxiety and depression and 2) CF and EA will mediate the relationships between predictors and symptoms of anxiety and depression.

Partial support for the hypotheses was found; however, differences were noted between the two samples studied. The main findings of the cross-sectional analysis addressing each hypothesis will first be discussed with relation to the student and clinical samples in turn. Following this, the results from the two samples will be drawn together, offering explanation on the differences found. Next, the results of the longitudinal analysis conducted in the student sample will be discussed. The theoretical and therapeutic implications of the findings will then be explored, before highlighting strengths and limitations of the study. Finally, future direction for research will be considered.

4.2   Main Cross-Sectional Findings

4.2.1   Student Sample

Hypothesis 1: CF and EA will explain variance in anxiety and depression.

Firstly, CF and EA's relationship to anxiety and depression was investigated. Three specific predictions were made: a) CF will explain unique variance in anxiety and depression, controlling for EA; b) EA will explain unique variance in anxiety and depression, controlling for CF; and c) CF will have an additional effect on anxiety and depression, indirectly through EA. Support for the first of the three predictions was found. CF explained significant variance in both anxiety and depression, having controlled for EA. The more cognitively fused students were with their thoughts, the more they reported symptoms of anxiety and depression. However, support for the next two predictions was not found. EA did not explain unique variance in anxiety and depression, over and above its shared variance with CF, nor did it mediate the relationships between CF and anxiety and depression. Therefore, these results only partially supported ACT theory, which proposes both CF *and* EA are key processes in the development and maintenance of psychological distress (S. C. Hayes et al., 1999).

This is the first study to investigate and support CF's unique role, over and above EA, in both anxiety and depression in students. This reflects ACT theory highlighting that CF leads to suffering (S. C. Hayes et al., 1999). ACT proposes that when CF is high, individuals view their thoughts as literal facts about reality. In this mode of mind, cognitions have excessive influence on emotion and

behaviour to the exclusion of other contextual information (S. C. Hayes et al., 2011). Our findings are also consistent with the limited research investigating CF's relationship with mental ill health (Bernstein et al., 2015; Levin et al., 2012; Masuda et al. 2004). A strength of the present study over the majority of previous research was the ability to control for EA to illuminate CF's *unique* effect. Fergus (2015) documented similar findings whereby CF and health anxiety showed a significant relationship, independent of EA (measured using BEAQ). Our study extended this understanding by investigating CF's unique relationship with anxiety and depressive symptoms more generally, on the premise that CF is a transdiagnostic process (S. C. Hayes et al., 2011).

The current finding that EA did not explain unique variance in anxiety and depression contradicted previous research supporting EA's relationship with psychological distress (e.g., Blakey et al., 2015; S. C. Hayes et al., 2006; Kashdan et al., 2013; Kumpula et al., 2011; Ruiz, 2010; Spinhoven et al., 2014). To reconcile these differences, one could consider the measure of EA (AAQ) previously used. A particularly high correlation (r=.72-.87) has been documented between the AAQ and CFQ (measuring CF). This is considerably higher than the relationship exhibited between the BEAQ (measuring EA) and CFQ in the current study (r=.52 [student sample], .58 [clinical sample]). While originally designed to assess EA, the AAQ has also been recognised to more broadly measure psychological inflexibility (S. C. Hayes et al., 2006), of which CF is one of the core processes (S. C. Hayes et al., 2011). As a result, the AAQ's shared variance and overlap with CF may have partially accounted for the relationships previously

found between the AAQ and anxiety and depression. This leads onto the second difference between the present study and previous research: previous research has not controlled for CF when determining EA's relationship with psychological distress. In the present study, bivariate correlations (not controlling for CF) showed highly significant relationships between EA and depression (r=.48, p<.001), and anxiety (r=.44, p<.001), matching previous research. However, when controlling for CF, EA no longer predicted variance in outcomes. This could be because EA did not have a *unique* relationship with students' MH. Alternatively, controlling for CF may have reduced this effect such that the current sample size was not powerful enough to detect it. Indeed, the relationship between EA and depression, controlling for CF, showed a trend towards significance (p=.06).

The present results also contradicted those found by one of the only other studies to have researched CF and EA together, whereby both CF *and* EA provided additive value in explaining variance in anxiety and depression (Bardeen & Fergus, in press). Looking at the relationship between CF and outcome variables at high, low and mean levels of EA, the authors found high CF and low EA was most associated with symptomology in the minimal range, with high CF and high EA consistently associated with elevated distress. The mean and spread of anxiety and depression symptoms in our student sample appeared similar to their general population sample. However, with anxiety and depression scores bunched towards the lower end of the scale (i.e. positively skewed) in our student sample, only a small proportion of participants were

experiencing elevated distress. This may have limited the power to detect the increasing importance of EA as symptomology increased, as compared to Bardeen and Fergus' (in press) much larger sample (n=955). However, it should also be noted that Bardeen and Fergus (in press) did not seek to examine the temporal ordering of CF leading to higher EA as symptomology increased. In other words, the combination of high EA and low CF may also have been predictive of reduced distress. Additionally, alongside using a different analysis and studying a different population, Bardeen and Fergus (in press) used a different measure of EA (AAQ) to the present study. Further research is needed to help clarify the discrepancy between these two studies' findings.

ACT has also conceptualised CF and EA as serially related (S. C. Hayes et al., 1999), such that people who are more entangled with their thoughts are less willing to remain in contact with their difficult internal experiences, in turn impacting MH. However, evidence supporting this proposition was not presently found in the student sample: EA did not mediate the relationship between CF and symptoms of anxiety and depression. This was inconsistent with the results of the only other study to have investigated this (Dinis et al., 2015). Dinis et al. (2015) used a cross-sectional questionnaire study in a general population sample (n=181) to find that a significant proportion of the relationship found between CF and depressive symptoms occurred through EA. In contrast to the present study, Dinis et al. (2015) measured EA using the AAQ, used a larger sample (i.e. increasing power) and specifically excluded student participants. It is possible the present study required greater power to find this effect, with the

indirect effect of EA in the relationship between CF and depression appearing to show a trend towards significance (ab=.100, CI -.017 to .246).

Hypothesis 2: CF and EA will mediate the relationships between predictors and symptoms of anxiety and depression.

ACT views difficult thoughts, feelings and life-events as ubiquitous to human existence. Only when we relate to these experiences in an unhelpful way do they become problematic (S. C. Hayes et al., 1999). Therefore, beyond just predicting a simple relationship of CF and EA with anxiety and depression, ACT theory would further expect CF and EA to explain the impact of 'negative' thinking and difficult life-experiences known to predict these MH conditions (S. C. Hayes et al., 1996; Kashdan et al., 2006). Hypothesis 2 thus predicted that both CF and EA would individually mediate the relationships between a) worry and anxiety, b) rumination and depression, and c) stressful life-events and anxiety and depression. Furthermore, given the interrelationship between CF and EA, CF was predicted to additionally act in series through EA (i.e. worry/rumination/life-events→CF→EA→anxiety/depression). As predicted, the results showed CF significantly mediated the positive relationships between worry and anxiety, and stressful life-events and both anxiety and depression, controlling for EA. However, unexpectedly, neither a unique indirect effect through EA, nor an indirect effect of CF working through EA in series, was found. This mirrors the results of Hypothesis 1. In the relationship between rumination and depression,

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the specific and double mediation effects only reached significance when summated, but not individually.

While these mediational models had not been tested before, previous research had supported the constituent relationships, particularly with relation to EA. For example, relationships between worry, EA and anxiety had been cross-sectionally found in non-clinical samples (Buhr & Dugas, 2012; Lee et al., 2010; Roemer et al., 2005), as had relationships between rumination, EA and depression (Bjornsson et al., 2010; Cribb et al., 2006). This research supported theories highlighting worry and rumination's avoidant nature (Nolen-Hoeksema et al., 2008; Roemer & Orsillo, 2002; Watkins & Moulds, 2005). Furthermore, EA has been associated with psychological distress following stressful life-events (Plumb et al., 2004; Shallcross et al., 2010). However, the present findings were inconsistent with this body of research.

As discussed with respect to Hypothesis 1, the AAQ used in prior research may have confounded the measurement of EA and CF. The present study is the only research to have measured EA using the BEAQ and controlled for CF when investigating these relationships. These two factors may have reduced EA's unique effect in mediating the relationships of worry, rumination and life-events with anxiety and depression, to the degree to which it was either non-existent or no longer detectable with the current study's power. Furthermore, our results are consistent with a recent study by Spinhoven et al. (2016), which also found

EA (measured using AAQ) did not mediate the longitudinal relationships of rumination or worry with depression in a mixed sample.

Looking specifically at the relationship between worry and anxiety, CF was rather indicated to be the key mediator. While this had not been studied before, the results bare resemblance to the first element of Roemer and Orsillo's (2002, 2005) model of GAD highlighting the role of an individual's 'fused' relationship with their experiences. This also reflects the theory underpinning ACT, which predicts that when cognitively fused with worrisome thoughts, the same anxiety is triggered as if the worries were true (S. C. Hayes et al., 2001). Contrary to the second element of Roemer and Orsillo's (2002, 2005) model and ACT theory (S. C. Hayes et al., 1999), CF did not show an additional effect through its influence on EA. In light of the lack of consensus on power analysis in mediation, especially for more complex multiple mediation models, it is possible that the current sample was not large enough to find this potentially smaller double mediation effect. Indeed, Thoemmes et al. (2010) highlighted that the product term of the three paths in serial mediation can in many circumstances be very small making it hard to detect. In line with this, the lower confidence limit of the double mediation path in this relationship was notably close to zero (CI: -.008 to .061). This requires further research with larger samples to help clarify.

CF also mediated the relationships between stressful life-events and anxiety and depression. This had not been tested before, however our results can be seen to extend that found by Dinis et al. (2015), whereby CF uniquely mediated the

relationship between a narrower categorisation of life-events (i.e. memories of early-life shameful experiences with traumatic-like characteristics) and current depressive symptoms. While reporting unstandardised coefficients in mediation is recommended, thereby making meaningful quantification of effects as 'large' or 'small' in a practical or theoretical sense hard (A. F. Hayes, 2013), it is notable in our study that the coefficients of CF's indirect effects in these relationships were close to zero, possibly indicating a 'small' effect. However, this would also be symptomatic of the very wide range (and correspondingly high SD [179.94]) of possible scores on the life-events measure (LESS) compared to the other measures used. This means one unit increase on the LESS would correspond to a fraction of a unit on the other questionnaires. Indeed, the model summary information still showed that all the predictor variables explained 37% of variance in anxiety and 43% in depression. As in the relationship between worry and anxiety, evidence for an additional indirect effect of CF operating through EA was not found, inconsistent with ACT theory (S. C. Hayes et al., 1999) and Dinis et al.'s (2015) research. Power constraints may have reduced the ability to detect this effect, as discussed above. The retrospective measurement of life-events over the *past year* may also have affected the strength of these relationships.

In the relationship between rumination and depression, none of the indirect effects reached significance on their own. The lessened role of CF in this relationship, compared to the relationship between worry and anxiety, may reflect Gillanders et al.'s (2015) research in cancer patients, whereby CF only mediated the relationship between illness-related cognitions and anxiety, not

depression. Alternatively, CF's indirect effect may not have reached significance due to the RRS including confounding content with depression (Treynor et al., 2003), reflected in the high correlation between rumination and depression presently found (r=.71). Indirect effects are calculated from the product of path a and b in the mediational model. Here, path b reflects the relationship between CF and depression, controlling for EA and *rumination.* Controlling for the RRS may have inadvertently controlled for variance in depression given the measures' overlap, attenuating path b's effect size. This explanation was supported in the exploratory analysis. Although exploratory analyses should be interpreted with considerable caution, when the confounding content of the RRS was removed, a significant indirect effect of CF emerged, similar to the other mediational models studied. This would be consistent with prior research finding the combination of high CF and rumination particularly detrimental to MH (Romero-Moreno et al., 2015).

