Why do Psychological Treatments Work? A Process Analysis Comparing Cognitive Behavioural Therapy and Behavioural Activation in the Treatment of Depression

Submitted by Asha Ladwa to the University of Exeter as a thesis for the degree of Doctor of Philosophy in Psychology, March 2022

I certify that all material in this thesis which is not my own work has been identified and that any material that has previously been submitted and approved for the award of a degree by this or any other University has been acknowledged.

Signature ……

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Abstract

Depression is a debilitating and recurrent mental health problem. Although there are a number of effective psychological treatments for adult depression, around 50% of individuals do not recover (Cuijpers et al., 2021). To improve the effectiveness of these treatments we need to understand how they work. Previous research has identified times in treatment when there are patterns of discontinuous depression change and these times have been used to examine processes of change to further understand how treatments lead to depression change. The aim of this thesis was to build upon this research to further understand discontinuous depression changes in and outside of treatment, the processes of change surrounding these times of depression variability, and how they relate to treatment outcomes. This thesis primarily focused on two patterns of discontinuous change; rapid improvements in depression symptoms, known as 'sudden gains' (Tang & DeRubeis, 1999) and 'depression spikes' which are transient increases in depression symptoms (Hayes, Feldman, Beevers, et al., 2007). To examine this four studies were conducted. Study one investigated the rates, timing, and association with treatment outcomes of sudden gains and depression spikes in a large scale clinical practice dataset. Study two explored client cognitive and behavioural processes of change surrounding sudden gains in cognitive behavioural therapy (CBT) and behavioural activation (BA), and their association with treatment outcomes in a trial dataset. Study three used the same trial dataset to explore predictors of depression spikes in CBT and BA, and their relation to treatment outcomes. Study four focused on how cognitive and behavioural avoidance are associated with depression variability outside of treatment across a stressful life period in a student sample. The thesis ends with a discussion of the methodological,

theoretical, and clinical implications of the findings and suggestions for future research.

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Author's Declaration

This thesis contains four studies that are being prepared for journals. The first three studies in this thesis were collaboratively designed and conducted with colleagues at the University of Delaware, United States.

In study one (chapter two) Professors Heather O'Mahen (University of Exeter), Kim Wright (University of Exeter), Adele Hayes (University of Delaware) and I designed the study. All analysis and write up of the study was conducted by myself.

Within study two (chapter three) and three (chapter four) client processes and therapist strategies were coded from therapy tapes. Coding for study two was conducted by myself, Leigh Andrews (University of Delaware), Elizabeth Alpert (University of Delaware), and Jesse McCann (University of Delaware). Coding for study three was conducted by myself, Leigh Andrews, and Elizabeth Alpert. For both studies the coding was supervised by Professor Adele Hayes. Both studies two and three were designed by myself, Heather O'Mahen, Kim Wright, and Adele Hayes. All analysis and write up of the study was conducted by myself.

Study four was designed by myself, Heather O'Mahen, and Kim Wright. All analysis and write up of the study was conducted by myself.

Chapter One: Introduction

1.1 Major Depressive Disorder (MDD)

Major Depressive Disorder (MDD) is a debilitating and recurrent mood disorder that is characterised primarily by low mood and loss of interest. MDD is currently the single largest contributor to global disability (WHO, 2017). It is estimated that MDD affects around 4.7% (4.4-5%) of the global population (Ferrari et al., 2013) and has a lifetime prevalence between 7-21% (Kessler & Bromet, 2013). Gender differences in prevalence rates of depression are often observed across the literature with higher a prevalence of MDD in women compared to men (Whiteford et al., 2013). MDD is also associated with substantial economic and social burden. In the United States (US) alone between 2010 and 2018 the number of adults diagnosed with MDD increased from 15.5 to 17.5 million and the economic burden increased by 37.9% (from $236.6 billion to $326.2 billion) (Greenberg et al., 2021). Further, MDD considerably impacts on other aspects of life, including daily functioning, work, home life, social activities, and relationships (Brody et al., 2018; Lépine & Briley, 2011).

The substantial burden of depression can, in part, be attributed to the highly chronic (ten Have et al., 2018) and recurrent nature of MDD. Of the individuals who recover (no longer meet the diagnostic criteria for MDD) more than 50% will relapse within two years (Cuijpers et al., 2008; Vittengl et al., 2007). In a systematic review of recurrence of MDD in specialised mental health settings, Hardeveld et al. (2010) found the rate of recurrence of MDD was 60% after five years, 67% after 10 years, and 85% after 15 years. The authors conclude, "…in this population it is better to ask *when* instead of *whether* the patient will have a recurrence [of depression]."

(Hardeveld et al., 2010, p. 189). In a general population study it was estimated that the cumulative recurrence of MDD at 5 years is 13.2% and this increased at 10 years to 23.2% and to 42% at 20 years (Hardeveld et al., 2013). It is also the case that with each additional episode of depression there is a higher risk of recurrence (Eaton et al., 2008; Hardeveld et al., 2010; Moffitt et al., 2010; Mueller et al., 1999; Steinert et al., 2014). Furthermore, depression is highly co-morbid with physical problems (Kang et al., 2015) and other mental health problems such as anxiety disorders (Almeida et al., 2012; Hasin et al., 2018; Hirschfeld, 2001; Lamers et al., 2011), substance use disorders (Hasin et al., 2018), and Post-Traumatic Stress Disorder (PTSD) (Rytwinski et al., 2013).

Due to the complexity and heterogeneity of depression it is important to consider what treatment works for whom and why (Paul, 1967). Psychotherapy research has explored this question over the past 70 years in attempts to help personalise therapy approaches (Zilcha-Mano, 2019). With advances in statistical methods we are now able to examine individual patient level changes in depression symptoms across therapy, which can allow us to further investigate what works in treatment and whether individuals with certain characteristics (demographic or clinical) may be best suited to a particular psychotherapy. Another aspect of this is to understand *how* psychotherapy works and leads to reductions in depression symptoms. Understanding this can also help clinicians to make evidence informed, personalised judgements on suitable treatments for individuals with depression.

1.1.2 Diagnosis and Assessment of MDD

To diagnose mental health problems diagnostic systems are used. These provide a standardised definition of mental health disorders, including MDD, and are continually reviewed to incorporate new research evidence. The two most widely

used systems are the Diagnostic and Statistical Manual of Mental Disorders (DSM) produced by the American Psychiatric Association (APA), and the International Statistical Classification of Disease and Related Health Problems (ICD) developed by the World Health Organisation (WHO). Both the DSM and ICD diagnostic systems outline core symptoms of depression and define thresholds of severity and duration for an MDD diagnosis. While there is considerable overlap between the systems there is also some slight variation.

The DSM-IV (APA, 1994), used between 1994 and 2013, defines MDD as occurring if five or more A1-A9 symptoms (see Table 1.1), including at least one of A1 or A2 symptoms, have been present most of the day, nearly every day in the previous two weeks. The symptoms must cause significant distress or impairments in social, occupational, or in other important areas of functioning, and not be a direct cause of medication or a medical condition. Further, individuals must have no manic or hypomanic episodes and the symptoms must not be better accounted for by bereavement. In the latest update to the DSM (Fifth edition, DSM-V; APA, 2013) the bereavement criteria was removed. However, this thesis uses trial data in chapters three and four that assess MDD using the DSM-IV criteria and therefore this version of the DSM will be discussed. The ICD-10 (WHO, 1993) criteria for MDD broadly enquires about similar symptoms of depression to the DSM-IV, (i.e. depressed mood, anhedonia, weight or appetite changes, sleep problems, psychomotor activity, worthlessness, and suicidal ideation) but includes reductions in energy as a core symptom and loss of self-esteem or confidence as an additional symptom. In the ICD-10 depressive episodes are categorised into mild, moderate and severe. Similarly to DSM-IV criteria the symptoms must be present for at least two weeks.

Table 1. 1

Comparison of DSM-IV Major Depressive Disorder and ICD-10 Depressive Disorder Symptoms

	DSM-IV Major depression	ICD-10 depressive episode
Core symptoms	• Depressed mood (A1) • Markedly diminished interest or pleasure in all, or almost all, activities (anhedonia) (A2)	• Depressed mood • Loss of interest • Reduction in energy
Additional symptoms	• Appetite and/or weight change (5% change) (A3) • Sleep disturbance (insomnia or hypersomnia) (A4) • Psychomotor agitation or retardation (A5) • Fatigue or loss of energy (A6) • Feelings of worthlessness or excessive guilt (A7) • Diminished concentration (A8) • Recurrent thoughts about death, recurrent suicidal ideation, or actual suicide attempts (A9)	• Change in appetite with weight change • Sleep disturbance • Change in psychomotor activity with agitation or retardation • Unreasonable self-reproach or inappropriate guilt • Loss of confidence/self-esteem • Diminished ability to think • Recurrent thoughts of death or suicide

Number of symptoms for diagnosis	Minimal- above the minimum (5 symptoms) Moderate – between mild and severe Severe- several symptoms (more than 5)	Mild- 4 symptoms Moderate- 5-6 symptoms Severe -7+ symptoms
Impairment	Symptoms must cause significant distress or impairment of functioning	Not specified
Duration for diagnosis	2 weeks	2 weeks

Note. DSM-IV = Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition; the ICD-10 = International Statistical Classification of Disease and Related Health Problems, 10th Edition. Some descriptions of symptoms were shortened for the table

Within the literature there is debate as to whether mental health disorders, including depression are categorical or dimensional in nature (Bowins, 2015). The discussion and resolution of this debate is beyond the scope of the current thesis. Another consideration is how to assess depression and there are a variety of categorical and dimensional approaches used within the field. A number of assessment tools, which incorporate MDD diagnostic criteria, have been developed and validated to assess depression symptoms and are used in both mental health services and research settings. Clinician administered interviews are considered to be the gold standard. One example of a commonly used clinician administered

interview is the ‘Structured Clinical Interview for DSM-IV’ (SCID; First & Gibbon, 2004). The SCID is a semi-structured interview used to diagnose mental health problems including MDD and uses a categorical classification of depression symptoms. However, clinician administered interviews can be time consuming and costly and therefore briefer tools are often used to identify and monitor depression. The ‘Patient Health Questionnaire’ (PHQ-9; Kroenke et al., 2001) and the’ Beck Depression Inventory’ (BDI; Beck et al., 1961) are two examples of client self-report measures of depression which enquire about a range of depression symptoms that are used in the MDD diagnostic criteria; for example low mood, appetite changes and thoughts of self-harm/suicide. The PHQ-9 is currently used within England’s mental health service, the Improving Access to Psychological Therapies (IAPT) service to assess depression symptoms over the course of therapy and report depression treatment outcomes.

Further, there are a variety of ways in which to assess depression levels following treatment. Treatment response is defined as a clinically significant reduction of depression symptoms following a treatment, and a state of remission is a period in which an individual is asymptomatic, which may be followed by either relapse/recurrence or recovery (Frank et al., 1991). Relapse describes the return of depression symptoms following remission but before achieving full recovery, whereas recurrence is the onset of a new episode of depression following recovery. Recovery is defined as a sustained period of remission which signals the end of a depression episode (Frank et al., 1991). There are also a number of ways depression treatment response can be operationalised. Continuous and categorical classification of depression severity can be used to see if a certain threshold is met by the end of treatment. Additionally, the statistical or clinical significance of the

symptom changes can be assessed. A commonly use method to assess statistical change is examining reliable change in scores using the Reliable Change Index (RCI; Jacobson et al., 1984) which specifies the amount of improvement on a scale for an individual to be classed as reliably improved or deteriorated using thresholds of 'normal' and pathological populations. On the other hand, clinically significant change (Jacobson & Truax, 1991) assesses change from clinical to non-clinical ranges in symptoms. Data from non-clinical populations are used to establish a threshold at which symptoms are deemed to be non-clinical and a significant amount of clinical change has occurred. Trial studies tend to look at changes in continuous and categorical depression outcomes. In the current thesis continuous treatment outcomes are examined in studies one, two and three (chapters two, three, and four respectively). Reliable and clinically significant change are commonly used in healthcare evaluation and these outcomes are examined in study one (chapter two).