In this way, this preliminary research suggests CF may provide a shared explanation for the impact of three different internal and external predictors of anxiety and depression in students. Over-identification with verbal processes, including worry, rumination (based on the exploratory analysis) and cognitions following stressful life-events, appears to be a key mechanism by which difficult thoughts can lead to suffering, consistent with ACT theory (S. C. Hayes et al., 1999). However, contrary to ACT-based predictions, EA did not mediate these relationships, either individually or in tandem with CF. In addition, rumination and stressful life-events also showed direct relationships with symptomology,

independent of CF and EA. This suggests other factors were at play too, and/or the content of one's experiences had at least some direct influence over MH.

4.2.2 Clinical Sample

Hypothesis 1: CF and EA will explain variance in anxiety and depression.

While CF was the most pertinent factor in student's MH, EA showed increased importance in the clinical sample. As predicted, higher EA was uniquely associated with heightened anxiety and depression, controlling for CF. Additionally, increased CF was uniquely associated with higher anxiety, but not depression, independent of EA. Finally, CF had an additional effect on anxiety and depression indirectly through EA. Other than the lack of unique relationship between CF and depression, the results supported all three predictions made in Hypothesis 1 and the central premise of ACT highlighting the importance of people's *relationships* to their experiences (S. C. Hayes et al., 1999).

EA's relationship with anxiety and depression mirrors previous research (e.g. S. C. Hayes et al., 2006; Ruiz, 2010; Spinhoven et al., 2014). In the present study, controlling for CF added credence to EA's role in MH beyond its shared variance with CF. This could reflect the paradoxical effects of EA, whereby attempts to avoid aversive internal experiences leads to rebound effects (Wenzlaff & Wegner, 2000) and reduced value-congruent living (S. C. Hayes et al., 2011). CF's unique relationship with anxiety also fits ACT theory (i.e. entanglement with

verbal processes leads to suffering; S. C. Hayes et al., 1999) and previous research (Fergus, 2015). It is unclear why the direct relationship between CF and depression was not significant. One possibility is the study's sub-optimal power could not detect this perhaps smaller effect, having controlled for EA. Alternatively, CF's influence on depression may wholly occur via EA, with CF's *unique* effect only influential in anxiety. Gillanders et al. (2015) found a similar result in cancer patients, whereby CF was uniquely predictive of anxiety, but not depression, having controlled for avoidant coping. Finally, the serial relationship between CF and EA was supported in both anxiety and depression. This was consistent with ACT theory (S. C. Hayes et al., 1999) and the previously discussed research of Dinis et al. (2015); however, not with the results presently found in the student sample where CF did not act in conjunction with EA. The differences found between samples are discussed in more detail later in this chapter.

Hypothesis 2: CF and EA will mediate the relationships between predictors and symptoms of anxiety and depression.

Contrary to predictions, in the clinical sample neither CF nor EA uniquely mediated the relationships of worry and stressful life-events with symptomology. However, EA did uniquely mediate the relationship between rumination and depression. Additionally, consistent with predictions and the results of Hypothesis 1, but in contrast to the student sample, the double mediation route through CF and EA was significant in all relationships. The more entangled individuals were with their worries, ruminations and cognitions

following stressful life-events, the more they tried to avoid or control their aversive internal experiences and the situations that elicited them. This was related to heightened anxiety and depression. While the study design does not permit empirically supported causal conclusions, these results are indicative of the causal relationship between CF and EA highlighted in the ACT model (S. C. Hayes et al., 1999). Only one other study had investigated this double mediation pathway (Dinis et al., 2015).

Dinis et al. (2015) particularly focused on the relationship between memories of early-life shame experiences and current depressive symptoms. They found that the perceived impact of shame experiences was related to depressive symptoms through CF and EA in series. This is comparable to the current findings. Dinis et al. (2015) also found additional unique mediational effects of CF and EA in this relationship, controlling for one another. It is unclear why the current study did not consistently find similar unique indirect pathways in the mediational models studied. ACT theory conceptualises CF and EA as distinct processes, which make their own contributions to MH (as largely reflected in the results of Hypothesis 1), in addition to having a serial relationship (S. C. Hayes et al., 2011). It is possible that unique effects through CF and EA were present but smaller than the serial pathway and therefore not detectable in this underpowered clinical study. Indeed, the lower confidence limit of the indirect effects of CF in the relationships between worry and anxiety (CI: -.002 to .115) and life-events and anxiety (CI: -.001 to .013) only marginally fell below zero. Alternatively, CF and EA may solely operate in series in these relationships. Future research in this

area should continue to investigate CF and EA together within the same research design in order to further decipher whether CF and EA's effect on MH is wholly dependent on one another.

Looking specifically at the relationship between worry and anxiety, the double mediation (worry→CF→EA→anxiety) is consistent with ACT theory (S. C. Hayes et al., 1999) and resembles Roemer and Orsillo's (2002, 2005) model of GAD whereby excessive fusion with cognitions motivates the individual to try and avoid these aversive experiences. This increases anxiety due to EA's rebound effect (Wenzlaff & Wegner, 2000) and impact on valued-living (Roemer & Orsillo, 2002, 2005). The results also fit an ACT conceptualisation of Well's (1997) metacognitive model of GAD, whereby hypothesised dangers of worry may more readily initiate avoidance strategies when CF is triggered. This mediational model had not been tested before, however research had supported the relationships of EA (Lee et al., 2010; Roemer et al., 2005), and less extensively CF (Arch et al., 2012), with worry and anxiety (and in particular GAD). Unlike previous research and contrary to predictions, EA did not presently show its own relationship with worry. EA may only be triggered as a result of high CF. A cognitively defused relationship to worries may instead support a more accepting stance in which thoughts and emotions are free to come and go and workable valued action can be pursued.

Researchers have also conceptualised rumination as functionally similar to worry, largely focussing on one's experientially avoidant relationship to

ruminative thoughts in non-clinical samples (e.g. Bjornsson et al., 2010; Cribb et al., 2006; Kashdan et al., 2006). More recent research had supported a mediating role of behavioural avoidance in the relationship between rumination and depression in a clinical sample (Brockmeyer et al., 2015). These findings are consistent with the unique mediational effect of EA presently found in the relationship between rumination and depression. Note, however, that confounding depressive content of the RRS may have inflated path a in the mediational model (i.e. rumination's relationship with EA) since we already know EA is related to depression. While exploratory analyses should be interpreted with caution, when confounding depressive content was removed from the RRS, EA's indirect effect, while still showing a trend towards significance, did not reach significance. These results are more similar to Spinhoven et al.'s (2016) findings that EA did not mediate the relationship between rumination and depression in their longitudinal design. It may rather be that EA's mediational effect is largely dependent on its interrelationship with CF, as supported by the present findings using both the full and reduced RRS. Over-identification with ruminative thoughts may be what drives EA and subsequent depression. Like worry, in a more cognitively defused context, rumination may function in a more adaptive and reflective way. Until now, the role of CF in this relationship had been neglected.

CF and EA demonstrated a similar double mediation effect in the relationships between stressful life-events and anxiety and depression. Extending ideas from theories of trauma, adjustment to stressful-life events relies on the processing of

all event-related information (including difficult thoughts, memories, emotions and physiological arousal; Foa & Kozak, 1986). This would be compromised where EA is high (Batten et al., 2005). Indeed, EA has shown a relationship with anxiety and depression following difficult life-events (Plumb et al., 2004; Shallcross et al., 2010). The current results again suggest that this relationship is dependent on EA's interrelationship with CF, such that EA did not mediate the effect of stressful life-events on symptomology when controlling for CF, contrary to a priori predictions. Becoming overly caught up with difficult thoughts and memories about stressful life-events may be what motivates attempts to avoid aversive internal experiences, leading to subsequent maladjustment (S. C. Hayes et al., 2011). This is consistent with the serial mediation effect found by Dinis et al. (2015), as previously discussed. Like the student sample, while unstandardised coefficients for these indirect effects appeared small, this is unsurprising given the high SD (98.44) of the life events measure (reflecting the very large range of possible scores) compared to the other measures used. Small product terms are also common in serial mediation effects (Thoemmes et al., 2010).

Finally, direct effects of worry, rumination and stressful life-events on symptomology, independent of CF and EA, were also found. This suggests the pathway through CF and EA is not the only way in which these vulnerabilities impact MH.

4.2.3 Summary

Different patterns of results were found in the two samples. In students, CF, as opposed to EA, was the key process in explaining variation in symptoms of anxiety and depression and the impact of different internal (worry and possibly rumination) and external (stressful life-events) vulnerability factors. In the clinical sample, EA played an increasingly important role, with CF and EA working in series giving the most explanatory value across the different analyses. The overall results of both samples are partially consistent with the central ACT premise that people's relationship with their difficult experiences plays a role in MH (S. C. Hayes et al., 1999), however CF and EA did not ubiquitously show both unique and interrelated roles in explaining variance in MH across both samples. Indeed, the differences found between the core processes at play in the two samples were not predicted and have not previously been found.

Before commenting on these differences, it is worth highlighting that the student and clinical samples were not matched and different measures of anxiety and depression were used. Therefore, it is unknown which of the dissimilarities in sample characteristics underpinned the different results. For example, the participants in the student sample were by their nature students, younger than the clinical participants and a non-clinical sample. Furthermore, students were recruited as a convenient and feasible means of obtaining a non-clinical sample and so may not have been representative (with relation to differences in demographics, socioeconomic status and clinical characteristics) of a non-clinical

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general population sample more broadly. This makes it hard to draw comparisons across the two samples studied and any speculation on the differences found needs to be approached with considerable caution.

One difference between the two samples was their clinical characteristics, with one sample representing a clinical population of treatment-seeking individuals with MH difficulties and the other sample consisting of a non-clinical convenience sample of students. In students, DASS Depression and Anxiety Scales were positively skewed, with scores bunched towards the lower end of the continuum and only a few cases falling in the higher range. Only a small proportion (16%) reported having a history of MH problems. In contrast, the majority of the clinical sample displayed clinically significant symptoms of anxiety and/or depression, with a sizeable proportion falling in the 'severe' (Kroenke et al., 2001; Spitzer et al., 2006) categories of anxiety (37%) and depression (47%).

Tentatively speculating that it was the non-clinical versus clinical nature of the two samples driving the different results, it may follow that an entangled relationship with difficult thoughts (CF) is more fundamental to variation in anxiety and depression symptoms towards the lower end of the MH continuum. Only when this cognitively fused relationship with one's difficult thoughts leads to a certain degree of general non-acceptance and avoidance of these cognitions, resultant feelings, and situations that trigger them (EA), might CF become highly problematic and clinically significant symptoms present. Thus, the combination

of high CF and EA may be especially toxic. The restricted range of symptoms in the student sample might explain why some effects involving EA showed a trend towards, but did not reach, significance. This is consistent with the findings of Bardeen and Fergus (in press) whereby high CF and low EA was most associated with anxiety and depressive symptomatology in the minimal range, and high CF *and* high EA associated with more elevated distress. However, neither Bardeen and Fergus (in press) nor our study's design could determine causality between study variables (discussed further in Section 4.5, 'Strengths and Limitations'), including the assumption that CF precedes EA in a causal chain. Therefore, our results can only be tentatively interpreted within the knowledge of ACT theory. As CF and EA have only recently been researched together, the intricacies of their relationship with MH across the continuum may have previously been missed. This requires future research to investigate further. Comparing matched clinical and non-clinical samples, with a non-clinical sample not simply taken from a student population, would help confirm whether it was indeed the clinical versus non-clinical characteristics driving the different results.

4.3   Main Longitudinal Findings

Cross-sectional designs have dominated literature in this area. This has great value in establishing relationships between variables. However, longitudinal designs are needed to extend this understanding to determine temporal associations. A longitudinal analysis was conducted in the student sample to determine CF and EA's role in predicting change in anxiety and depression (i.e. a

longitudinal test of Hypothesis 1). Contrary to the cross-sectional findings, CF and EA showed no capacity to explain significant change in anxiety and depression over time, either individually or in series. Consequently, it was not thought appropriate or necessary to continue to longitudinally investigate their mediating properties in the relationships between predictors and symptoms of anxiety and depression (Hypothesis 2).

Bjornsson et al. (2010) found a similar discrepancy between their cross-sectional and longitudinal findings: while EA (measured using AAQ) and depression were cross-sectionally related, EA did not predict depression 8-12 weeks later, controlling for baseline symptoms. Bjornsson et al. (2010) highlighted that the AAQ confounds the measurement of EA and depression. Thus, controlling for baseline depression may have led to a Type II error. However, our research measured EA using the BEAQ, which has not suffered the same criticisms (Gámez et al., 2014). It may rather be that both studies investigated small (n=97 [present study], 72 [Bjornsson et al., 2010]), non-clinical samples over short time periods where there is limited change in symptoms. Cole and Maxwell (2003) highlighted a certain amount of time must pass for one variable to affect another and therefore determination of the optimal time-frame for longitudinal research deserves careful consideration. The time-delay used in the present study (average=48 days) was selected to match the expected delay in the clinical sample where time constraints were inherent in the procedure, and based on preceding research (Bardeen et al., 2014; Bjornsson et al., 2010). However, previous researchers may themselves have selected time-delays based on

tradition and convenience, rather than theory or careful research, as often done (Cole & Maxwell, 2003). This threatens longitudinal designs and might have impacted the current results. Any effects are likely to have been small having accounted for baseline symptoms, with a greater power needed to detect them.

This does not necessarily preclude a relationship between these ACT variables and future MH from existing. Indeed, CF's ability to predict unique change in depression symptoms showed a trend towards significance (p=.08) in the student sample, reflecting CF's role in the cross-sectional findings. Regrettably, as discussed, insufficient T2 data in the clinical sample meant we could not determine whether these cross-sectional results were supported longitudinally. While previous longitudinal research is limited, EA has been found to significantly predict change in anxiety and depressive symptoms over longer durations (two-four years) in larger mixed samples (Spinhoven et al., 2014; Spinhoven et al., 2016). However, this relationship did not hold true when controlling for worry, rumination and neuroticism (Spinhoven et al., 2016). As discussed in Chapter 1, 'Introduction', this may have been because the AAQ (measuring EA) has limited discriminant validity, including with neuroticism. Therefore, controlling for neuroticism may have inadvertently controlled for variance explained by the AAQ.

In summary, two competing explanations exist when explaining the lack of longitudinal results: either EA and CF provide no value in predicting future MH, or methodological limitations have prevented these effects from being found.

The lack of longitudinal designs investigating CF and EA together, along with the methodological limitations outlined in the present study, need to be addressed in future research in order to help determine the absence versus presence of these longitudinal relationships.

4.4   Implications

The present findings have key theoretical implications. This study sought to use the theory underpinning ACT to develop understanding of two highly prevalent MH conditions: anxiety and depression. Investigating CF and EA in the context of each other has helped advance existing research in this area to further illuminate their *unique* and *interrelated* contributions to mental ill health. This uncovered unpredicted differences in CF and EA's relative influence in the two samples studied. While the present research is unable to firmly conclude what underpinned these differences between the two unmatched samples, one possible explanation is that CF, as opposed to EA, might be a core factor in variation in anxiety and depression towards the lower end of the MH continuum. Only when CF leads to a certain degree of unwillingness to remain in contact with aversive experiences (EA) might clinically significant symptoms emerge. Given the preliminary nature of this research, these findings and possible explanations deserve further investigation and if consistent results are found, integration into the ACT model. Furthermore, it should not be overlooked that these relationships are yet to be established longitudinally, and future research is needed to clarify the non-significant longitudinal results found in students. As

yet, it is unclear whether these processes hold value in explaining change in psychological distress over time.

Since CF and EA have rarely been studied together, it is worth considering their value as distinct constructs. Gillanders et al. (2014) highlighted a particularly high correlation between the AAQ and CFQ, possibly indicating considerable construct overlap between EA and CF. Alternatively, this high correlation may have been driven by the confounding content of the AAQ with CF (Gillanders et al., 2014), as previously discussed. In the current study, the BEAQ and CFQ were less highly correlated and showed incremental validity, explaining *unique* variance in anxiety and depression over and above one another. Additionally, in the clinical sample, CF impacted anxiety and depression through its effect on EA in a possible causal chain. Our results thereby support CF and EA as two distinct but interrelated constructs, however contrary to predictions, these processes showed differential relative importance within the two samples studied and EA only exhibited explanatory value in the clinical sample.

The present study additionally moved beyond investigating just basic associations of CF and EA with MH, to determine whether these processes helped explain the mechanisms by which common everyday thinking patterns (worry and rumination) and stressful life-events may trigger anxiety and depression. The cross-sectional findings suggested that the negative effects of these internal and external vulnerabilities to anxiety and depression was in part due to CF in students, and CF and EA in concert in the clinical sample. Thereby, rather than

just focusing on the frequency, nature or content of worry, rumination and stressful life-events, deciphering one's relationship to those experiences may help explain their association with MH. *Within* each sample, the same pattern of results was largely found across dependent variables (worry, rumination, life-events) and outcome variables (depression and anxiety). This could support a transdiagnostic approach to MH, whereby topographically disparate vulnerabilities have shared underlying mechanisms (Boulanger et al., 2010; S. C. Hayes et al., 1996; Kashdan et al., 2006). Isolating a small set of core functional dimensions applicable to a wider range of clinically-relevant problems could provide a useful theoretical and therapeutic framework to MH (S. C. Hayes et al., 2011) and would help explain the high comorbidity among emotional disorders (Brown, Campbell, Lehman, Grisham & Mancill, 2001). Theory and research seeking to understand the negative effects of worry, rumination and stressful life-events had previously overlooked CF as a core process, despite its prominence in the ACT model (S. C. Hayes et al., 1999). The present results suggest CF deserves further exploration in future research.

However, *between* samples, unexpected differences were found, with EA not showing significant explanatory value in the student sample contrary to expectations and the results of the clinical sample. Additionally, the indirect effect of CF in the relationship between rumination and depression in students was indicated by exploratory analysis and needs verification from future research. Furthermore, before making premature conclusions, longitudinal and experimental designs across more diverse samples are needed to verify causal

associations, especially in light of the present non-significant longitudinal findings in the student sample.

As discussed by S. C. Hayes et al. (2011), the acid test for any treatment model is its ability to translate into clinically meaningful interventions. ACT interventions move the focus away from trying to change the content, nature or frequency of 'problematic' internal experiences known to predict MH difficulties, towards targeting common unhelpful relationships (CF and EA) to these experiences (Ciarrochi et al., 2010). While our findings partially support CF and EA as possible targets for treatment, they also indicate different approaches may be required in the two populations studied (with EA not exhibiting explanatory value in the student sample), should these findings be replicated in future research.

University can be a particularly difficult time in one's life (Eisenberg et al., 2007; Grant, 2002). It is therefore important to develop effective interventions to both prevent and treat anxiety and depression in this population. Should the present results be replicated both cross-sectionally and longitudinally in future research using larger and more diverse samples, this would suggest a target for intervention could be student's cognitively fused relationship with their thoughts. Cognitive defusion techniques aim to reduce CF by teaching people to see thoughts for what they are (i.e. symbols of one's experience) and not what they say they are (i.e. descriptive 'realities'; Ciarrochi et al., 2010). In this way, cognitions are not challenged or restructured to deter maladaptive

thought→action/emotion relations, but rather, the illusion of language is penetrated to undermine the *context* that automatically supports such thought→action/emotion relations. This skill is considered valuable within the ACT community given the intensely verbal world humans live in (S. C. Hayes et al., 2001). Emerging support for cognitive defusion as a core mechanism of change in reducing distress has been found (Levin et al., 2012). It is of note that given the present study focussed on a student sample, these potential treatment implications may not be applicable to the general population.

In the clinical sample, CF and EA appeared to play a connected role in anxiety and depression. Should these results be replicated across larger, more diverse samples using longitudinal and experimental designs, this could indicate that the ACT approach of changing one's relationship to difficult experiences so that they are no longer viewed as 'symptoms' may have potential to improve therapeutic outcomes. This is usually not the agenda of a client coming to therapy, who often wants help to control and get rid of their difficult emotions. However, ACT techniques aim to reduce CF and EA in the service of increasing valued, workable action and less restrictive behavioural patterns. EA is targeted through exploring the ineffectiveness of emotional control and avoidance while encouraging the individual to accept difficult private experiences when doing so helps them engage in valued living (Ciarrochi et al., 2010). A more comprehensive account of ACT interventions has been summarised by S. C. Hayes et al. (2011). Given the present preliminary findings have indicated that CF may lead to heightened EA which is linked to worse MH, anticipatory interventions to reduce EA could

potentially be helpful in individuals prone to become excessively entangled with their thoughts.

Evolving research is suggesting ACT interventions exhibit promise in treating anxiety disorders and depression (Avdagic et al., 2014; Forman et al., 2007; Tamannaeifar et al., 2014; Zettle et al., 2011). Furthermore, Ruiz (2010) concludes his review of ACT research, by stating that ACT therapy was effective across different diagnoses where a pattern of high EA, in the context of CF, was present. However, this research is still of a preliminary nature and larger scale, methodologically rigorous randomised controlled trials with longer-term outcomes are needed to confirm ACT's efficacy and value as an evidence-based treatment in MH, (Öst, 2014; Swain, Hancock, Hainsworth, & Bowman, 2013) with ACT as yet not recommended by NICE guidelines.

4.5   Strengths and Limitations

This study has a number of strengths. Firstly, it utilised measures of EA and CF with good psychometric properties and the ability to operationalise these constructs well (Gámez et al., 2014; Gillanders et al., 2014). Researchers using the AAQ to assess EA have themselves highlighted their findings need replication using more robust measures, citing the BEAQ due to its superior psychometrics (Bardeen & Fergus, in press). Furthermore, the CFQ provided the means to advance our limited understanding of CF (Gillanders et al., 2014).

Secondly, unlike the majority of previous research, this study investigated CF and EA together to determine their relative and interrelated contributions to understanding anxiety and depression. Including multiple mediators in analyses allows the assessment of more ecologically valid models grounded in theory, rather than less realistic simple models that consider only one mediator (A. F. Hayes, 2013; Preacher & Hayes, 2008).

This study's analytic approach is a third strength. Multiple mediation analysis with bootstrapping (Preacher & Hayes, 2008) shows advantages over other analytic strategies used. For example, Baron and Kenny's (1986) causal steps approach determines the presence of an indirect effect based on the outcome of a series of null hypotheses, increasing the likelihood of error (A. F. Hayes, 2013). In contrast, bootstrapping explicitly estimates indirect effects, is a higher-powered test with reduced chance of Type I and II errors and makes no assumptions about sampling distributions, making it more appropriate for smaller samples (A. F. Hayes, 2013; MacKinnon, Lockwood, & Williams, 2004).