In summary MDD is a highly prevalent, debilitating, multifaceted mental health disorder. The significant impact of MDD demonstrates the need for effective treatments. The treatments currently used for MDD will be reviewed in the next section.

1.2 Treatments for Depression

In England, United Kingdom (UK), the National Institute for Health and Care Excellence (NICE) develops evidence-based guidelines for health and care, including treatment and management recommendations for depression in adults (over the age of 18 years). A revision of the guidelines for the management of depression are due to be published in June 2022 (NICE, 2022). However, as this revised guidance has not been released at the time of writing this discussion focuses on current guidance which was published in 2009. Within the current guidelines individuals with mild to moderate symptoms of depression are treated in primary care services, which include General Practitioner (GPs) and IAPT services who support patients and deliver psychological interventions according to a stepped-care framework (Clark, 2011). In IAPT's stepped care model the majority of individuals coming into the service are first given low-intensity therapies such as guided self-help, psychoeducation groups, or briefer forms of cognitive behavioural therapy (CBT). Individuals who have not benefited from adequate low-intensity treatment are 'stepped-up' to receive high-intensity treatments which are more intensive forms of treatment and include greater session lengths. Patients who present to the service with more severe forms of depression can sometimes go straight into high-intensity treatments. Some individuals who do not recover from treatment in primary care and/or have more severe and complex mental health problems are treated in secondary care mental health services, which include coordination of care, medication, and high-intensity psychological interventions.

Various evidence-based treatments are available for adult depression and treatment recommendations are dependent on the severity of the depression symptoms. Psychological therapies are recommended for mild to moderate

depression (NICE, 2009). For moderate to severe depression psychological therapies, or a combination of psychological intervention and antidepressant medication are recommended (NICE, 2009). Within primary care in England there are a range of evidence based psychological therapies available, including group therapy, counselling, CBT[1], and behavioural activation (BA). The current thesis focuses on counselling, group therapy and CBT (low- and high-intensity) in study one (chapter two) and then primarily focuses on high-intensity CBT and BA in studies two and three (chapters three and four respectively).

Counselling for depression typically involves discussions about the clients' feelings, emotions, relationships, patterns of behaviour and life events with the therapist being there to listen, empathise and challenge in order for the client to find better ways to cope (Pybis et al., 2017). The treatment is typically six to twelve sessions in length, delivered face-to-face and is recommended for mild to moderate depression symptoms. Counselling has been found to be effective in reducing depression symptoms and is comparable to CBT in primary care settings (Bower et al., 2003; Clark, 2011; Gyani et al., 2013; Pybis et al., 2017) but there is some indication that the effects may be better in the short, rather than long term (Bower et al., 2003).

Cognitive behavioural therapy is perhaps the most widely used and researched psychological therapy for depression. The goal of CBT is to modify

[1] The terms 'cognitive behavioural therapy' (CBT) and 'cognitive therapy' (CT) are often used interchangeably within the literature. The current thesis will focus on CBT which uses cognitive and behavioural strategies. Where authors describe the therapy as CT but the protocol states there is a behavioural element, this will be referred to as CBT. Where the therapy protocol is purely cognitive this will be referred to as CT.

dysfunctional and negative thoughts, as well as alter maladaptive behaviours to reduce depression symptoms (Beck et al., 1979). There is substantial evidence examining the effectiveness of CBT in reducing depression symptoms. Meta-analyses suggest CBT is superior in reducing depressive symptoms compared to waiting list or no treatment (Hofmann et al., 2012), control conditions such as wait list, care as usual (CAU) or pill placebo (Cuijpers et al., 2016; Cuijpers, Sijbrandij, et al., 2013), and treatment as usual (TAU) (Hedge's g = 0.70) (Watts et al., 2015). Some evidence suggests that CBT in conjunction with antidepressant medication (ADM) can be more effective than ADM alone (Cuijpers, Berking, et al., 2013) or CBT alone (Cuijpers, Oud, et al., 2021). Other research finds both CBT and ADM have comparable effectiveness in reducing depression symptoms (DeRubeis et al., 2005; Driessen & Hollon, 2010; Hofmann et al., 2012; Roshanaei-Moghaddam et al., 2011). Compared to other psychological treatments early meta-analyses suggested CBT was superior (Shapiro & Shapiro, 1982; Smith & Glass, 1977), but these have been criticised for comparing the effectiveness of CBT to inactive control conditions rather than active treatments (Baardseth et al., 2013) and failure to define the comparative therapies (Gloaguen et al., 1998). In comparison to studies of bona fide therapies, there is evidence that CBT is comparable for treating depression to a number of treatments (Baardseth et al., 2013; Barth et al., 2013; Butler et al., 2006; Cuijpers, Berking, et al., 2013; Wampold et al., 2002) including psychodynamic therapy, problem-solving therapy, interpersonal therapy (Cuijpers et al., 2008; Hofmann et al., 2012), social skills training, nondirective supportive treatment (Cuijpers, Berking, et al., 2013; Cuijpers et al., 2008), BA (Cuijpers et al., 2011; Cuijpers, Berking, et al., 2013; Richards et al., 2016), and counselling (Pybis et al., 2017). Nevertheless, CBT is considered to be a gold standard therapy for depression

(David et al., 2018) and is recommended by clinical guidance because of the large number of RCTs conducted as well as its efficacy.

There are a number of different ways of delivering CBT. Within IAPT services in England CBT is delivered in 'low' (low intensity CBT [LiCBT] or guided self-help group treatment) and 'high' (HiCBT) formats. HiCBT is offered to individuals with moderate to severe depression and is typically delivered weekly in a one-to-one, face-to-face format by high-intensity therapists who have completed two years of training in CBT (Clark, 2011). The HiCBT therapy lengths varies between 12 and 20 sessions of therapy. Low-intensity formats of treatment have been developed to widen access to psychological therapies while reducing resources (e.g. therapist time) and therefore becoming more cost effective for therapy services, but still achieve therapeutic gains for patients (Bennett-Levy et al., 2010; Bockting et al., 2016). Low-intensity interventions, like LiCBT and guided self-help group therapy, are for individuals with low to mild depression and typically involve shorter and fewer treatment sessions (six-eight sessions), a reduction of therapist time, and use of self-help materials (Bennett-Levy et al., 2010). There is variation between services in how LiCBT is delivered and can be in group-based format, guided self-help, or computerised CBT (Clark, 2011). Furthermore, non-psychological professionals who are specifically trained to deliver LiCBT treatments may also be utilised.

With regard to efficacy, LiCBT has been found to be more efficacious compared to waitlist or usual care control groups (Coull & Morris, 2011), beneficial for individuals with moderate and severe depression (Bower et al., 2013), and shows comparable effectiveness to counselling and problem solving therapy at reducing depression symptoms (Cape et al., 2010). However, some evidence suggests that the effects of LiCBT may not be long-lasting. In a sample of 439 individuals who

received LiCBT for depression and/or anxiety, Ali et al. (2017) found 53% of individuals relapsed within a year, with eight out of ten relapses occurring within the first six months after therapy. Similarly in a prospective longitudinal study of patients with depression and/or anxiety symptoms in IAPT receiving LiCBT, Delgadillo et al. (2018) observed 65.8% relapsed (occurring within 12 months following treatment end) or had a recurrence of symptoms (after 12 months) within 24 months of receiving LiCBT. In contrast, a meta-analysis found 29% of individuals who received HiCBT relapsed within a year of ending treatment (Vittengl et al., 2007). On the other hand, guided self-help group treatment encompass both psychoeducation about depression and utilise CBT principles (Coull & Morris, 2011; Delgadillo, 2018). A meta-analysis examining the effects of guided self-help and face-to-face therapies for depression and anxiety found they do not differ in terms of effectiveness or drop-out rates between the two formats of treatment (Cuijpers et al., 2010). Further, guided self-help has been found to be more effective for depression than TAU (Williams et al., 2013).

Another psychotherapy recommended for individuals with depression is BA. The aim of BA is to reduce depression symptoms by encouraging engagement in valued and reinforcing behaviours despite negative mood to counter a learned propensity to avoid positively reinforcing stimuli in one's environment (Martell et al., 2001). The current thesis focuses on the BA therapy protocol outlined by Martell et al. (2001). The therapy is a structured treatment and typically the number of treatment sessions range between 6-20 sessions (Clark, 2011; Ekers et al., 2008; Richards et al., 2016), but some have also delivered single sessions of BA (Gawrysiak et al., 2009; Nasrin et al., 2017; Read et al., 2016). Within therapy services BA is delivered by trained therapists, but BA has also been found to be

effective when delivered by non-specialists such as mental health nurses with no previous formal psychotherapeutic training (Ekers et al., 2011) and lay individuals (Arjadi et al., 2018; Ekers et al., 2011; Raue et al., 2019). Although originally developed to be an individual treatment, there is some initial evidence that BA is also effective in group formats (Kellett et al., 2017; O'Mahen et al., 2019; Porter et al., 2004).

With regard to treatment efficacy, BA has been found to be more effective at reducing depression symptoms than wait list, TAU and control groups (Ekers et al., 2014; Mazzucchelli et al., 2009; Stein, Carl, et al., 2021; Sturmey, 2009), as well as antidepressant medication (Ekers et al., 2014; Moradveisi et al., 2013). In meta-analyses, BA has been found to have equivalent effect sizes in reducing depression symptoms compared to other bona fide therapies including psychodynamic therapy, interpersonal therapy (Braun et al., 2013), and CBT (Braun et al., 2013; Mazzucchelli et al., 2009; Richards et al., 2016). Additionally, BA has also been found to have enduring effects on depression which are similar to CBT (Dobson et al., 2008; Lorenzo-Luaces & Dobson, 2019; Richards et al., 2016).

1.2.1 Improvement of Psychotherapies for MDD

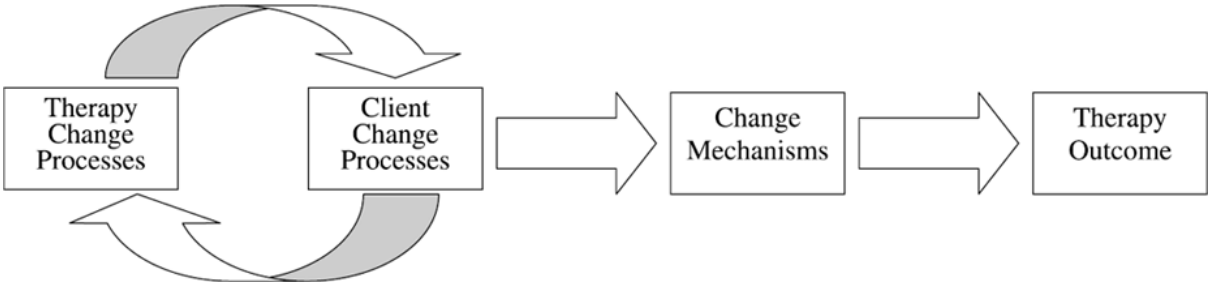
Although the evidence shows that psychotherapies for depression, in both RCT samples (Cuijpers et al., 2021; Cuijpers et al., 2008) and clinical service samples like IAPT (Wakefield et al., 2021) are effective in reducing depression symptoms there is still a substantial proportion (~50%) of individuals who do not achieve clinical recovery (Cuijpers et al., 2021; Cuijpers et al., 2014; Hollon & Ponniah, 2010; Novick et al., 2017). This is further exemplified within naturalistic settings, like IAPT where between April 2020 and March 2021 there were 1.46 million referrals (across disorders) to psychological therapies in England, of which

634,649 completed a course of psychological treatment (including counselling, group therapy, CBT and BA), but only 51.4% of individuals recovered (NHS Digital, 2021). Altogether this highlights the need to understand how therapies for depression work in order to continue to improve the efficacy of treatments.