Fourthly, this study used both student and clinical samples to further understand anxiety and depression in different populations across the MH continuum. Taking a dimensional view of MH, ACT research often uses non-clinical (and particularly student) samples as a convenient means of testing theoretical ideas. However, unexpectedly, this research found slightly different results across the student and clinical samples. As this is the first study of its kind using new

measures, these results need replicating to further elucidate whether CF and EA are differentially important in these two populations.

However, this research is not without limitation. Bootstrapping relies on a sample representative of the population from which it was drawn (A. F. Hayes, 2013). Both samples were female-dominated (84% females, student sample; 74%, clinical sample), like much of previous research in this area. The generalisability of the results to men needs to be established, especially in view of gender differences previously found in constructs measured, including rumination (Nolen-Hoeksema, Larson, & Grayson, 1999), CF (Dinis et al., 2015) and depression (Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993). Participants were also predominately white ethnicity, especially within the clinical sample (93%). Moreover, participants were self-selected. This may have meant, for example, that more depressed, anxious or experientially avoidant individuals were less likely to sign up. Further research using more diverse samples is needed and larger samples would have allowed increased power to control for additional variables in analyses; for example, demographic and clinical characteristics (e.g. anxiety where depression was the outcome, and visa versa, given their high comorbidity; Hirschfeld, 2001).

The clinical sample were recruited from an IAPT primary care MH service, and therefore only consisted of treatment-seeking individuals, not necessarily representative of all individuals with MH conditions (Andrade et al., 2014; Wang et al., 2007). Demographic and attitudinal differences have been found between

those that do and do not seek treatment (Andrade et al., 2014). Furthermore, within the treatment-seeking population, an IAPT sample is likely to exhibit milder symptoms of anxiety and/or depression than individuals with more severe and/or treatment-resistant presentations referred to secondary care MH services. The clinical participants were also adults seeking psychological help due to symptoms of anxiety *and/or* depression, in other words a clinically heterogeneous sample. The use of a mixed sample accepts the ACT assumption that the processes studied are transdiagnostic (S. C. Hayes et al., 2011), without explicitly testing this. Future research would benefit from looking at participants with anxiety and participants with depression separately to confirm EA and CF's transdiagnostic status and whether the present results translate across diagnosis-specific clinical groups. Unfortunately, the limited sample size of the clinical sample prohibited this from being examined in the present study.

This study sought to test the ACT prediction that CF and EA would be relevant across the spectrum of experience. To do this, along with the clinical sample, students were recruited as a convenient and feasible means of obtaining participants from a non-clinical population. However, utilising a student sample comes with limitations as it represents a particular sub-population within the general population, as opposed to being representative of the general population as a whole (for example, in relation to differences in demographics and socioeconomic status). Furthermore, a higher level of psychological distress has also been reported in students compared to the general population (Eisenberg et al., 2007; Grant, 2002). Additionally, the majority of the student sample were

psychology undergraduates, potentially further biasing the sample. In light of this, this study needs replication in more diverse non-clinical samples from the general population to clarify the generalisability of the results.

Additionally, the clinical sample (n=57) was underpowered, with 80 participants recommended (Fritz & Mackinnon, 2007). Fritz and MacKinnon's (2007) method gives sample sizes all at a power of .80, based on simple mediation models. By their estimations, the clinical sample would only have been able to detect large effect sizes at this power (having accounted for the additional mediator in our study). Furthermore, there is a general lack of both clarity and accessible methods for power calculations in more complex multiple mediations (Fritz & Mackinnon, 2007; Thoemmes et al., 2010). The present power calculation aimed to provide the best estimate of the sample sizes required and accounted for the additional mediating variable as far as possible by cross-referencing Cohen's tables (1992). However, sample size requirements may still have been underestimated given (i) mediated effects can remain underpowered even when individual paths have high power; (ii) complex relationships exist within multiple mediation models (with their magnitude not possible to directly predict where not previously tested); (iii) product terms of serial mediation paths are often small; and (iv) different mediated pathways within one model can have quite different power estimates (Thoemmes et al., 2010). In the longitudinal analysis, adding a further variable (i.e. controlling for baseline symptoms) may too have required a larger sample to detect effects. Given the potential unreliability around these more complex power analyses, where insignificant

results were found (and especially where a trend towards significance was demonstrated), replication with larger samples would determine the reliability of *not* finding an effect.

This study used self-report measures, which to some extent are the most appropriate method for assessing subjective experiences (Kashdan et al., 2006). However, dynamic psychological processes may not reliably translate into responses derived from static self-report measures (Wolgast, 2014) and self-reports rely on the accuracy of the respondent. Moreover, questionnaire completion may be especially difficult for individuals with particular clinical presentations. For example, individuals with GAD have shown difficulties recognising and describing their internal experiences (Mennin, Heimberg, Turk, & Fresco, 2005). Individuals highly avoidant of aversive internal experiences may too be less likely to report symptomology.

Self-report measures are also only so good as their ability to operationalise the construct. The RRS has suffered criticisms for including confounding content with depressive symptoms (Treynor et al., 2003). When confounding content was removed, slightly different results to the main analysis were found. *A priori* hypothesis testing is needed to clarify this exploratory analysis. Furthermore, the RRS showed a particularly high correlation with the CFQ in the student sample (r=.75), possibly indicating construct overlap. However, consistent with previous research supporting rumination and CF as distinct constructs (Gillanders et al., 2014), the current study did not find evidence of

multicollinearity and CFQ helped explain the relationships between rumination and depression in the student (using the reduced RRS) and clinical samples. Nonetheless, with research in its infancy, future work needs to continually develop CF into a well-defined construct and further understand its similarities, differences and relationships with other variables.

In the clinical sample we were constrained by the standard measures of anxiety and depression used at the service (as these questionnaires, completed as standard-practice at participants' first appointment, would have formed the T2 data). This included the GAD-7, originally designed to assess GAD (Spitzer et al., 2006); however, this was not a large concern given it is also considered useful in measuring anxiety more generally (IAPT National Programme Team, 2011), as discussed in Section 2.5, 'Measures'. A further consideration is that, along with assessing other symptoms of anxiety, this questionnaire included two items related to worry. While worry is considered causally related to anxiety and not just part of its phenomenology (Gana et al., 2001; Purdon & Harrington, 2006), this would have lead to some degree of construct overlap between the GAD-7 and PSWQ. Despite this, these questionnaires did not demonstrate a concerningly high correlation (r=.36). Moreover, when calculating CF and EA's relationship to anxiety (path b) within the mediational model (IV=worry), the PSWQ was controlled for, mitigating against the influence of worry-related questions in the GAD-7 in these mediation effects. The direct relationship between worry and anxiety, however, may have been inflated.

Additionally, the life-events measures are worth consideration. While well validated and widely used, a few items could represent outcomes of stressful life-events as opposed to stressful life-events themselves (e.g. 'change in sleeping habits' [SRRS]; Scully et al., 2000). The SRRS has further been criticised for including both desirable and undesirable events, and controllable and uncontrollable events; however, these criticisms were unfounded, with all four categories of events explaining variance in stress-related outcomes (Scully et al., 2000). The life-event questionnaires also did not allow subjective quantifications of how stressful respondents found selected events, with certain events possibly not stressful to some individuals. Despite this, these questionnaires were the most appropriate and valid measures available. Other measures did not cover such a breadth of events (e.g. List of Threatening Experiences, Brugha, Bebbington, Tennant, & Hurry, 1985), focussed more on daily 'hassles' (e.g. The Survey of Recent Life Experiences, Kohn & Macdonald, 1992), or were too lengthy (e.g. Life Events Questionnaire, Norbeck, 1984). Future research may benefit from the use of semi-structured interviews as an alternative assessment method.

When considering the design and analysis, it should be noted that mediation models inherently imply causality. Three primary criteria are required to establish causality: 1) the variables are associated, 2) the association is not spurious and 3) the cause precedes the effect (Hoyle & Smith, 1994; Menard, 1991). Non-experimental cross-sectional designs can only establish associations between variables (Frazier, Tix, & Barron, 2004). Therefore, while based on

theory, the causal and temporal relations of worry/rumination/life-events impacting the mediators, CF impacting EA, and the mediators impacting symptomology could not be empirically determined, nor the reverse relationships ruled out. Indeed, bidirectional relationships are quite possible (e.g. EA's rebound effect increasing worry/rumination; higher anxiety/depression increasing EA). To this effect, A. F. Hayes (2013) pointed out that that someone can often piece together a sensible argument supporting another direction of causal flow to the researcher's preferred model. One procedure to explore the possibility of alternative explanations is to test restructures of variable sequencing in mediation models. However, this was not thought appropriate in the present research for the following reasons. Firstly, even a simple three-variable mediation model has six possible directions of causal flow, with the more complex multiple mediation models currently tested having many more alternative formations. Multiple testing of all possible model structures would have led to insupportably high risk of error. Secondly, even where alternative models produce significant results, A. F. Hayes (2013) asserts that the author can only conclude that the data is simply uninformative about causal order and additional study using designs that better afford causal claims and directionality is required. Despite these limitations, non-experimental cross-sectional mediation studies are still considered useful as long as they are embedded in theory and previous research (A. F. Hayes, 2013), and are indeed the norm (Frazier et al., 2004). The present study therefore formulated and interpreted the mediational models based on ACT theory and previous research.

Future research should develop this preliminary work to empirically establish causal and temporal associations.

Controlling for prior levels of the DV, an almost ubiquitous 'third variable' confound, is deemed one of the most important benefits of longitudinal designs (Gollob & Reichardt, 1991). However, insufficient longitudinal data was obtained in the clinical sample. As discussed in Section 2.7, 'Procedures', average service waiting times between triage and participants' first appointment (i.e. T1-T2 time-delay) were shorter than expected based on a previous audit, due to unforeseen service and staff changes. Furthermore, a reflection with the team revealed time pressure was often a barrier to obtaining consent-to-contact on the triage call. In such instances, consent-to-contact had to be obtained when clients were later telephoned to be given their first appointment date, again reducing T1-T2 time-delays. In the majority of cases the time-delay was too short (less than 2-weeks) to be valid for use.

In the student sample, no significant longitudinal results were found, possibly due to short time-delays and insufficient power, as previously discussed. However, even if significant effects had been found, causality still could not be established given correlational data cannot rule out other 'third variable' causes (i.e. epiphenomenality, A. F. Hayes, 2013). Furthermore, the present study was a 'half-longitudinal design' (Cole & Maxwell, 2003), with IVs and mediators measured concurrently. Therefore, baseline CF/EA could not be controlled for.

Finally, this study tested a series of mediational models in two samples. Multiple testing increases the risk of Type I error. However, adjusting for Type I error can also elevate the risk of Type II errors to unacceptable levels, especially in underpowered studies (Nakagawa, 2004). Nakagawa (2004) rather suggests reporting confidence intervals for effects, as done presently. Nevertheless, further research is needed to replicate and verify our findings.

4.6   Future Research

Research had previously neglected CF's role in MH, and instead focussed on EA. This study has highlighted CF's potential in helping understand anxiety and depression. Both CF and EA should be included within future research using larger, more diverse samples to continue to explore their unique and interrelated roles in MH. The finding that CF was only uniquely associated with anxiety, and not depression, in the clinical sample (Hypothesis 1) needs further exploration, as well as the differences found between the clinical and student samples and in particular the lack of significant effects of EA in the student sample. ACT research would benefit from comparing matched clinical and non-clinical (extending beyond just students) samples, rather than assuming the processes underpinning MH in non-clinical populations will mirror those found in individuals with diagnosed MH conditions.

This research has highlighted the need to use more robust measurement tools when assessing CF and EA, moving away from the AAQ. These constructs are difficult to operationalise in that they refer to relationships to, and not the

content of, an individual's experiences. Despite this, the CFQ and BEAQ have proved useful in the absence of any other psychometrically sound questionnaires. However, given they are relatively new and therefore, as yet, underutilised, they may have unidentified psychometric weaknesses. Indeed, the BEAQ's internal consistency in the clinical sample (α=.66) was lower than previously documented (Gámez et al., 2014). Further research using these questionnaires is thus required before unequivocally claiming they are the gold standard measurements. Furthermore, recent research has highlighted a possible multidimensional structure to EA (McMullen et al., 2015), which would not have been possible to assess with the short BEAQ (Sahdra, Ciarrochi, Parker, & Scrucca, 2016). Indeed, the heterogeneous structure of EA may have contributed to the lower than expected internal consistency of the BEAQ presently found. Since the implementation of our research, another 30-item version of the MEAQ has been developed, shorter than the full scale but longer than the BEAQ (Sahdra et al., 2016). The MEAQ-30 captures the same subscales of EA as found in the full-scale MEAQ and may prove useful in future research to unpick how different EA dimensions differentially relate to outcomes.

As previously discussed, while the mediational models presently investigated were structured upon prior research and ACT theory, the design prevented causality from being determined. Therefore, before premature conclusions are made, longitudinal and experimental designs need to verify the temporal and causal relationships implicit within the mediational models. Furthermore, longitudinal research using larger samples and over longer time-delays would

help clarify the non-significant findings in the student sample. Indeed, the optimum time between re-measurement of study variables is unknown and it would be helpful for researchers to report effects for a variety of time intervals (Cole & Maxwell, 2003).

While this study was unable to employ Structural Equation Modeling (SEM) due to large sample size requirements (R. B. Kline, 2011), this might be a useful statistical method for future research. SEM provides model fit information testing the consistency of a hypothesised mediational model to the data and the plausibility of causal assumptions made (Gunzler, Chen, Wu, & Zhang, 2013). It also addresses the presence of measurement error within the statistical model (T. D. Little, Card, Bovaird, Preacher, & Crandall, 2007).

This study and the large proportion of research before it have relied on self-report measures. In light of the associated limitations, other methods including informant reports, behavioural measures and experimental designs should be used to verify the present findings. Additionally, retrospective measurement of stressful life-events could be advanced by measuring reactions to life-events as they unfold over time using experience sampling and diary methodology. This has proved to be a useful methodology in previous research (e.g. Kashdan et al., 2006; Machell, Goodman, & Kashdan, 2015).

Furthermore, while this research was interested in the overall construct of rumination, Treynor et al. (2003) found evidence for a bi-dimensional structure,

including reflective and brooding components. CF and EA may be particularly linked to the more maladaptive 'brooding' dimension. This should be further explored in future research. Additionally, while not the focus of this research, one surprising observation was that anxiety was more highly correlated with rumination (r=.53 [student], .38 [clinical]) than with worry (r=.40 [student], .36 [clinical]). While worry is typically associated with anxiety and rumination with depression, researchers have suggested that these processes may be relevant across both disorders (Harrington & Blankenship, 2002; McEvoy, Watson, Watkins & Nathan, 2013). Rumination and worry's shared function may underpin such transdiagnostic relationships, and CF and EA's role in these cross-correlations should be considered in the future.

It is likely that other unmeasured constructs may have partially accounted for additional variance in the relationships presently tested. Indeed, direct relationships between predictors and symptoms of anxiety and depression were found, independent of CF and EA. This study focussed on CF and EA as these have been conceptualised as the cornerstone of psychological inflexibility and mental ill health (Ciarrochi et al., 2010; S. C. Hayes et al., 2011). However, the other processes in the ACT model (e.g. attachment to the conceptualised self, lack of present moment awareness and values clarity) also deserve further consideration. This will help unpick their differential roles in MH and refine our understanding of the boundaries and interrelationships between these constructs. Studies should also be expanded beyond traditional outcome measures of distress and include indices of values-congruent living to more

accurately tap into this ACT conceptualisation of 'wellbeing' (S. C. Hayes et al., 2006). ACT views 'living better' as the key indicator of psychological wellbeing, rather than 'feeling better' (S. C. Hayes et al., 1999).

Finally, this research focused on anxiety and depression given their high prevalence and impact on society. The ACT assumption that CF and EA are transdiagnostic processes (S. C. Hayes et al., 2011) should be addressed further by replicating the current study across different diagnostically discrete clinical groups and across different predictors of MH. Further research of theoretically grounded and ecologically valid models will continue to advance knowledge around the complex interrelations among potential risk factors to psychopathology.

4.7   Conclusion

Further understanding the key processes involved in anxiety and depression helps identify optimum targets for prevention and treatment, thereby potentially reducing their unrelenting impact on society. Traditionally, core vulnerabilities to these common MH conditions have been formulated in terms of the content and nature of people's internal and external world. More recently, ACT has shown promise in advancing this understanding by instead turning our attention towards core unhelpful relationships (namely CF and EA) we may have with these difficult psychological and situational challenges (S. C. Hayes et al., 1999). However, despite its prominence in ACT theory, CF had previously been

neglected in research, and CF and EA's contributions to MH in the context of one another overlooked.

This study simultaneously considered both CF and EA in understanding anxiety and depression. While CF showed value in helping explain variance in symptomology as well as the impact of three topographically different vulnerabilities (worry, rumination and stressful life-events) across both samples studied, EA only exhibited explanatory value in the clinical sample and this was largely dependent on its interrelationship with CF. Differences in findings between the two samples studied could possibly indicate that CF is more predictive of variance in anxiety and depression symptoms towards the lower end of the MH continuum. Only when CF leads to higher levels of EA might clinical presentations of psychopathology emerge. However, given the research design could not establish causality and in light of the non-significant longitudinal results in the student sample, fully longitudinal and experimental designs are needed in more diverse, larger samples to avoid premature conclusions being made. Should the present preliminary findings continue to be replicated, then this would increase support for the use of ACT interventions targeting CF and EA to encourage people to relate to difficult internal and external experiences in a more helpful way that promotes valued living.

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APPENDICES

Appendix 1: Participant Information Sheet [Student Sample]

Participant Information Sheet

We are inviting you to take part in a research study. Before you decide whether to take part, take some time to read the following information about why the research is being done and what would be involved.

SUMMARY OF THE RESEARCH

We wish to understand more about why people feel the way they feel. We are particularly interested in a relatively new theory called Acceptance and Commitment Therapy (ACT). So far, research suggests that ACT is a helpful theory for understanding what influences our wellbeing. We would like to increase the knowledge in this area.

WHAT WOULD BE INVOLVED IF I DECIDED TO TAKE PART IN THIS RESEARCH?

Taking part involves completing a consent form and then a set of questionnaires online, which should take an average of 15 minutes to complete and will earn you one research credit.

We will also ask you to complete one of the questionnaires again in six weeks. We will send you an email when six weeks has elapsed to allow you access to complete this questionnaire. This will take a maximum of 10 minutes and is also completed online. This will earn you a further research credit.

All information given will be kept confidential and anonymous. Data collected will be saved as an encrypted and password protected file. Only the researchers will have access to these. We will ask for your email address at the end of the first part of this study, in order to email you to remind you to take part in the second part of the study. When we email you, we will also send you a unique personal code to use when completing part 2 of the study so that we can link your questionnaires completed at these two time points, while keeping the questionnaires anonymous.

WHAT ARE THE POTENTIAL BENEFITS AND DISADVANTAGES TO TAKING PART?

We hope that the information that we get from this study will help us to better understand why people feel the way they feel. We can send you a report of what we found in the study, which you may find interesting to read. Additionally you will receive research credits for participating.

We do not anticipate that taking part in the research will disadvantage you in any way. However, some of the questionnaires contain questions relating to difficult feelings, thoughts and experiences, which some people may find upsetting to think about. If you do decide to take part and are concerned about your mood, we would urge you to contact the University Counselling Service on welfare@royalholloway.ac.uk or 01784 443394/01784 443132 and/or your GP. The Royal Holloway website: https://www.royalholloway.ac.uk/ecampus/welfare/counselling/home.aspx also offers further information about how to access support.

WHAT WILL HAPPEN IF I NO LONGER WISH TO PARTICIPATE IN THIS STUDY?

You do not have to take part in this study if you do not want to and it is entirely voluntary. If you decide to take part, you may withdraw at any time without having to give a reason. This decision will not affect your education in any way. We will destroy any data you have already given, should you wish. If this is the case, please contact us using the contact details found at the bottom of this information sheet. Please note however that we hope to analyse the data in February 2016 and therefore after this point it may not be possible to withdraw your data from the study.

WHAT WILL HAPPEN TO THE RESULTS OF THIS STUDY?

We will write up the results of the study in a report to send to you, if you are interested. The study will also form part of a PhD thesis and we would hope to publish our findings in a scientific journal. Data included in any reports will be anonymous and you will not be identifiable.

FURTHER INFORMATION AND CONTACT DETAILS

If you have any questions, or wish to withdraw from the study at any point, please do not hesitate to contact Camilla Cookson (Trainee Clinical Psychologist, Chief Investigator) at pava049@live.rhul.ac.uk.

Appendix 2: Participant Information Sheet [Clinical Sample]

Understanding the factors that predict anxiety and depression.

Participant Information Sheet

We are inviting you to take part in a research study. Before you decide whether to take part, it is important you understand why the research is being done and what is involved in taking part. Therefore please take some time to read the following information. Whether or not you wish to participate is **entirely voluntary** and **will not affect the care you receive from this service**.

Summary of the research

We wish to understand more about why people experience anxiety and/or depression. We are particularly interested in a relatively new theory called *Acceptance and Commitment Therapy* (ACT). The ACT model proposes that the normal human mind often relates to difficult thoughts, feelings and experiences in an unhelpful way and that this can lead to anxiety or depression.

As a relatively recent theory and therapy, we are in the process of testing some of its ideas. So far, research suggests that ACT is a helpful theory for understanding mental health. We would like to increase the knowledge in this area, by looking in more detail at the relationship between ACT ideas and other factors that are known to predict anxiety and depression (stressful life events, rumination and worry). Understanding these relationships can help us to develop psychological treatments.

To do this, we ask participants to complete a set questionnaires, which ask about your thoughts and feelings, whether you have experienced any stressful life events in the last 12 months, and your experiences of anxiety and depression.

What would be involved if I decided to take part in this research?

Taking part involves completing a consent form and then completing a set of questionnaires, which should take approximately 30 minutes to complete. You can complete these online in a place of your choosing (e.g., at home) or we can send you paper copies in the post. There is telephone support for answering the questionnaires if required.

At your initial therapy session, Centre for Psychology ask all clients to complete a questionnaire as part of standard practice. We would like to use your answers to this questionnaire as part of the study as well. We will inform your GP that you are taking part in the study.

Please remember: whether you decide to take part or not will not affect the care that you receive from the Centre for Psychology service. The research and the care you receive from the service are separate and will not affect each other.

This research has been approved by the Research Ethics Committee.

The following flow chart summarises what you will be asked to do if you take part:

```
┌──────────────────────────────────────────────────────────────────┐
│   Fill out a set of questionnaires taking approximately 30 minutes │
│                          to complete.                              │
└──────────────────────────────────────────────────────────────────┘
                              ⬇

┌──────────────────────────────────────────────────────────────────┐
│  Before your first appointment all clients complete a              │
│  questionnaire. We would like your permission to use your answers  │
│  to this questionnaire for the study.                              │
│                                                                    │
│  PLEASE NOTE: Everyone who sees a therapist at Centre for          │
│  Psychology are asked to complete a questionnaire at their first   │
│  session. Therefore this questionnaire would be completed whether  │
│  or not you choose to participate in the research.                 │
└──────────────────────────────────────────────────────────────────┘
```

Who can take part in this research?