One way to identify the causes of change in psychotherapy is the examination of ‘active ingredients’ within psychotherapy which focuses on identifying and examining which aspects of therapy may influence treatment outcomes (Doss, 2004). Alternatively there is also a focus on examining variables that change in the client such as adaptive or maladaptive processes which are a consequence of receiving therapy and reductions in depression symptoms (Doss, 2004). The different components of change in therapy are presented in Figure 1.1 from Doss (2004). Change processes refer to processes occurring during treatment or as a result of therapeutic homework and can be differentiated into therapy and client change processes. Therapy change processes occur directly from the therapeutic framework that is guided by the therapist and aims to result in a change of client processes (e.g. the therapist working with the client to identify and schedule pleasant activities in BA). Conversely client change processes are experiences or behaviours that take place as a result of the therapy change processes (e.g. the client engages in the pleasant activities in BA and feels enjoyment) which are expected to subsequently lead to changes in mechanisms (e.g. the client reduces depressive behaviours and increases valued, pleasurable behaviours) (Doss, 2004). Mechanisms of change explain how the skills learnt within the psychological intervention translate into generalised events in the person’s life which lead to desired therapeutic outcomes (i.e. reduction in depression symptoms) (Doss, 2004; Kazdin, 2007).

Figure 1. 1

Components of Change in Psychotherapy from Doss (2004)



The following section of the literature review will focus on outlining research to date that examines how CBT and BA work to reduce depression symptoms. Both treatments are closely related but have different theoretical underpinnings. One advantage of comparing processes of change between CBT and BA is that CBT contains some of the behavioural strategies used in BA, but BA proscribes any cognitive change strategies. Comparing processes of change across both treatments may allow inferences about when behavioural strategies alone are associated with change in depression symptoms, versus behavioural strategies plus cognitive strategies. This dismantling approach was highlighted in an influential component analysis by Jacobson et al. (1996) which demonstrated the behavioural activation component of CBT was not inferior in reducing depression symptoms to the full CBT package. In the study, 150 individuals with a DSM-III-TR diagnosis of MDD were randomly assigned to one of three treatment conditions; BA, BA and modification of automatic thoughts (this condition proscribes working on underlying core beliefs or schemas), or a full CBT package. Participants completed a minimum of 12 and maximum of 20 therapy sessions. The results showed all three conditions resulted in reductions in depressive symptoms which did not significantly differ at the end of treatment, six months (Jacobson et al., 1996), and two years follow up (Gortner et

al., 1998). Together the finding that CBT is not more effective than just the components by themselves called into question the necessity of using cognitive strategies to treat depression. It is important to note, however, that all three conditions contained elements of behavioural activation strategies and therefore the analysis was examining whether cognitive strategies in the presence or absence of behavioural activation strategies were effective in reducing depression symptoms.

Meta-analyses of studies show that CBT and BA are comparably effective at treating adult MDD (Braun et al., 2013; Ekers et al., 2008; Mazzucchelli et al., 2009; Shinohara et al., 2013). In the largest trial comparing CBT and BA for the treatment of adult MDD, Richards et al. (2016) found BA and CBT had similar effects on depression symptoms at the end of treatment and at 6, 12 and 18 month follow up. In one RCT study which examined BA in 241 individuals with depression, Dimidjian et al. (2006) found that in a subgroup of individuals with greater depression severity, BA outperformed CBT on the continuous (BDI) depression measurement. Less severely depressed individuals had similar response to CBT and BA (measured on the BDI) (Dimidjian, et al., 2006). In a follow-up study, Dobson et al. (2008) found BA and CBT performed similarly at two year follow up, suggesting both treatments have enduring effects on depression symptoms. In an attempt to replicate Dimidjian et al.'s (2006) findings, Lorenzo-Luaces and Dobson (2019) applied the same statistical analysis used by Dimidjian et al. to the data from Jacobson et al.'s (1996) trial and found, contrary to Dimidjian's findings, that CT and BA are comparable in reducing depression symptoms in individuals with high symptom severity. Despite the evidence that CBT and BA are comparably effective at reducing depression symptoms it is still unclear how CBT and BA operate to reduce depression symptoms. The comparable clinical effectiveness of CBT and BA does not

necessarily mean that there are shared processes of change leading to depression symptom alleviation in both treatments. Understanding the processes of change in CBT and BA that result in depression symptom change is imperative to continue to improve the effectiveness of these treatments.

1.3 Behavioural and Cognitive Theories of Depression and Hypothesised Processes of Change

Before reviewing the literature to date on the hypothesised processes of change for each therapy, the behavioural and cognitive theory of depression will be outlined as well as the treatment protocols for BA and CBT.

1.3.1 The Behavioural Theory of Depression, BA Therapy, and the Hypothesised Processes of Change

This section outlines the behavioural theory of depression and the BA protocol for depression. Additionally the research examining processes of change in BA is reviewed.

1.3.1.1. The Behavioural Theory of Depression.

Behavioural theories of depression explain the development and maintenance of depression as consequence of decreased environmental reward and positively reinforced interactions with one's environment, and the negative reinforcement of avoidance and passive behaviours (Ferster, 1973; Lewinsohn, 1974; Skinner, 1953). Depression is conceptualised as a change in context in an individuals' life (Martell et al., 2001) and the theory focuses on the importance of the interactions between an individuals' environment, their behaviours or actions, and the consequences of those actions.

Contemporary behavioural therapies (Martell et al., 2001) emphasise the importance of understanding the function of one's behaviours within an individuals' context. This originated from Skinner's behaviourism principles which suggested that depression results from a disruption of healthy behaviour sequences that used to be positively reinforced by an individual's environment (Skinner, 1953). Behavioural

theories suggest that we receive positive reinforcement from the environment around us and the activities that we engage in. However, when a change in context (i.e., a stressful life event, a transition) occurs the relationship between an individual's actions and the consequences of engaging in behaviours may change. What was once a behaviour or activity that provided an individual with positive reinforcement (regardless of whether that activity itself was perceived to be positive or not), or response-contingent positive reinforcement (RCPR) (Lewinsohn, 1975; MacPhillamy & Lewinsohn, 1974) and was followed by an increased likelihood in that behaviour occurring again, no longer elicits the same response. Additionally, an individual may engage in avoidance or withdrawal and this may be negatively reinforced so that the individual engages in this behaviour to prevent a negative outcome from occurring. Avoidance is viewed as a coping strategy which, in the short term, leads to temporary relief from aversive environmental stimuli. Avoidance can be conceptualised in both behavioural and cognitive domains. In behavioural theory there is an emphasis of behavioural avoidance where an individual withdraws or avoids valued activities that could provide them with sources of positive reinforcement. This is thought to both produce and sustain low mood. Although the content of covert, cognitive avoidance such as unproductive worry and rumination is not explicitly targeted in BA, the behaviour of worrying or ruminating is viewed as a barrier to engaging in valued activities that provide an individual with positive reinforcement and hence is targeted (Martell et al., 2001). In the absence of problem solving, which can help reduce or remove barriers to accessing positive reinforcement, increased avoidance leads to reductions of positive reinforcement within one's environment which further reduces mood and acts to negatively reinforce depressed behaviour (Martell et al., 2001). This negatively reinforced

behaviour may be increased in the future to avoid aversive stimuli and as an individual learns to behave in a way that focuses on alleviating aversive states, there is less opportunity to develop a range of behaviours that are positively reinforcing and this contributes to the narrowing of an individuals' repertoire of behaviour (Ferster, 1973). The theory emphasises that it is the environmental context which does not provide an opportunity to receive positive reinforcement (Ferster, 1981). This lack of engagement with positively reinforcing stimuli in one's environment leads to behavioural symptoms that are commonly seen within clinical presentations of depression such as withdrawal from valued and pleasurable activities, including social withdrawal, and increased behavioural avoidance (e.g. staying in bed). These behaviours are viewed as coping strategies to further avoid situations that provide low levels of reinforcement and in turn creates a vicious cycle of avoidance, chronically low levels of RCPR and depressed mood (Martell et al., 2001).

In support of this model of depression, research has found strong evidence for the association between low levels of positive reinforcement/environmental reward and greater depressed mood (Armento & Hopko, 2007; Grosscup & Lewinsohn, 1980; Hopko et al., 2003; Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972; MacPhillamy & Lewinsohn, 1974). In further support of the link between decreased reinforcement and low mood in a qualitative study, Hopko and Mullane (2008) found compared to non-depressed individuals, those with depression engaged less frequently in a range of different behaviours including social, physical, and education related activities. Early behavioural treatments further support the model by showing increasing pleasant events to attain positive reinforcement resulted in reductions in depression symptoms (Barrera, 1979; Lewinsohn et al., 1980). Neurobiological

evidence also finds deficits in reward functioning in individuals with depression (Forbes, 2020; Nagy et al., 2020).

In addition to approach deficits in depression, the behavioural model also emphasises the role of avoidance as a barrier to activation and experiencing positive reinforcement. There is good support for the association between increased avoidance and depression symptoms (Aldo et al., 2010; Carvalho & Hopko, 2011; Grant et al., 2013; Trew, 2011) and during times of stress individuals with depression are more likely to use avoidant or escape behaviours (Connor-Smith & Compas, 2002; Kuyken & Brewin, 1994; Penland et al., 2000).

1.3.1.2 BA Treatment and Treatment Protocol.

The behavioural theory of depression has led to the development of a number of different behavioural treatments (see Kanter et al., 2010 for an overview). Following Jacobson et al.'s (1996) influential component analysis which renewed interest in using behavioural therapy for depression, two contemporary behavioural activation protocols were developed for depression; BA (Martell et al., 2001) and Brief Behavioural Activation Treatment for Depression (BATD; Lejuez et al., 2001). While both behavioural treatments are grounded in traditional behavioural models of depression they utilise different strategies. The current thesis focuses on the behavioural activation protocol set out by Martell and colleagues.

The aim of BA therapy is to help the client to understand how their environment affects their mood and help them to make changes to their context to increase contact with positively reinforcing behaviours. This may be shifting goals, or changing how an individual responds to a life stressor by helping them to engage in more goal-directed proactive (rather than passive) behaviours using an 'outside-in'

approach (Martell et al., 2001). It is posited this will help increase natural sources of positive reinforcement in an individuals' environment. Within BA there is an emphasis on understanding the context in which certain factors influence desired and undesired behaviours- the study of this is known as 'functional analysis' (Ferster, 1973). Functional analysis is used to help to understand barriers to activation and engaging with sources of positive reinforcement. Furthermore in BA negative cognitions such as rumination are viewed as behaviours and with the continued focus on contextual factors, rather than engaging in the content of the thoughts, functional analysis is used to examine the context of the thoughts and the behavioural response to cognitions like rumination.

Within the current thesis, studies two (chapter three) and three (chapter four) utilise data from an RCT comparing CBT and BA for depression (COBRA trial; Richards et al., 2016) which uses a revised treatment manual which follows the standard BA set out by Martell and colleague (2001) with the addition of optional modules (Appendix 1). The COBRA trial required session-by-session manualisation of both therapies for the purposes of training and comparability. This BA protocol will be discussed here.

Initially within treatment the therapist will conduct an assessment to gather information on the patient's presenting problem and review the rationale for BA to treat depression. Goals are set at the beginning of treatment and are used to help identify further important activation targets (Kanter et al., 2010). Throughout therapy goals are continually reviewed.

The first phase of treatment aims to highlight the link between mood and behaviour. A number of different strategies are used to understand the function of an

individuals' behaviour, to increase awareness to these behaviours and how they are linked with mood. Formulations are used to understand the factors (e.g. life events) and coping behaviours that precipitate and maintain an individual's low mood and there is an emphasis on examining the contextual factors that contribute to low mood. Formulations can also help to identify avoidance or escape behaviours that narrow an individual's repertoire and facilitate the planning of alternative, approach behaviours. Additionally, activity monitoring is used early in BA to understand baseline levels of activity and mood, and to demonstrate the link between activity and mood. Throughout treatment behaviours are monitored to help the client identify behaviours that keep mood low and those that can increase positive mood. This is used to help facilitate behaviours to increase sources of positive reinforcement in the client's environment through activity scheduling. Activity monitoring charts are used to record behaviours and ratings of mood during that behaviour, on an hour-by-hour basis. The charts are then reviewed within therapy sessions with the therapist to highlight the mood and behaviour link.