Anyone who plans to have psychology sessions at Centre for Psychology can participate in this research. However, as we only have questionnaires in English language, unfortunately participants must be fluent in English. We are looking for 80 people to take part.

What are the potential benefits and disadvantages to taking part?

While this project may not directly benefit you, we hope that the information that we get from this study will help us to better understand the kind of difficulties you may be experiencing. This information will help us to develop psychological treatments for people experiencing anxiety and/or depression. Additionally, we can send you a report of what we found in the study, which you may find interesting to read.

We do not anticipate that taking part in the research will disadvantage you in any way. However, it will require approximately 30 minutes of your time and some of the questionnaires contain questions relating to difficult feelings, thoughts and experiences, which some people may find upsetting to think about. **If you do decide to take part and you experience distress having completed the questionnaires, or if you are increasingly concerned about your mood, please contact Centre for Psychology on: 01483570765.**

Will the information I give be kept confidential?

All information will be kept confidential. We will give you a personal code to use when completing the questionnaires so that these are kept anonymous. Anonymised questionnaires will be kept in a locked cabinet. Answers to questionnaires will also be saved on a computer. This data will again be completely anonymous and will be saved as an encrypted and password protected file. Only the researchers will have access to these.

This research has been approved by the Research Ethics Committee.

The measures of anxiety and depression are used as part of standard practise within the service and therefore your therapist and the clinical team would have access to information disclosed on these questionnaires completed at your first therapy session.

What will happen if I don't want to carry on with the study?

Taking part is **entirely voluntary**. You can withdraw from the study at any point without giving any reason and without the care you receive from the Centre for Psychology service being affected. We will destroy any data you have already given, should you wish. If this is the case, please contact us using the contact details found at the bottom of this information sheet. Please note however that we hope to analyse the data in February 2016 and therefore after this point it may not be possible to withdraw your data from the study.

What will happen to the results of this study?

After we have finished the study, we will write up the results in a report to send to you, if you are interested. We are keen to get participants' feedback on this report (for example, to check it is written clearly) before circulating it to all participants. Please do let us know if you would be interested in consulting with us on this.

The results of this study will also form part of a PhD thesis and we would hope to publish our research findings in a scientific journal. We would like to assure you that **all the data included in any reports will be anonymous and you will not be identifiable.**

We would like to thank you...

We very much appreciate the time spent reading this information sheet and any time you give to participating in this research study. As a token of our appreciation, we will enter all participants into a price draw. Four participants will be selected at random to receive a prize. The available prizes are £50, £20 and 2x £10.

Further information and contact details

If you have any questions, or wish to withdraw from the study at any point, please do not hesitate to contact:
- **Camilla Cookson** (Trainee Clinical Psychologist, Chief Investigator) at pava049@live.rhul.ac.uk.
- **Dr Jessica Kingston** (Clinical Psychologist) at Jessica.Kingston@live.rhul.ac.uk or on 01784414105.

If at any point you feel distressed, then please call Centre for Psychology on **01483570765**. If you have a problem or complaint about the study, please call Dr John Newland on **01483570765**.

SUMMARY OF THE KEY INFORMATION:

❖ Research helps us to understand why people experience symptoms of anxiety and depression. This information helps us to develop psychological treatments.
❖ We are interested in understanding anxiety and depression, using ideas from a relatively new theory (ACT).
❖ Whether or not you decide to take part in this research will not affect the care that you receive from this service. Participation in this research study is fully voluntary.
❖ All information given during the research will be anonymous.
❖ If you choose to take part in the research study, we would ask you to complete a set of questionnaires. These take approximately 30 minutes to complete.
❖ Additionally, when you attend your first therapy session, you complete a questionnaire as part of treatment as usual and we would like to use the information that you give on this questionnaire.
❖ You are free to withdraw from the study at any time.

Appendix 3: Consent Form [Student Sample]

ROYAL HOLLOWAY UNIVERSITY OF LONDON

Study Part 1 Credit Pool

Consent Form

1. I confirm that I have read the information sheet for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily. •

   ○ Yes ○ No

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason and without my education being affected. •

   ○ Yes ○ No

3. I agree to take part in this study.•

   ○ Yes ○ No

| Back | Next | Cancel |

Appendix 4: Consent Form [Clinical Sample]

CONSENT FORM

Understanding the factors that predict anxiety and depression.

Name of Researchers: **Camilla Cookson and Dr Jess Kingston**

Please tick box

1. I confirm that I have read the information sheet for the above study. I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

2. I understand that my participation is voluntary and that I am free to withdraw at any time without giving any reason and without my care being affected.

3. I am happy to be contacted about the study by (*please tick at least one option*):

   Telephone ☐

   Letter ☐

   Email ☐

4. I am happy for my GP to be informed that I am taking part in this study. ☐

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

Name of Participant: _____

Date: _____

Signature _____

Appendix 5: Letter to GP [Clinical Sample]

Date: [Insert Date]

[Recipient]
[Address 1]
[Address 2]
[Address 3]

Dear Dr [Recipient],

RE: Name, D.O.B, address of client

I am contacting you to inform you that (name) has consented to participate in a research study taking place at the Centre for Psychology service. This research involves completing questionnaires (taking approximately 30 minutes) online or via post. We will also be interested in their responses to questionnaires measuring symptoms of anxiety and depression completed as part of standard practise at the Centre for Psychology service. This study has been granted ethical approval by the Brent NRES Committee.

Although your direct involvement is not requested, it is clearly important for you to be informed. Should you have any questions, comments or concerns, or would like more information about the research study, please do not hesitate to contact me ([pava049@live.rhul.ac.uk;](mailto:pava049@live.rhul.ac.uk) **01483570765**). The full research proposal and a summary report of our findings are available on request.

Yours sincerely,

Camilla Cookson (Chief Investigator)

Supervised by Dr Jessica Kingston (Research Supervisor)

Appendix 6: Demographic Information [Student Sample]

ROYAL HOLLOWAY UNIVERSITY OF LONDON

Study Part 1 Credit Pool

1. Please enter today's date•

   [_____] dd/mm/yyyy

2. Please select your gender

   ○ Male

   ○ Female

3. Please enter your age

   [_____]

4. Which of the following groups best describes your ethic origin:

   ○ White

   ○ Mixed / multiple ethnic groups

   ○ Asian / Asian British

   ○ Black / African / Caribbean / Black British

   ○ I prefer not to answer

   ○ Other ethnic group, please specify

   [   ]

5. What year of study are you in?

   [   ]

6. What course are you studying?

   [   ]

7. Do you have a history of mental health problems?

   ○ Yes

   ○ No

   ○ I prefer not to answer

   [Back]   [Next]   [Cancel]

Appendix 7: Demographic Information [Clinical Sample]

Questionnaire Booklet

Participant number: ……………………………………………

Date of completion: …………………………………………..

Please select your gender: *Male* ☐   *Female* ☐

Please state your age: ……………..

Which of the following groups best describes your ethic origin?

White	☐
Mixed / Multiple ethnic groups	☐
Asian / Asian British	☐
Black / African / Caribbean / Black British	☐
I prefer not to answer	☐

Other ethnic group, please specify ………………………………

What is your highest level of education achieved?

No academic qualifications	☐
GCSEs or equivalent	☐
A-Levels or equivalent	☐
Undergraduate degree	☐
Postgraduate certificate or diploma	☐
Master's degree	☐
PhD or doctoral degree	☐

Other, please specify …………………………

What is your current employment status?

Employed full-time	☐
Employed part-time	☐
Student	☐
Unemployed	☐
Retired	☐

Other, please specify ………………………………

Appendix 8: Penn State Worry Questionnaire
(PSWQ, Meyer et al., 1990)

Instructions: Rate each of the following statements on a scale of 1 ("not at all typical of me") to 5 ("very typical of me"). Please do not leave any items blank.

		Not at all typical of me				Very typical of me
1.	If I do not have enough time to do everything, I do not worry about it.	1	2	3	4	5
2.	My worries overwhelm me.	1	2	3	4	5
3.	I do not tend to worry about things.	1	2	3	4	5
4.	Many situations make me worry.	1	2	3	4	5
5.	I know I should not worry about things, but I just cannot help it.	1	2	3	4	5
6.	When I am under pressure I worry a lot.	1	2	3	4	5
7.	I am always worrying about something.	1	2	3	4	5
8.	I find it easy to dismiss worrisome thoughts.	1	2	3	4	5
9.	As soon as I finish one task, I start to worry about everything else I have to do.	1	2	3	4	5
10.	I never worry about anything.	1	2	3	4	5
11.	When there is nothing more I can do about a concern, I do not worry about it any more.	1	2	3	4	5
12.	I have been a worrier all my life.	1	2	3	4	5
13.	I notice that I have been worrying about things.	1	2	3	4	5
14.	Once I start worrying, I cannot stop.	1	2	3	4	5
15.	I worry all the time.	1	2	3	4	5
16.	I worry about projects until they are all done.	1	2	3	4	5

Appendix 9: Ruminative Response Scale

(RRS, Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema & Morrow, 1993)

People think and do many different things when they feel depressed. Please read each of the items below and indicate whether you almost never, sometimes, often, or almost always think or do each one when you feel down, sad, or depressed. Please indicate what you generally do, not what you think you should do.

almost never	sometimes	often	almost always
1	2	3	4

1. Think about how alone you feel ....

2. Think "I won't be able to do my job if I don't snap out of this" ....

3. Think about your feelings of fatigue and achiness ....

4. Think about how hard it is to concentrate ....

5. Think "What am I doing to deserve this?" ....

6. Think about how passive and unmotivated you feel. ....

7. Analyze recent events to try to understand why you are depressed ....

8. Think about how you don't seem to feel anything anymore ....

9. Think "Why can't I get going?" ....

10. Think "Why do I always react this way?" ....

11. Go away by yourself and think about why you feel this way ....

12. Write down what you are thinking about and analyze it ....

13. Think about a recent situation, wishing it had gone better ....

14. Think "I won't be able to concentrate if I keep feeling this way." ....

15. Think "Why do I have problems other people don't have?" ....

16. Think "Why can't I handle things better?" ....

17. Think about how sad you feel. ....

18. Think about all your shortcomings, failings, faults, mistakes ....

19. Think about how you don't feel up to doing anything ....

20. Analyze your personality to try to understand why you are depressed ....

21. Go someplace alone to think about your feelings ....

22. Think about how angry you are with yourself ....

Appendix 10: Life Events Scale for Students
(LESS, Clements & Turpin, 1996; Linden, 1984)

Please mark down whether any of these life events have happened to you during the previous year.
Please select as many life events as are applicable to you in the previous year.

- ☐ Death of parent
- ☐ Major personal injury or illness
- ☐ Major argument with parents
- ☐ Beginning an undergraduate program at university
- ☐ Moving away from home
- ☐ Getting an unjustified low mark on a test
- ☐ Failing a number of courses
- ☐ Minor violation of the law (e.g. speeding ticket)
- ☐ Getting kicked out of college
- ☐ Seeking psychological or psychiatric consultation
- ☐ Vacation alone/with friends
- ☐ Pregnancy (either yourself or being the father)
- ☐ Minor car accident
- ☐ Seriously thinking about dropping college
- ☐ Getting your own car
- ☐ Jail term (self)
- ☐ Moving out of town with parents
- ☐ Vacation with parents
- ☐ Establishing a new steady relationship with partner
- ☐ Finding a part-time job
- ☐ Sex difficulties with boy/girlfriend
- ☐ Failing a course
- ☐ Major change of health in close family member
- ☐ Major car accident (car wrecked, people injured)
- ☐ Death of your best or very good friend
- ☐ Family get-togethers
- ☐ Break-up of parent's marriage/divorce
- ☐ Losing a part-time job
- ☐ Major and/or chronic financial problems
- ☐ Major argument with boy/girlfriend
- ☐ Parent losing a job
- ☐ Switch in program within same college or university
- ☐ Losing a good friend
- ☐ Change of job
- ☐ Break-up with boy/girlfriend
- ☐ Minor financial problems

Appendix 11: Social Readjustment Rating Scale
 (SRRS, Holmes & Rahe, 1967)

INSTRUCTIONS: Please mark down whether any of these life events have happened to you during **the previous year**.
Please select as many life events as are applicable to you in the previous year.

	Please tick
Death of spouse	
Divorce	
Marital separation	
Jail term	
Death of a close family member	
Personal injury or illness	
Marriage	
Fired at work	
Marital reconciliation	
Retirement	
Change in health of family member	
Pregnancy	
Sex difficulties	
Gaining of new family member	
Business readjustment	
Change in financial state	
Death of a close friend	
Change to a different line of work	
Major change in the number of arguments with spouse.	
Major mortgage	
Foreclosure on a mortgage or loan	
Changes in responsibilities at work	
Son or daughter leaving home	
Trouble with in-laws	
Outstanding personal achievement	
Partner beginning or stopping work	
Begin or end school	
Change in living conditions	
Revision of personal habits	
Troubles with the boss	
Change in work hours or conditions.	
Change in residence	
Change in schools	
Change in recreation	
Change in church activities	
Change in social activities	
Minor mortgage or loan	
Change in sleeping habits	
Change in number of family get-togethers	
Change in eating habits	
Vacation	
Christmas	
Minor violations of the law	

Appendix 12: Cognitive Fusion Questionnaire
(CFQ, Gillanders et al., 2014)

α

Below you will find a list of statements. Please rate how true each statement is for you by circling a number next to it. Use the scale below to make your choice.

1	2	3	4	5	6	7
never true	very seldom true	seldom true	sometimes true	frequently true	almost always true	always true

1. My thoughts cause me distress or emotional pain	1 2 3 4 5 6 7
2. I get so caught up in my thoughts that I am unable to do the things that I most want to do	1 2 3 4 5 6 7
3. I over-analyse situations to the point where it's unhelpful to me	1 2 3 4 5 6 7
4. I struggle with my thoughts	1 2 3 4 5 6 7
5. I get upset with myself for having certain thoughts	1 2 3 4 5 6 7
6. I tend to get very entangled in my thoughts	1 2 3 4 5 6 7
7. It's such a struggle to let go of upsetting thoughts even when I know that letting go would be helpful	1 2 3 4 5 6 7

45

Appendix 13: Brief Experiential Avoidance Questionnaire
(BEAQ, Gámez et al., 2014)

Please indicate the extent to which you agree or disagree with each of the following statements

1----------------2----------------3----------------4----------------5----------------6

| strongly disagree | moderately disagree | slightly disagree | slightly agree | moderately agree | strongly agree |

		1 2 3 4 5 6
1.	The key to a good life is never feeling any pain	1 2 3 4 5 6
2.	I'm quick to leave any situation that makes me feel uneasy	1 2 3 4 5 6
3.	When unpleasant memories come to me, I try to put them out of my mind	1 2 3 4 5 6
4.	I feel disconnected from my emotions	1 2 3 4 5 6
5.	I won't do something until I absolutely have to	1 2 3 4 5 6
6.	Fear or anxiety won't stop me from doing something important	1 2 3 4 5 6
7.	I would give up a lot not to feel bad	1 2 3 4 5 6
8.	I rarely do something if there is a chance that it will upset me	1 2 3 4 5 6
9.	It's hard for me to know what I'm feeling	1 2 3 4 5 6
10.	I try to put off unpleasant tasks for as long as possible	1 2 3 4 5 6
11.	I go out of my way to avoid uncomfortable situations	1 2 3 4 5 6
12.	One of my big goals is to be free from painful emotions	1 2 3 4 5 6
13.	I work hard to keep out upsetting feelings	1 2 3 4 5 6
14.	If I have any doubts about doing something, I just won't do it	1 2 3 4 5 6
15.	Pain always leads to suffering	1 2 3 4 5 6

Appendix 14: Depression Anxiety Stress Scale
(DASS-42, S. Lovibond & Lovibond, 1996)

DASS

Name: *Date:*

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

The rating scale is as follows:

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

1	I found myself getting upset by quite trivial things	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I just couldn't seem to get going	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I had a feeling of shakiness (eg, legs going to give way)	0	1	2	3
8	I found it difficult to relax	0	1	2	3
9	I found myself in situations that made me so anxious I was most relieved when they ended	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting upset rather easily	0	1	2	3
12	I felt that I was using a lot of nervous energy	0	1	2	3
13	I felt sad and depressed	0	1	2	3
14	I found myself getting impatient when I was delayed in any way (eg, lifts, traffic lights, being kept waiting)	0	1	2	3
15	I had a feeling of faintness	0	1	2	3
16	I felt that I had lost interest in just about everything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I perspired noticeably (eg, hands sweaty) in the absence of high temperatures or physical exertion	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life wasn't worthwhile	0	1	2	3

Please turn the page 🖉

0 Did not apply to me at all
1 Applied to me to some degree, or some of the time
2 Applied to me to a considerable degree, or a good part of time
3 Applied to me very much, or most of the time

22	I found it hard to wind down	0	1	2	3
23	I had difficulty in swallowing	0	1	2	3
24	I couldn't seem to get any enjoyment out of the things I did	0	1	2	3
25	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
26	I felt down-hearted and blue	0	1	2	3
27	I found that I was very irritable	0	1	2	3
28	I felt I was close to panic	0	1	2	3
29	I found it hard to calm down after something upset me	0	1	2	3
30	I feared that I would be "thrown" by some trivial but unfamiliar task	0	1	2	3
31	I was unable to become enthusiastic about anything	0	1	2	3
32	I found it difficult to tolerate interruptions to what I was doing	0	1	2	3
33	I was in a state of nervous tension	0	1	2	3
34	I felt I was pretty worthless	0	1	2	3
35	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
36	I felt terrified	0	1	2	3
37	I could see nothing in the future to be hopeful about	0	1	2	3
38	I felt that life was meaningless	0	1	2	3
39	I found myself getting agitated	0	1	2	3
40	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
41	I experienced trembling (eg, in the hands)	0	1	2	3
42	I found it difficult to work up the initiative to do things	0	1	2	3

Appendix 15: Generalised Anxiety Disorder Assessment

(GAD-7, Spitzer et al., 2006)

Over the <u>last 2 weeks</u>, how often have you been bothered by the following problems? *(Use "✔" to indicate your answer)*	Not at all	Several days	More than half the days	Nearly every day
1. Feeling nervous, anxious or on edge	0	1	2	3
2. Not being able to stop or control worrying	0	1	2	3
3. Worrying too much about different things	0	1	2	3
4. Trouble relaxing	0	1	2	3
5. Being so restless that it is hard to sit still	0	1	2	3
6. Becoming easily annoyed or irritable	0	1	2	3
7. Feeling afraid as if something awful might happen	0	1	2	3

Appendix 16: Patient Health Questionnaire
(PHQ-9, Kroenke et al., 2001)

Over the last 2 weeks, how often have you been bothered by any of the following problems? (Use "✔" to indicate your answer)	Not at all	Several days	More than half the days	Nearly every day
1. Little interest or pleasure in doing things	0	1	2	3
2. Feeling down, depressed, or hopeless	0	1	2	3
3. Trouble falling or staying asleep, or sleeping too much	0	1	2	3
4. Feeling tired or having little energy	0	1	2	3
5. Poor appetite or overeating	0	1	2	3
6. Feeling bad about yourself — or that you are a failure or have let yourself or your family down	0	1	2	3
7. Trouble concentrating on things, such as reading the newspaper or watching television	0	1	2	3
8. Moving or speaking so slowly that other people could have noticed. Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual	0	1	2	3
9. Thoughts that you would be better off dead or of hurting yourself in some way	0	1	2	3

Appendix 17: Participant Debrief Letter [Student Sample]

Participant Debrief Form

You have now completed the second part of this two part study and have achieved a further research credit. Thank you for taking part in this study. We greatly appreciate the time that you have given to help us with our research. In this sheet, we have summarised the main aims of the study, should you be interested in knowing more.

This study aimed to develop our understanding of why we may feel more or less anxious or low in mood, using ideas from a relatively new theory, namely Acceptance and Commitment Therapy (ACT). ACT suggests that feelings of anxiety and low mood are not 'abnormal' - they are actually very common. ACT suggests that the way we relate to difficult thoughts, feelings and experiences may help us better understand why people may feel more or less low or anxious in mood. For example, we may:

- Dislike difficult thoughts and feelings and therefore try very hard to push them away, not think about them, and avoid places that trigger them. We might do this so much that we then stop doing things that used to make us happy and things that really matter to us (avoiding our experiences).

- Get really caught up with difficult thoughts and feelings so that it is hard to step back from them and get a new perspective (getting tangled up with thoughts and feelings).

In this study, we looked at both of these ideas and how they relate to feelings of anxiety and low mood. Specifically, we were looking at whether difficult life events and the tendency to ruminate (go over and over how you are feeling in your head) and worry are linked to feelings of anxiety and low mood because of the two ideas above.

For example, people respond very differently to similar life situations. ACT suggests that if people get very tangled up with their thoughts and feelings and/or excessively try to avoid their thoughts and feelings (e.g., by using drink, not going to certain places), then they are more likely to feel low in mood and/or more anxious.

Understanding this can help us to support people who are feeling distressed in mood in the most effective way.

The results of this study will form part of a PhD thesis. We also hope to publish our findings in a scientific journal. We would like to remind you that all the data in any reports will be anonymous (i.e., will not include any information that identifies you).

If you have any questions about the study or would like to receive a summary on what we found once we have finished the study, please do contact me on pava049@live.rhul.ac.uk. If you have any concerns about your mood, we would urge you to contact the University Counselling Service on welfare@royalholloway.ac.uk or 01784 443394/01784 443132 and/or your GP. The Royal Holloway website: https://www.royalholloway.ac.uk/ecampus/welfare/counselling/home.aspx also offers further information about how to access support.

Camilla Cookson (Trainee Clinical Psychologist; Royal Holloway University; pava049@live.rhul.ac.uk)
Supervised by Dr Jess Kingston (Clinical Psychologist)

Back   Done   Cancel

Appendix 18: Participant Debrief Letter [Clinical Sample]

DATE
ADDRESS

Dear _____ ,

Thank you for taking part in this study. We greatly appreciate the time that you have given to help us with our study. In this sheet, we have summarised the main aims of the study, should you be interested in knowing more.

This study aimed to develop our understanding of anxiety and depression using ideas from Acceptance and Commitment Therapy (ACT). ACT suggests that anxiety and depression are not 'abnormal' – they are actually very common. ACT suggests that the way we relate to difficult thoughts, feelings and experiences may help us understand anxiety and depression better. For example, we may:

- Dislike difficult thoughts and feelings and therefore try very hard to push them away, not think about them, and avoid places that trigger them. We might do this so much that we then stop doing things that used to make us happy and things that really matter to us (avoiding our experiences).

- Get really caught up with difficult thoughts and feelings so that it is hard to step back from them and get a new perspective (getting tangled up with thoughts and feelings).