Functional analysis is also emphasised in this BA protocol. Functional analysis is used to understand patterns of behaviours in certain contexts and determine under which contexts desired and undesired behaviours occur. Activity monitoring charts are used to identify and discuss patterns of behaviour that may be adaptive or maladaptive and under which contexts they occur. Two acronyms are used to help facilitate functional analysis of situations. The first is 'ABC' which is used to understand the antecedent or trigger of the situation, behaviours and the consequences of the behaviours in instances where mood is low. The second, a version of 'ABC', is 'TRAP' (trigger, response, avoidance- pattern) which helps to increase a client's awareness to cues (both internal and external) that result in a

negative emotional responses and behavioural avoidance. If a pattern of negatively reinforced behaviour is identified within functional analysis the therapist moves onto helping the client identify alternative healthy behavioural coping strategies to a response. Here the 'TRAC' (trigger, response, alternative-coping) acronym is used.

Another component of BA is activity scheduling. Self-monitoring helps to identify activity and mood links, functional analysis aids understanding of triggers and positive reinforcers in an individual's environment, and activity scheduling encourages the practice a of more adaptive behaviours. Activity monitoring charts are used as a basis to generate and schedule activities for clients to engage with. These may be new or old behaviours that an individual has learned to avoid. The therapist and client work together to schedule in activities likely to be associated with increased positive affect, or goal attainment, and typically through homework assignments a client will be able to test out these alternative behaviours and assess their mood. Clients are encouraged to experiment with the scheduled activities and rate their mood and feelings of achievement which are evaluated in therapy sessions. Ratings of mood and the frequency of activity can be compared throughout treatment to exemplify progress and the importance of continuing to review, expand upon activities, and experiment with behaviours that are valued and provide positive reinforcement.

Once an individual has started to re-engage in activity and avoidance has reduced, the second phase of the BA protocol moves to deliver mandatory and optional therapeutic modules. The mandatory modules focus on rumination and problem-solving. The rumination module aims to reduce repetitive negative thoughts. Functional analysis is utilised to identify the context in which rumination occurs and its function. Strategies are taught to develop alternative responses including more

concrete thinking. The problem-solving module aims to help clients reduce or remove barriers to accessing positive reinforcement. The module takes a step-wise approach to help develop strategies to recognise the problem in concrete terms, identify alternative behaviours, and test and implement alternative behaviours. Following these mandatory modules clients are given a choice of optional modules to aid recovery, which in this protocol includes finding functionally equivalent behaviours within a BA framework, strategies to help with anxiety, punishment, communication, alcohol and/or substance use.

The last phase of BA treatment is to highlight that the treatment is coming to an end, review therapy goals, and to self-plan without the support of the therapist to help maintain clinical progress. Early warning signs of relapse are discussed and the tools that have been used throughout treatment (e.g. TRAP/TRAC) are used to plan further mood enhancing activities. Within this BA protocol, clients were also offered the option of up to four booster sessions to help maintain clinical gains, practice skills, discuss any barriers or difficulties in implementing BA strategies or to activities to help with relapse prevention.

1.3.1.3 Processes of Change in Behavioural Therapy.

The predominate focus of research has been examining the effectiveness of BA in reducing depression symptoms and despite strong evidence of this (Cuijpers et al., 2007; Ekers et al., 2014; Mazzucchelli et al., 2009; Moradveisi et al., 2015) and the clear theoretical rationale of BA, relatively little research has explored the processes that drive symptom change in BA. The importance of activation (and implicit within this is the reduction of avoidance) and positive reinforcement is highlighted in behavioural theories of depression (Ferster, 1973; Lewinsohn, 1976;

Lewinsohn et al., 1976; Lewinsohn et al., 1970; Trew, 2011) and are important therapeutic targets in BA for depression (Martell et al., 2001). Another important consideration of these mechanisms of change is that they are within a valued domain to that individual. To receive positive reinforcement from the engagement of a behaviour (activation) the behaviour itself needs to be one that the individual values. Thus, from the theory and the focus of BA treatment we would expect that activation, positive reinforcement and increased activity in a valued domain would be mechanisms of change in BA for depression. The current empirical evidence for each of these mechanisms of action and the association with reductions of depression will be reviewed.

Activation is the engagement of behaviour that provides an individual with a range of sources of positive reinforcement and reduces avoidance (Manos et al., 2010). In support of activation being a mechanism of change in BA, the association between increased activation and depression symptom reduction has been evidenced in several studies of BA (Bailey & Arco, 2010; Hopko et al., 2003; Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972; MacPhillamy & Lewinsohn, 1974). For instance, in a single session of BATD treatment for 46 (22 in BA treatment; 22 wait list control) individuals with MDD, Nasrin et al. (2017) observed a small effect for increases in activation (measured on the Behavioural Activation for Depression Scale) mediating the effect between treatment condition and depression change (pre-post treatment). Similarly in a sample of 43 depressed individuals receiving BA for depression, Petts et al. (2016) found changes in activation measured on the Behavioural Activation for Depression Scale-Short Form (BADS-SF) were associated with reductions in depression symptoms only during the intervention and not during a baseline phase.

However within these studies the temporal relationship between activation and depression cannot be assessed. According to the BA theory, activation should precede depression changes (Kanter et al., 2010). Some research with small sample sizes provides support for activation temporally preceding depression changes. For instance, in a study of four moderately depressed adolescents using a single subject mediation analysis design, Gaynor and Harris (2008) observed over the course of BA treatment that increased activation was followed by subsequent decreases in depression symptoms in half of the individuals. Similarly, in another small study (*n* = 2) examining the use of the BADS-SF Manos et al. (2011) examined cross-lagged correlations between activation measured on the BADS-SF and depression. For one client change in activation preceded change in depression scores one week later, whereas for the other client there was a concurrent correlation between activation and mood change. Examining this relationship in an RCT of BA compared to TAU for depressed pregnant women, Dimidjian et al. (2017) found over the course of treatment women in BA, compared to TAU, reported higher levels of BADS-SF activation. Increases in activation significantly mediated the relationship between baseline depression and lowered subsequent depression symptoms in BA (Dimidjian et al., 2017). In another trial sample of 43 patients meeting MDD (DSM-IV-TR) criteria who were randomised to BA for Latin speaking communities or TAU, Santos et al. (2017) assessed the cross-lagged correlations to see whether activation preceded depression change or was a consequence of depression reductions. The results showed that in 79% (*n* = 11/14) of clients in the BA condition changes in activation preceded or co-occurred with depression reductions and no clients in TAU showed this association between activation and depression symptom change.

However, it is also the case that research finds concurrent changes in activation and mood, no association, or that mood changes precede activation. In a study of a 10-session BATD treatment adapted for Spanish speaking individuals Collado et al. (2014) found over the course of treatment as activation levels increased depression levels concurrently decreased. Similarly using a multiple baseline study design, Folke et al. (2015) examined approach changes in BA for six inpatients with depression symptoms and other psychiatric disorders including obsessive compulsive disorder (OCD), PTSD, mania, generalised anxiety disorder (GAD), borderline personality disorder (BPD) and schizophrenia. Daily changes in activation preceded or concurrently changed with depression symptoms for half of the patients, whereas hourly diary ratings showed that mood preceded changes in activation. This highlights that it is important to consider the timing and frequency of measuring activation levels in research. In a recent study Hoyer, Hoefler, et al. (2020) used time-lagged analyses to investigate the temporal relationship between activation and mood in a sample of 160 individuals with unipolar depression receiving group BA. Both activation (measured on the BADS) and depression symptoms were measured at the beginning of each session. They found that greater activation scores predicted subsequent reductions in depression symptoms, *and* lowered depression scores predicted higher subsequent activation scores. Further, in a longitudinal design across eight sessions of BATD for 23 breast cancer patients with diagnosis of MDD, Ryba et al. (2014) found no association between the number of activities completed and reductions in depression. Engagement in activities was not associated with self-reported environmental reward, and environmental reward did not mediate the relationship between activation and depression symptoms.

The evidence to date suggests that activation may be a mechanism of change within BA, but it is difficult to assess because of methodological differences between studies. In a recent systematic review examining mediators in BA treatment for depressive symptoms across ages, Janssen et al. (2021) identified 14 studies that performed a formal test of mediation, 10 of which were in RCT settings. They found that most of the studies had examined activation as a mediator but only two studies (Dimidjian et al., 2017; Hopko et al., 2016) demonstrated a mediation effect. The authors note that differences in study design and quality made it difficult to assess the strength of this mediation effect. It is also the case that other methodological differences make comparison between studies difficult. Often studies have small sample sizes (e.g. Folke et al., 2015; Manos et al., 2011), utilise different age groups (e.g. Gaynor & Harris, 2008), the treatments have different lengths (e.g. Hoyer, Hoefler, et al., 2020; Nasrin et al., 2017), and use different behavioural activation therapy protocols (Lejuez et al., 2001; Martell et al., 2001). Furthermore the studies use different time frames to assess activation as a mechanism of change and depression. For instance Folke et al. (2015) demonstrated differences in the relationship between activation and mood when participants rated hourly or daily, whereas in the study by Santos et al. (2017) participants rated activation over the previous week. This highlights the importance of understanding when to examine mechanisms of change in treatment.

Related to understanding whether activation is a mechanism of change in BA is also the considerations of barriers to activation. If activation and avoidance are on the opposite ends of a continuum, with an increase in activation there is also a decrease in avoidance to the same behaviour. In the behavioural activation model avoidance serves as a barrier to activation and accessing sources of positive

reinforcement (Ferster, 1973) which is important both within the development and maintenance of depression. There is empirical evidence which shows the links between avoidance and depression (Aldao et al., 2010; Carvalho & Hopko, 2011; Trew, 2011) but less so within the context of BA. Within the literature the BADS scale, which is widely used to assess activation, has a subscale that enquires about avoidance symptoms, but less research has looked at the role of reduced avoidance (either in the behavioural or cognitive domain) and the impact on mood symptoms in BA (Manos et al., 2010). In the limited evidence available, Nasrin et al. (2017) found no evidence that a single session BATD intervention lead to reductions in self-reported cognitive avoidance (i.e. rumination and experiential avoidance). However, further research is needed to examine the role of avoidance, both behavioural and cognitive, in BA for depression.

The behavioural activation model of depression also highlights the role of reduced positive reinforcement (also referred to interchangeably in the literature as 'environmental reward') in the development and maintenance of depression (Ferster, 1973; Lewinsohn et al., 1980). Positive reinforcement is the process by which the likelihood of a behaviour being repeated is increased due to previous acts of that behaviour being followed by positive consequences. Within BA therapy, through activity monitoring and scheduling, the goal is to increase engagement with sources of positive reinforcement within an individuals' environment, suggesting this is another mechanism of change in BA. The role of positive reinforcement has been highlighted by several studies. For example in an RCT of single session BATD compared to a non-treatment control for university students with depression, Gawrysiak et al. (2009) observed a strong relationship between greater positive reinforcement, measured on the Environmental Reward Observation Scale (EROS;

Armento & Hopko, 2007) and reductions in depression symptoms. Furthermore, within an RCT of BA compared to TAU for depressed pregnant women, Dimidjian et al. (2017) found over the course of treatment women in BA compared to TAU reported higher ratings of environmental reward and this predicted subsequent decreases in depression symptoms in BA compared to TAU. Another study assessed depression, activity engagement and environmental reward at each session in a 10-session BATD treatment adapted for Spanish speaking individuals (Collado et al., 2014). They found over the course of treatment depression symptoms decreased and environmental rewards increased. Lagged analyses provided evidence for a temporal association between environmental reward and depression symptom reduction; increased contact with environmental reward preceded depression symptom reduction in the next therapy session (Collado et al., 2014). Positive reinforcement has also been found to mediate the relationships between activation/avoidance and depression symptoms. In one study Takagaki et al. (2016) found positive reinforcement mediated the relationship between activation and depression symptoms in a sample of depressed adolescents receiving a behavioural activation treatment that focuses on solely increasing access to positively reinforcing activities. In another study Carvalho and Hopko (2011) found that positive reinforcement mediated the relationships between avoidance (cognitive, behavioural and total avoidance) and depression symptoms. This further strengthens the support for positive reinforcement being an influential process of change.

Positive reinforcement may lead to greater activation and subsequent reductions in depression symptoms through improved reward functioning. Reward functioning describes an individual's tendencies to seek out, anticipate and respond to rewarding stimuli (Forbes, 2020). Only one study to date has examined neural

reward changes following BA treatment. In this study Dichter et al. (2009) assessed brain activation to a reward choice selection task in 12 patients with MDD who received BATD treatment and 15 individuals without MDD. They found individuals with MDD following the BATD treatment, relative to the non-depressed group, had increased activation in brain structures when anticipating reward (in the dorsal striatum) and during reward selection (in the paracingulate gyrus). Although only preliminary and in need to replication, this suggests that BA treatment may increase activation of neural circuits that are relevant to reward functioning.

Although this research supports the link between reinforcement and depression reductions, as Manos et al. (2010) highlights there are methodological limitations with trying to measure behaviour and reinforcement and much of the previous research examining reinforcement (both positive and negative) focuses on the amount of behaviour change over time. Integral to this is the assumption that if an individual chooses to engage in a behaviour more frequently then this must have provided some positive reinforcement (Manos et al., 2010). However, there are also difficulties with measuring behaviours that are functional in producing contact with sources of positive reinforcement as simple lists of activities do not take into account an individuals' own goals and valued activities. Instead reinforcement is measured through other indicators or proxy variables, such as the relationship between pleasant events or daily diaries (proxy for positive reinforcement) and mood changes. Another concern here is understanding the timing between reinforcement and mood changes and this may be contingent on the measure used or the type of reinforcement assessed.

It is also the case that simply engaging in activities that appear pleasant and enjoyable does not necessarily provide positive reinforcement and research

suggests that simply increasing pleasurable activities does not necessarily alleviate symptoms of low mood (Hammen & Glass, 1975). Instead an important consideration is the relevance of the behaviours to the individual given their current life circumstances and values. Engaging or re-engaging in behaviours that are meaningful and valued (Santos et al., 2021; Stein, Tian, et al., 2021) rather than simply increasing activity may increase the chances of gaining positive reinforcement from the behaviour (Martell et al., 2001). Although this is recognised in BA treatment manuals (Martell et al., 2001) relatively little research has examined the importance of valued activities and depression symptom reductions in BA. This has been briefly illustrated in a single-participant assessment of values based BA treatment for adolescent depression by Gaynor and Harris (2008) where in two out of four patients, an increase in valued-driven activities was associated with decreases in depression symptoms. Outside the BA and depression literature, increasing engagement or re-engagement with valued behaviours precedes change in symptoms, not vice versa, in individuals receiving acceptance and commitment therapy (ACT) for panic disorder (Gloster et al., 2017). This association between increases in values-driven behaviour and reductions in depression symptoms in ACT has also been observed (Bramwell & Richardson, 2018). In CBT, which includes some behavioural activation techniques, increases in valued-orientated behaviours were also found to precede depression symptom changes (Hoyer, Čolić, et al., 2020). Outside psychotherapy in the social identity literature, greater social identification is a strong predictor of good mental health, wellbeing, and reductions in depression symptoms (Cruwys et al., 2014). This suggests that engaging in activities that are congruent with one's social identity and values is important for mood. Although this area is in need of further research, altogether it suggests that activation

of valued and meaningful activities provide positive reinforcement in BA and help to reduce depression symptoms.

Although it is hypothesised that BA works through behavioural processes of change it is conceivable that, even in the absence of cognitive change procedures, cognitive change processes may work to alleviate depression symptoms in BA (Lorenzo-Luaces et al., 2015). In support of this Bolinski et al. (2018) observed large reductions of maladaptive cognitions (dysfunctional attitudes) in early BA therapy (between sessions 1-4) which coincided with a large amount of depression symptom change. Similarly Lee et al. (2021) found a BATD treatment for depression was associated with reductions in dysfunctional attitudes from pre-post treatment. Further, in Jacobson and colleagues' (1996) dismantling study of CBT, reductions in negative attributions in early treatment was followed by reduction in depressive symptoms in later treatment in BA. However, other studies have failed to evidence of cognitive change in BA (Janssen et al., 2021). Although other research examines baseline levels of cognitive process (e.g. dysfunctional attitudes) to see whether it predicts depression symptom change in treatment (e.g. Hunnicutt-Ferguson et al., 2012; O'Mahen et al., 2021) this does not elucidate whether cognitive change in BA treatment may explain depression change. Further research is needed to examine whether in-session cognitive processes in BA may facilitate depression changes in the absence of cognitive change procedures.

In summary, compared to the research on the efficacy of BA there has been relatively little research that has examined processes of change in BA. Much of the available research in this area is conducted in small sample sizes and the temporal precedence between processes of change and depression symptom reductions has not been definitively established. Comparability between studies is difficult because

different BA protocols have been used and because of methodological challenges with measuring processes of change. Additionally, more research is needed to understand whether cognitive change processes may facilitate depression change in BA.

1.3.2 The Cognitive Theory of Depression, CBT, and the Hypothesised Processes of Change

Here the cognitive theory of depression and CBT protocol for depression will be outlined before reviewing research examining processes of change in CBT.

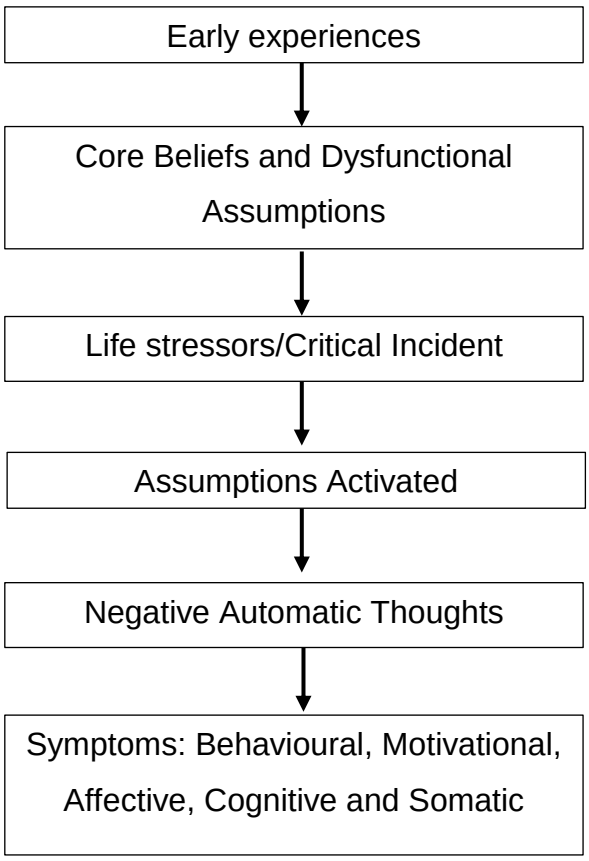
1.3.2.1 The Cognitive Theory of Depression.

The cognitive model of depression (Figure 1.2) suggests that through various influences in an individual's life such as early experiences, parental and peer influences, representations about the self, others and the world or schemas are developed (Beck et al., 1979). While these perceptions may be accurate for some, others may also hold distorted, negative schemas which are a result of negative experiences or messages in early life and remain stable over time. Schemas are organising cognitive frameworks that influence the representations of experiences. The cognitive theory of depression proposes that negative schemas may lie dormant, becoming activated and salient when life stressors occur (Beck, 1967; Ingram et al., 1998; Segal & Ingram, 1994). This can be thought of as a diathesis-stress model, in that if the vulnerability (in this case cognitive diathesis, but can also include others like genetic vulnerability) interacts with life stressors this results in psychopathology (Beck, 1967). For depression it is posited that a stressful life event triggers underlying negative schema which results in maladaptive information-processing styles that are thought to be key to both the development and

maintenance of depression (Beck et al., 1979). For example if an individual believes that they are unlovable due to negative early experiences with their caregivers and they break up with their partner (life event) this negative schema becomes salient. Negative schemas, which encompass core beliefs and dysfunctional attitudes, lead to information processing biases which influence the interpretation of experiences in a given context through biases in attention, memory and reasoning processes. These maladaptive interpretations may be about the self, world or the future (the negative cognitive triad) (Clark et al., 1999). As a consequence of information processing biases, individuals may be more likely to attend to stimuli and recall information that confirms their negative biases and reason about events more negatively. Dysfunctional attitudes reflect conditions under which the core belief would be shown to be true and are highly rigid, generalised conditional rules that individuals adopt. At the surface level negative automatic thoughts (NATs) are cognitions that are involuntarily activated in situations as a result of information processing biases and in depression, are frequently overgeneralised. NATs are proposed to maintain depression by increasing negative mood, prompting withdrawal and avoidance behaviours, exacerbating physical symptoms of depression, reducing motivation, and prompting cognitive symptoms of depression such as poor concentration. These aspects then interact, producing a vicious circle of depressive symptoms (known as the maintenance aspect of the model).

Figure 1. 2

A Schematic of Beck's Model of Depression (Beck, Rush, Shaw, Emery, 1979)



There is considerable empirical evidence for different aspects of the cognitive model of depression. Lending support to the diathesis-stress component of the model, the activation of negative schemas following life events have been found across the lifespan in children, adolescents and adults (Jacobs et al., 2008; Scher et al., 2005). For example, in a direct test of the diathesis- stress model, Abela and D'Alessandro (2002) found dysfunctional attitudes predicted depressed mood immediately following a negative event. Further, negative schemas have been found to emerge during stressful situations (Scher et al., 2005; Segal & Ingram, 1994). There is also support for cognitive vulnerability (Ingram et al., 1998; Scher et al., 2005; Segal & Ingram, 1994) and cognitive reactivity (Segal et al., 2006) which

describes fluctuations in negative attitudes that are directly in response to daily stressors or events (Butler et al., 1994), that create a diathesis for the onset, relapse or recurrence of depression. In a prospective study over two and a half years in undergraduate students, Alloy et al. (2006) found that individuals with higher cognitive vulnerability were 3.5-6.8 at greater odds of experiencing depression than those with lower cognitive vulnerability. Furthermore, there is evidence for a range of biological correlates of the cognitive model, including genetic vulnerability (Caspi et al., 2003; Uher & McGuffin, 2010) and neurobiological factors (Abler et al., 2007; Munafò et al., 2008; Siegle et al., 2007) involved in the development and maintenance of depression (Beck, 2008).

Empirical evidence also supports the negative cognitive triad of depression. Individuals with depression have been found to have negative schemas directed at the self (Dobson & Shaw, 1987; Kendall et al., 1989) and these negative self-schemas are stable over time (Dobson & Shaw, 1987). Further, Strunk et al. (2006) found individuals with high, but not low or medium depression symptoms, were significantly more likely to incorrectly predict a greater number of negative future life events, suggesting they had a greater pessimistic bias. Similarly, Strunk and Adler (2009) used three prediction tasks to examine the optimism or pessimism about predictions of future negative life events, views of the self and an individual's interpersonal world. Out of the 85 undergraduate students in the study 17 met SCID criteria for MDD. Consistent with the cognitive model of depression individuals with greater depressive symptoms reported more pessimistic bias about the future, the self and the world around them. Further, there is evidence that these cognitions are more negative in depressed individuals relative to non-depressed individuals (e.g. Blackburn et al., 1986; Blatt et al., 1982).

There is also evidence that suggests depression is associated with biases in attention and memory. With regard to biased attention, individuals with depression compared to non-depressed controls are biased towards sad stimuli (Gotlib et al., 2004; Kellough et al., 2008) and neurobiological evidence suggests there may be deficits in depressed individuals' ability to inhibit attention to negative stimuli (Disner et al., 2011). In a recent meta-analysis of eye-tracking studies, Suslow et al. (2020) found that depressed patients, compared to non-depressed individuals, were more likely to attend to sad faces and dysphoric pictures, suggesting there may be a bias towards negative stimuli in depression. Meta-analyses of studies examining memory and depression also show that individuals with depression are also more likely to retrieve depressed mood congruent information than depression incongruent information (Gaddy & Ingram, 2014; Matt et al., 1992).