In this study, we looked at both of these ideas and how they relate to anxiety and depression. Specifically, we were looking at whether difficult life events and the tendency to ruminate (go over and over how you are feeling in your head) and worry are linked to anxiety and depression because of the two ideas above.

For example, people respond differently to similar life situations. ACT suggests that if people get very tangled up with their thoughts and feelings and/or excessively try to avoid their thoughts and feelings (e.g., by using drink, drugs, not going to certain places), then they are more likely to feel anxiety and/or depression.

Understanding this can help us to build our therapeutic treatments.

If you are interested, we can send you a summary on what we found once we have finished the study. This would not be a summary of your responses, but of responses in general. Please do let us know if you would like this summary (pava049@live.rhul.ac.uk; **01483570765)**. We are also very keen to get some help on how we summarise our findings for the people that have taken part. If you would like to help us with this, please do contact us via email or letter. We would greatly appreciate your advice and thoughts.

The results of this study will form part of a PhD thesis. We also hope to publish our findings in a scientific journal. We would like to remind you that all the data in any reports will be anonymous (i.e., will not include any information that identifies you).

Finally, to thank you for participating in this research, we have entered your name into a prize drawer. At the end of the study, around March 2016, we will draw out four names from the prize drawer to receive a cash prize of £50, £20 or 2x £10. If your name has been selected we will contact you immediately to inform you of this.

If you have any questions about anything written in this letter or about the research or would like to provide us with any comments about taking part in this study, please do not hesitate to contact us. If you have experienced any distress having completed the questionnaires used in the study, or if you are increasingly concerned about you mood, please contact Centre for Psychology on: **01483570765**.

Yours sincerely,

Camilla Cookson   Dr Jess Kingston
(Trainee Clinical Psychologist)   (Clinical Psychologist)

Doctorate in Clinical Psychology
Department of Psychology
Royal Holloway, University of London
Egham, Surrey
TW20 0EX

Pava049@live.rhul.ac.uk (Camilla Cookson)
jessica.kingston@rhul.ac.uk (Dr Jess Kingston)

Appendix 19: Final Report for Participants

<u>**Understanding the factors that predict anxiety and depression**</u>

This study aimed to develop our understanding of two common mental health conditions: anxiety and depression. We know from past research that *worry* (fearing bad things will happen in the future), *rumination* (going over and over past upsetting events, unresolved concerns, and depressed symptoms) and *stressful life-events* can make people feel anxious and depressed. Our study wanted to understand how these factors impact how we feel, using ideas from a psychological therapy called "Acceptance and Commitment Therapy" ('ACT', Hayes, Strosahl, & Wilson, 1999). ACT believes that *all* humans experience difficult thoughts and feelings, often triggered by stressful life-events. From this perspective, ACT proposes that having difficult thoughts (including worry and rumination) and stressful life-events is not problematic, per se. It is only when we relate to these thoughts and life experiences in an unhelpful way that we might experience anxiety and depression. Two unhelpful ways people relate to their experiences in the ACT model are:

- ❖ **'Experiential Avoidance':** This describes disliking difficult thoughts and feelings and therefore trying very hard to push them away, not think about them, and avoid places that trigger them. Research has shown that pushing away difficult thoughts and feelings can often have a rebound effect, where we experience them even more. We may also try and avoid places or doing things that trigger difficult thoughts and feelings so much that we stop doing the things that make us happy and really matter to us.

- ❖ **'Cognitive Fusion':** This describes getting really caught up with difficult thoughts so that it is hard to step back from them and get a new perspective (getting tangled up with thoughts). Sometimes these difficult thoughts can also get in the way of pursuing the things in life we really value.

We first wanted to confirm whether experiential avoidance and cognitive fusion were both related to anxiety and depression. We next wanted to understand whether experiential avoidance and cognitive fusion might explain why rumination can lead to depression, why worry can lead to anxiety and why stressful life-events can lead to

anxiety and depression. We investigated this in two groups: people experiencing symptoms of anxiety and depression (a clinical sample) and university students.

Doing this research, we found:

❖ *When people got very caught up with their negative thoughts and frequently tried to avoid difficult experiences, they were more likely to experience anxiety and depression.*

- In the students group, getting tangled up with one's negative thoughts was particularly linked with feeling more anxious and lower in mood.
- For those experiencing anxiety and depression, the results suggested that getting caught up with negative thoughts motivated people to avoid difficult thoughts and emotions at all costs. This was associated with increased symptoms of anxiety and depression.

❖ *The way people related to their worries and ruminative thoughts, as well as their life-events, impacted how they felt.*

- In students, the results suggested worries, rumination and difficult thoughts triggered by stressful life-events lead to increased anxiety and lower mood, particularly when participants got very entangled with these thoughts.
- In the clinical sample, the results suggested that when people got very entangled with their worries, ruminative thoughts and difficult thoughts triggered by stressful life-events, they tried their hardest to avoid these negative thoughts and emotions at all costs. This was associated with increased anxiety and depression.

In conclusion, this study supported the role of experiential avoidance and cognitive fusion in anxiety and depression. It therefore follows that experiential avoidance and cognitive fusion may be important targets in the prevention and treatment of these mental health difficulties. ACT therapy teaches people techniques to help them to stop getting so caught up with their difficult thoughts and reduce cognitive fusion. ACT also helps people to become more accepting of difficult thoughts and feelings, which can reduce the rebound effect associated with experiential avoidance and increase valued living. Our research, together with previous research in this area, suggests this may reduce symptoms of anxiety and depression and have a positive impact on people's mental health.

Appendix 20: Royal Holloway University of London, Departmental Ethics

Committee Approval

2015/113 Ethics Form Approved

PS psychology.it.support@rhul.ac.uk
To: pava049@rhul.ac.uk; Kingston, Jessica; Cc: PSY-EthicsAdmin@rl...
Wed 14/10/2015 11:20

Reply all | v

Inbox

Application Details:	View the form click here Revise the form click here
Applicant Name:	**Camilla Cookson**
Application title:	**The mediating role of ACT processes in psychological distress: Revision 2**
Comments:	Approved.

Appendix 21: Brent Ethics Committee Approval

NHS
Health Research Authority

NRES Committee London - Brent
80 London Road
Skipton House
London
SE1 6LH

Telephone: 020 7972 2554

13 May 2015

Miss Camilla Cookson
Department of Psychology, Bowyer Building
Royal Holloway, University of London
Egham, Surrey
TW20 0EX

Dear Miss Cookson

Study title:	**Do experiential avoidance and cognitive fusion mediate the relationship between worry, rumination and stressful life-events (predictors) and psychological distress?**
REC reference:	**15/LO/0707**
IRAS project ID:	**178438**

Thank you for your letter of 11th May 2015, responding to the Committee's request for further information on the above research as submitting revised documentation.

The further information has been considered on behalf of the Committee by the Chair.

We plan to publish your research summary wording for the above study on the HRA website, together with your contact details. Publication will be no earlier than three months from the date of this favourable opinion letter. The expectation is that this information will be published for all studies that receive an ethical opinion but should you wish to provide a substitute contact point, wish to make a request to defer, or require further information, please contact the REC Manager, Ms Julie Kidd, nrescommittee.london-brent@nhs.net . Under very limited circumstances (e.g. for student research which has received an unfavourable opinion), it may be possible to grant an exemption to the publication of the study.

Confirmation of ethical opinion

On behalf of the Committee, I am pleased to confirm a favourable ethical opinion for the above research on the basis described in the application form, protocol and supporting documentation as revised, subject to the conditions specified below.

Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

You should notify the REC in writing once all conditions have been met (except for site approvals from host organisations) and provide copies of any revised documentation with updated version numbers. The REC will acknowledge receipt and provide a final list of the approved documentation for the study, which can be made available to host organisations to facilitate their permission for the study. Failure to provide the final versions to the REC may cause delay in obtaining permissions.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

Management permission ("R&D approval") should be sought from all NHS organisations involved in the study in accordance with NHS research governance arrangements.

Guidance on applying for NHS permission for research is available in the Integrated Research Application System or at http://www.rdforum.nhs.uk.

Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.

For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organisation.

Sponsors are not required to notify the Committee of approvals from host organisations

Registration of Clinical Trials

All clinical trials (defined as the first four categories on the IRAS filter page) must be registered on a publically accessible database. This should be before the first participant is recruited but no later than 6 weeks after recruitment of the first participant.

There is no requirement to separately notify the REC but you should do so at the earliest opportunity e.g. when submitting an amendment.   We will audit the registration details as part of the annual progress reporting process.

To ensure transparency in research, we strongly recommend that all research is registered but for non-clinical trials this is not currently mandatory.

If a sponsor wishes to request a deferral for study registration within the required timeframe, they should contact hra.studyregistration@nhs.net. The expectation is that all clinical trials will be registered, however, in exceptional circumstances non registration may be permissible with prior agreement from NRES. Guidance on where to register is provided on the HRA website.

It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).
Ethical review of research sites

NHS sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

Non-NHS sites

Approved documents

The final list of documents reviewed and approved by the Committee is as follows:

Document	Version	Date
Evidence of Sponsor insurance or indemnity (non NHS Sponsors only) [Professional Indemnity Policy]	1	08 April 2015
GP/consultant information sheets or letters [Letter to GP]	1	11 May 2015
IRAS Checklist XML [Checklist_08042015]		08 April 2015
IRAS Checklist XML [Checklist_11052015]		11 May 2015
Letters of invitation to participant [Letter of invitation to participant, version 1, 08.04.2015]	1	08 April 2015
Other [Participant Debrief Letter, version 1, 08.04.2015]	1	08 April 2015
Other [Clarification of Research Site Status]	1	08 April 2015
Other [Clarification of Non-NHS Research Site status, Insurance policy]	1	08 April 2015
Participant consent form [Participant Consent Form, version 1, 08.04.2015]	2	11 May 2015
Participant information sheet (PIS) [Participant Information Sheet, Version 1, 08.04.2015]	2	11 May 2015
Referee's report or other scientific critique report [Proposal submitted to Research Sub-Committee]	1	08 April 2015
Referee's report or other scientific critique report [Research Sub-Committee Provisional Approval and Comments]	1	08 April 2015
Referee's report or other scientific critique report [Response to comments from Research Sub-Committee]	1	08 April 2015
Referee's report or other scientific critique report [Research Sub-Committee Project Approval]	1	08 April 2015
Research protocol or project proposal [Research Protocol, version 1, 08.04.2015]	1	08 April 2015
Summary CV for Chief Investigator (CI) [CV Chief Investigator]	1	08 April 2015
Summary CV for student [CV for student (same as chief investigator)]	1	08 April 2015
Summary CV for supervisor (student research) [CV for Research Supervisor]	1	08 April 2015
Summary, synopsis or diagram (flowchart) of protocol in non-technical language [Flow chart of design, version 1, 08.04.2015]	1	08 April 2015

Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

After ethical review

Reporting requirements

The attached document *"After ethical review – guidance for researchers"* gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Notification of serious breaches of the protocol
- Progress and safety reports
- Notifying the end of the study

The HRA website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

User Feedback

The Health Research Authority is continually striving to provide a high quality service to all applicants and sponsors. You are invited to give your view of the service you have received and the application procedure. If you wish to make your views known please use the feedback form available on the HRA website:
http://www.hra.nhs.uk/about-the-hra/governance/quality-assurance/

HRA Training

We are pleased to welcome researchers and R&D staff at our training days – see details at
http://www.hra.nhs.uk/hra-training/

15/LO/0707	Please quote this number on all correspondence

With the Committee's best wishes for the success of this project.

Yours sincerely

PP

[signature: Kidd]

Chair

Email:nrescommittee.london-brent@nhs.net

Enclosures: "After ethical review – guidance for researchers"

Copy to: Ms Sharon Clutterbuck