1.3.2.2 CBT Treatment and Protocol.

Based on the principles of the cognitive theory of depression, CBT is a time-limited, structured psychotherapy for depression which focuses on the present. The aim of CBT is to reduce symptoms of depression by modifying the function, content and structure of negative thinking styles which are theorised to be key maintaining processes in depression (Beck et al., 1979). The treatment uses a number of behavioural and cognitive techniques to identify, understand and challenge negative thinking styles. As previously discussed (section 1.2) there is variation in CBT protocols in RCT settings and routine clinical practice (IAPT services in England). Studies two (chapter three) and three (chapter four) in this thesis utilise data from a trial of CBT and BA for depression (COBRA trial; Richards et al., 2016). The COBRA trial required session-by-session manualisation of both therapies for the purpose of

training and comparability (see Appendix 2). The CBT protocol from the trial will be discussed here.

The beginning of treatment focuses on the assessment of the client's problems, formulation of depression and goal setting. Formulation helps to identify the causes, precipitating and maintaining factors of an individual's depression symptoms (Eells, 1997). Formulations can be in both longitudinal (Beck et al., 1979) and cross sectional formats (Greenberger, 1995). Longitudinal formulations encompass early experiences which contribute to the development of core beliefs and later lead to dysfunctional assumptions which can be activated following a life stressor or event. The formulation also identifies NATs that lead to depression symptoms. Conversely, cross sectional formulations highlight how thoughts, feelings, behaviours and somatic symptoms interact. Perhaps the most widely used cross sectional formulation model is the 'Hot Cross Bun' model (Greenberger, 1995). Furthermore, goals are set during early therapy and are formulated using the 'SMART' acronym- Specific, Measurable, Achievable, Realistic and Time-limited. Goals help to provide a sense of what is being worked towards in treatment.

The second part of CBT treatment is activity scheduling to reduce avoidance and increase valued and pleasurable activities. The first stage of activity scheduling is to monitor a client's current activity using a diary where the client records their activity on an hour by hour basis. The client rates their mood during each activity, sense of achievement (sense of mastery), and enjoyment (pleasure) on a scale. Activity schedules are then reviewed to identify activities that promote and deplete mood which aids the client's understanding of how mood and activity are related. Activity scheduling is then used to plan rewarding activities into the client's week to help reduce low mood.

Next CBT focuses on developing skills to identify and challenge NATs. Skills to identify and consider alternative, more balanced perspectives can be facilitated by the therapist using ‘guided discovery’. Guided discovery involves the therapist facilitating the client to expand their awareness and thinking to discover alternative, balanced, and healthy perspectives and solutions to their problems by themselves (Kazantzis, Beck, et al., 2018). One aspect of guided discovery is using Socratic dialogue. This dialogue involves the therapist asking a series of open ended questions to help guide the client to discover alternative and adaptive solutions for themselves, e.g. ‘what was going through your mind just before you started to feel this way’, or ‘what is an alternative way of looking at this situation?’. Further, as NATS are habitual and clients may be unaware of them, often changes in emotion can help to signify a NAT. Thought records are a tool that can be used to help identify and challenge the thoughts (Greenberger, 1995). A commonly used thought record has seven columns where clients can (1) identify the situation or trigger, (2) rate emotions and body sensations, (3) identify the unhelpful thought or image, (4) provide evidence that supports the unhelpful thought, (5) provide evidence against the thought, (6) record an alternative, more balanced thought, and (6) rate how they are feeling having considered the alternative. Once NATs have been identified by the client, therapists may also help identify thinking errors and reasoning biases, such as mind reading, overgeneralisation, all or nothing thinking, or fortune telling (Moore & Garland, 2004). Methods such as reviewing evidence for and against a thought, behavioural experiments and role play can be used to help the client question NATs.

Once the client has developed the skills to effectively challenge surface-level NATs, the therapy moves to focus on working with schemas. The therapist will initially work to identify dysfunctional assumptions and/or core beliefs the client holds

that typically occur across time and in different situations and then use techniques to help challenge and modify the beliefs. One strategy used is ‘downward arrow technique’ which is a form of Socratic questioning to help uncover dysfunctional assumptions and core beliefs. Cross sectional formulations of these beliefs may be conducted to understand their impact. Other strategies involve examining these beliefs in everyday situations and looking at evidence for and against the belief to begin to challenge maladaptive beliefs. Following this the therapist will work with the client to generate an alternative, balanced beliefs and behavioural experiments are used to empirically test out the adaptive beliefs.

The final phase of CBT treatment is to focus on relapse prevention, consolidation of skills learnt throughout therapy and therapy ending. Relapse plans are devised to use all the skills learnt throughout therapy to support the client in detecting early warning signs of relapse and helping with setbacks. Within the last sessions of CBT there is focus on the client becoming self-sufficient and encouraging the client to become their own therapist and use the CBT tools learnt within therapy if they encounter setbacks. Four optional booster sessions were offered to support the client to maintain therapeutic gains and practice the skills learnt within therapy to help them stay well.

1.3.2.3 Mechanisms of Change in Cognitive Therapy.

As the cognitive theory of depression suggests information biases and resultant NATs maintain depression, the aim of CBT is to correct information processing biases by modify dysfunctional behaviours and restructure thinking patterns to alleviate depression symptoms (Beck et al., 1979). Within CBT, cognitive and behavioural processes that maintain depression are identified and strategies are

taught to modify these beliefs. The central assumption in CBT is that cognitive change is the mechanism that will alleviate depression and eventually lead to recovery (Garratt et al., 2007). This is referred to as the 'cognitive mediation hypothesis' (Beck et al. 1979). Although behavioural strategies are used within CBT an important assumption is that behavioural components of CBT (either directly or indirectly) facilitate cognitive change (Garratt et al., 2007).Throughout CBT, behavioural experiments are collaboratively, between the client and therapist, utilised to facilitate cognitive restructuring by testing out alternative, healthy thoughts and beliefs within an individuals' environment (Beck et al. 1979). Therefore, rather than behavioural strategies being hypothesised to be active mechanism of change within CBT they are hypothesised to facilitate cognitive change.

There are a number of different models that hypothesise how cognitive change may lead to depression recovery in CBT (Barber & DeRubeis, 1989). One suggests that core cognitions (such as schemas or dysfunctional attitudes) are modified during cognitive therapy which then leads to depression symptom reduction (*the accommodation model*) (Hollon et al., 1990). Another hypothesis suggests that depressionogenic schemas remain largely unchanged but over the course of CBT these schemas are deactivated which is then associated with a reduction in depression symptoms (*the activation-deactivation model*) (Hollon et al., 1990; Ingram & Hollon, 1986). Alternatively the underlying structure of depressionogenic schemas remain intact but throughout CBT compensatory skills are developed to help deal with low mood and life stressors where depressionogenic schema would normally be activated (*the compensatory skills model*) (DeRubeis et al., 1990; Hollon et al., 1990). There is little empirical support for either of these three models of cognitive change (Garratt et al., 2007; Lorenzo-Luaces et al., 2015). This is in part due to not

being able to directly observe schemas and schema change, and it is also possible that schema change is not the only route to depression symptom reduction and that other cognitive variables which span across domains may also lead to symptom reduction (Garratt et al., 2007). Cognitive change can be distinguished into cognitive change processes (e.g. change in dysfunctional attitudes or cognitive distortions), and cognitive change procedures which are cognitive techniques used by the therapist to foster cognitive change (Lorenzo-Luaces et al., 2015). Evidence for cognitive mediation being the mechanism of change in CBT would need to show that a) cognitive change processes in clients drives depression changes in CBT, b) cognitive change procedures are the most efficient in bringing about cognitive change in CBT, and c) that cognitive changes in CBT lead to larger changes in depression symptoms than cognitive changes from non-CBT procedures (cognitive specificity) (Lorenzo-Luaces et al., 2015; Whisman, 1993). The evidence for each of these will be discussed.

Regarding the first point, there is a substantial amount of research that explores the association between cognitive change processes and depression symptom change in CBT. There is evidence for concurrent associations between cognitive change and depression symptom change (Christopher et al., 2009; Garratt et al., 2007; Lorenzo-Luaces et al., 2015; Oei & Free, 1995). However studies assessing concurrent relationships ignore the possibility of reverse causation (i.e. that depression symptom change leads to cognitive change). Research has also examined the prospective relationship of cognitive change predicting subsequent symptom change (Lorenzo-Luaces et al., 2015). For example, changes in dysfunctional attitudes have been found to be associated with reduced depression symptoms in CBT (Cristea et al., 2015; DeRubeis et al., 1990; Furlong & Oei, 2002;

Quilty et al., 2008). In other studies examining prospective relationships, cognitive change also has been found to predict subsequent depression symptom reductions (Fitzpatrick et al., 2020; Schmidt et al., 2019). In particular, Schmidt et al. (2019) found sustained cognitive change mediated the relationship between immediate cognitive change and session-to-session depression symptom change. However, it is also the case that studies find evidence of concurrent but not longitudinal associations between cognitive change and depression symptom reduction (Burns & Spangler, 2001; Lemmens et al., 2017; Quigley et al., 2019; Vittengl et al., 2014; Warmerdam et al., 2010). One possibility for the discrepancies between studies could be the timing of measurement of cognitive change and depression symptom change, as it is unclear how rapidly cognitive change may have an influence on depression symptoms (Quigley et al., 2019). This highlights the importance of considering the timing of cognitive change measurements. Another avenue of research has examined the relationship between cognitive change in CBT and subsequent relapse to assess temporality of the cognitive change and depression relationship. Here studies have found cognitive change is associated with reduced likelihood of experiencing depression relapse (Beevers & Miller, 2005; Segal et al., 2006; Teasdale et al., 2001) which is further evidence for cognitive mediation in CBT. Although there is a proportion of evidence that supports the hypothesis that cognitive change is associated with depression reductions in CBT, it is also important to note that these studies do not rule out the influence of a third, unmeasured variable such as another cognitive process, non-cognitive process or patient characteristic for example (Driessen & Hollon, 2010).

The association between therapist cognitive change procedures and depression symptom changes would not allow us to elucidate the mechanism of

change in CBT. It could be possible that cognitive change (X) could lead to depression symptom change (Y) through cognitive change processes (M). Alternatively, it may be that the mediator in the relationship between cognitive change methods (X) and depression reduction (Y) are behavioural change processes (M). Instead, examining how effective cognitive change procedures are at producing cognitive change may help to understand how cognitive change is achieved. Some research has found clear associations between therapist cognitive methods and client cognitive change (Lorenzo-Luaces et al., 2015). For instance, therapist cognitive change procedures in early CBT treatment for depression were found to facilitate client cognitive change (Schmidt et al., 2019). In another study, Stone and Strunk (2020) selected sessions in which clients had high or low levels of cognitive change in CBT, and examined whether observer ratings of therapist strategies (cognitive methods, Socratic questioning, behavioural methods, and therapeutic alliance) could differentiate between sessions with differing levels of cognitive change. In the combined model with all therapist strategies only cognitive methods predicted greater cognitive change, suggesting that cognitive change procedures help to facilitate cognitive change in CBT (Stone & Strunk, 2020). Further, therapists' use of Socratic questioning in early sessions of CBT have been found to predict session-to-session depression symptom improvement (Braun et al., 2015). However, it is also the case that research has found that treatments which proscribe cognitive procedures also have an effect on cognitive changes (Lorenzo-Luaces et al., 2015).

Therefore another line of research has investigated whether cognitive changes in CBT lead to larger symptom changes in depression than do cognitive changes in non-CBT treatments. This research compares cognitive change in CBT

to other treatment modalities and has yielded mixed results. One avenue of exploration has been to compare associations between cognitive change and depression change in CBT to that in pharmacological treatments, where the mechanism of change is not expected to be cognitive (Garratt et al., 2007). Some research finds support for this hypothesis (Dozois et al., 2009; Quilty et al., 2008) . For example in a trial of individuals receiving either CBT or antidepressant medication (ADM) for depression found individuals in both treatment groups showed changes in cognitions (automatic thought, dysfunctional attitudes and hopelessness) but crucially cognitive change from pre-treatment to mid-treatment in the three cognitive domains predicted change in depression symptoms in CBT from mid-treatment to post-treatment, but not in the ADM group (DeRubeis et al., 1990). The authors suggest that cognitive change in the ADM group may have resulted from reductions in symptom change, whereas in CBT cognitive change may drive depression reductions. Conversely other studies find no differences in the effects cognitive change has on depression symptoms in CBT compared pharmacological treatments (Quilty et al., 2014). In research comparing CBT to other psychological treatments it is often found that cognitive change has an influence upon depression symptom change in both CBT treatment and other psychotherapies (Oei & Free, 1995), including mindfulness based intervention (Hofheinz et al., 2020), interpersonal therapy (Quilty et al., 2008) and problem solving therapy (Warmerdam et al., 2010). In a meta-analysis of studies, Cristea et al. (2015) found no significant differences between CBT and other psychotherapies or pharmacological therapies on the impact of change in dysfunctional thoughts. The mixed evidence suggests that, contrary to cognitive specificity hypothesis (Hollon et al., 1987), other psychotherapies and pharmacological treatment may also work by changing cognitions even if they are

not directly targeted in treatment. Although further research is needed, the current evidence suggests that cognitive change and the association with depression symptom reductions may not be specific to CBT.

Due to mixed research findings regarding cognitive mediation in CBT, the necessity of modification of cognitive processes to bring about change in depressive symptoms, and the necessity of cognitive change methods to modify cognitive processes has been questioned (Longmore & Worrell, 2007). In support of this, research examining depression symptom changes in therapy shows that a large majority of depression symptom change occurs early in therapy (Lambert, 2013) before cognitive change strategies are implemented (Ilardi & Craighead, 1999) . This suggests that other, non-cognitive processes or methods may facilitate depression symptom change. Although it has been noted that cognitive change methods can be used as early as session two in therapy (DeRubeis & Feeley, 1990), it is also possible that rather than cognitive processes, nonspecific and/or non-cognitive process may be mechanisms of change in CBT, such as therapeutic alliance and behavioural processes. Discussion of the role of common factors being mechanisms of change in CBT with a focus on therapeutic alliance is discussed in section 1.3.3. With regards to behavioural processes, the current literature has not extensively examined the role of behavioural change processes in CBT. Rather there is the focus on the necessity and the clinical effectiveness of behavioural change strategies (e.g. behavioural activation, activity scheduling) in CBT (Kazantzis, Luong, et al., 2018). Although the evidence is limited there are several studies that highlight the possibility that behavioural processes may also have an influence in CBT. For example, Jacobson et al. (1996) observed that change in the frequency of pleasant events in the early stage of CBT was associated with improvement in depression

symptoms. Another study observed steep increases in behavioural activation measured on the BADS in CBT in early treatment (Bolinski et al., 2018), and in a hospital setting Christopher et al. (2009) found that change in behavioural activation in CBT significantly predicted post-treatment depression scores. Further, Jacob et al. (2011) observed that increases in both cognitive and behavioural change in CBT were associated with decreases in depression symptoms. Similarly, in a recent study Lemmens et al. (2021) found both cognitive and behavioural processes in CBT were related to rapid depression symptom improvements early in therapy. This suggests that behavioural change processes may be involved in symptom change in CBT, but further research is needed.

Altogether, the research examining mechanisms of change in CBT has focussed on cognitive variables being responsible for change in depression symptoms. The support for the cognitive mediation hypothesis is mixed. There is evidence which supports the hypothesis that there is an association between cognitive change and depression symptom change in CBT, but some of the evidence fails to examine the temporality of the association. A substantial amount of evidence shows, contrary to the specificity hypothesis, that cognitive change can occur in therapies that proscribe cognitive change procedures and therefore cognitive change is not specific to CBT. It is also the case that non-specific factors and behavioural processes may contribute to depression reductions, questioning the sufficiency and even the necessity of cognitive processes of change, but less research has examined this in the context of CBT. Overall further research is needed to elucidate the mechanisms of change in CBT.

1.3.3 Common Factors across CBT and BA

In contrast to the view that psychotherapies result in beneficial effects through specific theorised treatment factors, there is also the suggestion that treatments work through common (non-specific) factors (Rosenzweig, 1936). Common factors are those that every psychotherapeutic intervention has in common such as therapeutic alliance, a rationale that can help clients understand their problems, and expectations or hope (Cuijpers, Reijnders, et al., 2019). The most contemporary common factors model is the 'contextual model' (Wampold, 2015; Wampold & Imel, 2015) which suggests that therapy works through three pathways. The first is creation of a bond between a therapist and client where there is care and empathy which creates a safe, healing setting. Secondly, the therapeutic rationale creates expectations and fosters hope that the patient can change to cope with their problems. The last pathway in the model suggests that specific factors, such as modifying negative thoughts in CBT or increasing contact with valued activities in BA, helps to encourage healthy actions that help to reduce their problems (Cuijpers, Reijnders, et al., 2019). Support for the notion that therapies work through common factors comes from studies that find treatments with different theoretical foundations lead to similar effects (e.g. Barth et al., 2013; Braun et al., 2013; Cuijpers, Karyotaki, et al., 2019). The equivalency of treatment outcomes is often referred to as the 'Dodo Bird Verdict', an analogy to Lewis Carroll's Alice's Adventures in Wonderland in which the Dodo announces that "Everybody has won, and all must have prizes" (Carroll [1865] 1998, pp.34).This is illustrated in the depression literature in meta-analytic studies which find different psychotherapies have comparable effects on depression symptoms (e.g. Barth et al., 2013; Cuijpers et al., 2020; Cuijpers et al., 2018; Linde et al., 2015), including CBT and BA for depression (Braun et al., 2013;

Ekers et al., 2011; Mazzucchelli et al., 2009). In the largest non-inferiority trial comparing the clinical effectiveness of CBT and BA for adult depression, both treatments were found to be as clinically effective as each other (Richards et al., 2016).

Although there are a range of common factors reviewed in the literature, this brief discussion of common factors focuses particularly on therapeutic alliance, which is one of the most widely studied common factors (Cuijpers, Reijnders, et al., 2019). However, it is important to acknowledge there are other aspects of therapy relationships which make contributions to therapy outcomes. The American Psychological Association Task Force on Evidence-Based Relationships and Responsiveness highlighted goal consensus and collaboration (Tryon & Winograd, 2011), empathy (Elliott et al., 2018), positive regard and affirmation(Farber et al., 2019), and collecting and delivering client feedback (Lambert et al., 2019) as elements of therapeutic relationships which have been shown in meta-analyses to be associated with psychotherapy outcomes (Norcross & Lambert, 2018). With regard to therapeutic alliance, a substantial amount of research suggest therapeutic alliance contributes to treatment efficacy across a number of different psychotherapies (Baier et al., 2020; Crits-Christoph et al., 2011; Falkenström et al., 2013; Martin et al., 2000). In a meta-analysis of 200 studies across psychotherapies Horvath et al. (2011) found that stronger alliance was associated with better treatment outcomes. However, again, the correlational nature of these examinations cannot allow for causal inferences to be made and to assess the temporal relationship between therapeutic alliance and symptom changes.

In the context of CBT treatment there is strong support for the correlational association between therapeutic alliance and beneficial effects on depression

symptoms (e.g. Baier et al., 2020; Cameron et al., 2018; Castonguay et al., 1996; Horvath & Luborsky, 1993; Kazantzis, Luong, et al., 2018; Krupnick et al., 1996). However, when assessing the temporality of the relationship there is evidence for therapeutic alliance being a consequence of symptom change *and* therapeutic alliance preceding depression symptom change. For example, DeRubeis and Feeley (1990) found therapeutic alliance was correlated with prior symptom change, suggesting it is a consequence of change in depression symptoms and this finding was replicated by Strunk et al. (2010) and Strunk et al. (2012) in larger samples. Conversely, Falkenström et al. (2016) found therapeutic alliance predicted improved depression symptom in the next therapy session, although this was in a relatively small sample size. Examining therapeutic alliance another way, Zilcha-Mano (2017) discuss the importance of distinguishing between trait (i.e. an individuals' general ability to form relationships) and state (i.e. cultivating alliance during treatment) elements of alliance in therapy. When distinguishing these factors, studies that consider temporality and between-within patient variability show that alliance may precede change in symptoms (Zilcha-Mano et al., 2018). However, there is also evidence for a reciprocal relationship between alliance and symptom change. In a meta-analysis of cross-lagged associations between alliance in early therapy and symptom change across therapies and disorders, Flückiger et al. (2020) found evidence for both higher alliance predicting lower symptom and reductions in symptom predicting higher alliance. Alternatively, some fail to find associations between therapeutic alliance and depression symptom change (Kaufman et al., 2005). In a recent study, examining therapeutic alliance and depression symptoms in 98 adult outpatients receiving CBT, Don et al. (2021) found that therapeutic alliance was not associated with prior or subsequent depression symptom change.

In contrast to the evidence for therapeutic alliance as a mechanism of change in CBT, less research has explored the role of therapeutic alliance in BA. There is an emphasis on establishing a good therapeutic relationship in the BA treatment manual (Martell et al., 2001) and in BA treatment quality assessments (e.g. Quality of Behavioural Activation Scale, QBAS; Martell, Dimidjian & Herman-Dunn, 2022). The QBAS specifically enquires about stylistic characteristics that encompass therapeutic alliance, such as taking a non-judgemental stance, providing validation and encouragement, and expressing warmth. Despite this, little research has looked at therapeutic alliance specifically in BA.

Overall the literature shows that common factors across different psychotherapeutic modalities do exist, like therapeutic alliance. However the evidence for the temporal relationship between therapeutic alliance and depression symptom change in CBT is mixed, and this area is under researched in BA.

1.3.4 Summary of Section

Understanding the mechanisms of change in both CBT and BA can allow us to refine and improve treatments for depression. Despite the evidence to date, it is still unclear which processes of change are important to reduce depression symptoms in CBT and BA, which are two related treatments with distinct theoretical backgrounds. A range of different methodologies to identify important treatment components have been used and these will be reviewed in the next section.

1.4 Methods Used to Examine Processes of Change in Psychotherapy for MDD

Within the psychotherapy literature a range of quantitative and qualitative methods have been used to examine processes of change. This thesis will focus on quantitative methods. This section will discuss statistical methods used to examine

processes of change and study design considerations, before outlining ways to measure processes of change in psychotherapy and how to identify *when* in treatment to examine change.

1.4.1 Statistical Methods Commonly used to Examine Processes of Change

Simple mediation is commonly used to identify whether an intervening process variable statistically accounts for the relationship between an independent variable (e.g. treatment modality) and a dependent variable (e.g. depression treatment outcomes) (Kazdin, 2007). Mediators are required to be specific and to some degree have a level of consistency across studies and samples. To be able to infer some causation there needs to be an established timeline of the mediator preceding the treatment outcomes, and there needs to be some plausibility and coherence of the mechanisms involved in influencing change in treatment (Kazdin, 2007). However, simple mediation is indirect and correlational and there is always the possibility that another, unknown variable is responsible for change in treatment outcomes. It is also the case that mediation examines change in process at the group level, ignoring variability between individuals. It is likely that therapy operates through multiple processes and simple mediation cannot account for this. Further, mediational tests cannot give an insight into the causality of the relationship. This is also true for other statistical methods such as those that use lagged models because some other confounding factor or unmeasured variable may be influencing the relationship.

It is also important to distinguish between analysis of mediation and that of moderation. Moderators are intervening variables that influence the relationship between an intervening variable and treatment outcome (Kazdin, 2007; Kraemer et al., 2002). They are variables that are not influenced by treatment, such as baseline

clinical characteristics or demographic variables. Although they are not directly used to examine mechanisms of change, examining moderators can be useful for identifying for whom and under what conditions a treatment is most efficacious.

1.4.2 Study Design Considerations

A number of study designs have been used to examine active ingredients of psychotherapeutic interventions and client processes in psychotherapy research. It is common within comparative RCT designs that hypothesised client process variables are measured to examine whether they are mediators of change, and they are often measured as secondary outcomes. However, comparative RCT studies are usually powered only to detect the difference in effectiveness of one treatment compared to another. Further, all treatment package components are delivered and confounded together so it can be difficult to identify specific treatment components or active ingredients that result in change within psychotherapy (Watkins & Newbold, 2020).

Component studies, either dismantling or additive designs (Bell et al., 2013), are another way to identify the active ingredients of therapy that are important to elicit symptom change. Additive designs add additional components which are believed to improve treatment outcomes to a treatment package, whereas dismantling studies disassemble psychotherapies with multiple components (such as CBT) and compare the components (e.g. BA) with the full therapy package to elucidate whether that component is responsible for change within the treatment (Borkovec & Castonguay, 1998). One of the most prominent dismantling studies in the psychotherapy literature was conducted by Jacobson et al. (1996) examining the behavioural component of cognitive therapy compared to cognitive therapy alone and finding the behavioural component was as equally as effective as the cognitive components in CBT. A particular strength of component designs relates to the

conclusions that can be drawn; if a therapy without a particular component is less effective than with the component this suggests that the component is accountable for (at least some of) the effects of the intervention. This is in comparison to mediational designs which are correlational and it is always possible that some other, unmeasured third variable which may be responsible for change in treatment outcomes (Kazdin, 2007). However, there are several drawbacks to dismantling designs. If a study finds there are no differences between treatment components this does not allow us to address whether specific or common treatment factors are mechanisms of change because no group receives a treatment with solely common factors (Bell et al., 2013). It has also been found across the literature that a large amount of depression symptom change occurs early in treatment (Lambert, 2013), and Rehm (2009) highlighted that whichever component is delivered first may appear to be the most effective. Furthermore, another drawback of dismantling designs is that there is the assumption that there are no interactions between study components (Watkins & Newbold, 2020).

Another approach to identify active intervention components in psychotherapy is to use factorial designs (Collins et al., 2014; Collins et al., 2009; Watkins & Newbold, 2020). These experimental study designs allow for the comparison between components of interest and allow to examine both the main effects and interactions between study components. Using Jacobson's (1999) seminal component analysis as an example, Watkins and Newbold (2020) illustrate how a factorial design may help understand which components in CBT and BA are facilitators of change. Instead of being assigned to either component as in the Jacobson study (full CBT package (including BA, cognitive restructuring (CR) and work on core schema (CS)), BA plus cognitive restructuring, or just BA) participants

would be randomly assigned to a combination of the presence (+) or absence (-) of the three factors, resulting in a possible one of eight combinations; all three elements ((BA+: CR+:CS+), two of the three elements (BA+: CR+:CS-; BA+: CR-:CS+; BA-: CR+:CS+), one of the tree elements (BA+: CR-:CS-; BA-: CR+:CS-; BA-: CR-:CS+) or none (BA-:CR-:CS-) (Watkins & Newbold, 2020). Although factorial design approaches have been used in other disciplines only a few studies have used them within psychotherapy research (e.g. Bruijniks, Lemmens, et al., 2020; Watkins & Newbold, 2020). With a large number of components, factorial designs can not only be more costly and require more resources (e.g. therapist trained in delivering different components) but also require a large sample size (Chakraborty et al., 2009; Collins et al., 2014). In instances where there are a large number of combinations, fractional factorial designs may instead be used. Within fractional factorial designs there is a systematic reduction in the number of treatment conditions used, which may help to reduce the sample size needed and make the study more manageable (Watkins & Newbold, 2020).

Although all of these designs have merits, for practical reasons the current thesis (studies two and three) uses existing RCT data to examine processes of change. The methods used to measure processes of change are outlined in the next section.

1.4.3 Methods Used to Measure Processes of Change

There are a variety of methods used to examine process of change within therapy. Perhaps the simplest method is the use self-report measures. A wide variety of self-report measures have been developed and validated to assess processes of change within psychotherapy (e.g. dysfunctional attitudes scale; Weissman & Beck, 1978) and they can be quick and easy to administrate. However,

there are also a number of limitations with using self-report methods. It is likely that it is necessary to measure more than one type of process (e.g. different types of cognitive change) in order to understand mechanisms of change in psychotherapy, and measuring multiple processes with self-report measures can be burdensome and time consuming. Similarly it is also likely that processes are changing and evolving throughout the therapy session (Zilcha-Mano, 2019) and this is difficult to examine with self-report measures. Another consideration is deciding when to administer the process measure. Instructing a participant to answer the measure at the beginning of the therapy session and recall over the past week may help to capture between-session change, but this may be prone to recall bias. To try and capture the process close the point at which the process is occurring (for example during or in between therapy sessions) may not be feasible.

Another method is to use ecological momentary assessment (EMA) approaches to measure process. Contemporary EMA approaches use mobile technology (e.g. watches or smartphone applications) to collect data within an individual's natural environment at repeated time points over the day (Colombo et al., 2019). There are several advantages to EMA methods over self-report measures. Unlike self-report measures which reply on retrospective reports of mood and processes (e.g. cognitions and behaviours) EMA can capture phenomena close to the point at which they are occurring. This can help reduce recall bias which may be particularly applicable to individuals with MDD where recall of events may be confounded by mood (Ben-Zeev et al., 2009; Ellison et al., 2020; Köhler et al., 2015). Furthermore EMA approaches may be able to give an insight into how learning is consolidated and used between therapy sessions in real life. In a recent example van Genugten et al. (2021) used EMA to examine the associations between

engagement in pleasant activities and related mood in BA. In support of BA theory (Lewinsohn, 1974), at the between-patient level greater engagement in pleasant activities was associated with greater pleasure which in turn was associated with better reported mood. As illustrated in this study EMA methods may also allow for the examination of temporal relationships between processes of change and mood which can further help to elucidate how treatments work to reduce depression. However, similarly to self-report measures, EMA approaches can be burdensome to patients and this is highlighted by reports of poor adherence to completing measures and high instances of missing data (van Genugten et al., 2021).

An alternative to self-report measures and EMA approaches is the use of observational coding systems to examine within therapy processes of change. This approach can be used from existing data, alleviates the burden on patients but at the same time allows for the detection of multiple psychotherapeutic processes. This method uses trained coders to identify, evaluate and rate processes of interest from live, audio or video recorded content (Schoenwald et al., 2011). Within the psychotherapy literature there are a number of different observational coding systems used to assess different mechanisms of change within therapy. For example, Tang and DeRubeis (1999) designed the Patient Cognitive Change Scale (PCCS) to measure seven categories of cognitive change, such as bring a belief into awareness, identifying an error in a cognitive process or belief, or arriving at a new schema. Coders listen to therapy sessions and rate the significance of the cognitive change (1 = a possible/potential cognitive change; 4 = a cognitive change with extraordinary personal significance). Subsequently, the PCCS has been used to examine cognitive change in therapy, including CBT in different samples (Lemmens et al., 2021; Tang et al., 2005). Within the current thesis (in studies two and three)

the ‘Change and Growth Experiences Scale’ (CHANGE; Hayes, Feldman, & Goldfried, 2007) coding system was used to assess processes of change. The CHANGE is a transtheoretical, observational coding system which assesses for a range of therapist (e.g. therapeutic relationship, the amount of cognitive and behavioural corrective information supplied by the therapist) and client (e.g. positive behaviour, avoidance, overgeneralisation, accommodation, cognitive-emotional processing) processes. Raters consider both within therapy session content as well as experiences from the week prior to the session within the coding. A particular strength of this approach is that processes can co-occur and are not mutually exclusive and therefore interactions between process variables can be examined. For example a client can be both hopeful but still be avoiding activities and overgeneralising. The CHANGE coding system has been used across disorders and therapies to examine process of change. For example in a depression treatment-resistant population receiving CBT, Abel et al. (2016) found changes in hope and cognitive-emotional processing during times of rapid depression symptom change within treatment. In a population of children and adolescents who received Trauma-Focused Cognitive Behavioural Therapy, Ready et al. (2015) found greater overgeneralisation of maladaptive cognitions predicted higher internalising and externalising scores at post-treatment.

It is of note that observational coding approaches require multiple coders to spend a lot of time training to ensure all coders have good inter-rater reliability, the degree to which two or more rates agree in their independent coding (Hallgren, 2012). Additionally, the coding can be labour intensive and only what is discussed within the session can be rated. Unlike other methods (e.g. qualitative methods) because coding is conducted retrospectively, client opinions and clarification on

change is not possible. However, a significant advantage of using observation coding systems, like the CHANGE, is that a wide range processes (both specific and common factors) can be coded. This may be particularly helpful to understand whether specific (e.g. cognitive or behavioural) or non-specific (common factors) processes of change are important for eliciting depression symptom change in therapies which use different therapeutic frameworks, like CBT and BA.

1.4.4 Identifying When in Therapy to Measure Process

Another important consideration is *when* in treatment to examine mechanisms of change. Traditionally, research examining mechanisms of change in psychotherapy has assessed processes of change within a single treatment session and then examined whether this mediates change from baseline to post-treatment (e.g. Feeley et al., 1999). There are a number of limitations to using this approach. First, this approach ignores individual variability in processes over treatment. Second, in order to understand how treatment works, it is important to know when in treatment the change is occurring and this may occur over a number of sessions and arbitrarily choosing a session to measure process cannot help to understand when is most important for change to occur. Additionally, within a single session it can be difficult to demonstrate the temporal sequence between a process and depression symptom change (Borckardt et al., 2008; Kazdin, 2007). Repeatedly measuring process across multiple sessions can not only help to understand these questions, but also it means that processes are treated as evolving and changing, rather than fixed entities (Zilcha-Mano, 2019).

One way to focus on when in treatment to examine mechanisms of change is to identify times of substantial depression symptom change. A common misconception used to be that change in depression symptoms occurs in a gradual

and linear fashion during therapy (Hayes, Laurenceau, et al., 2007). This may have resulted from assessing group averages and/or only measuring depressive symptoms at baseline and end of treatment. While for some individuals this may be the case, the recommendation of measuring depression symptoms in each therapy session (Harding et al., 2011; Pfeifer & Strunk, 2015) in both RCT samples (e.g. Richards et al., 2016) and some naturalistic therapy settings (e.g. IAPT; Clark et al., 2018) has allowed for the examination of the shape of depression symptom changes over therapy. Trajectory based approaches, where depression scores can be plotted over time, show differing patterns of depression change and have revealed subgroups of individuals who exhibit similar patterns of symptoms. For example, using individual course plots of depression symptoms over CBT treatment for MDD, Tang and DeRubeis (1999) identified large, rapid depression symptom reductions early in treatment which were found to be associated with positive treatment outcomes. Subsequently other trajectory research has also found that large shifts in depression symptoms are commonly observed over therapy. For instance in acute phase CT for individuals with recurrent MDD, Vittengl et al. (2013) examined linear, log-linear (large symptom improvements earlier followed by smaller improvements later in therapy), one-step (single, stable abrupt drop in symptoms) or undefined trajectories. They found those with defined trajectories (linear, log-linear, or one-step) had greater response to treatment and were in more stable remission than those with undefined trajectories. In another study examining depression trajectories in 4394 individuals who received high-intensity treatment (CBT, interpersonal therapy or counselling) in naturalistic clinic settings, Saunders et al. (2019) found slightly different patterns of depression change. Using latent class growth analyses they identified four distinct trajectories of depression symptoms across the

treatments; no response, slow initial response followed by a large response later in treatment, early initial response followed by a levelling out, and rapid early response followed by a levelling out. They did not examine how each trajectory was associated with treatment outcomes, but they found lower baseline levels of depression, better functioning at baseline, and lower phobic anxiety were associated with the responding trajectories, compared to the no response trajectory. This may suggest that patterns of depression change in naturalistic settings may differ from patterns in RCT samples (e.g. Vittengl et al., 2013). Nevertheless, this research encouragingly implies that different patterns of change can be beneficial, and this research has helped to identify key times when substantial, discontinuous depression symptom change occurs in therapy which may signal important times to focus on examining processes of change in treatment.

1.5 Using Discontinuous Patterns of Depression Symptoms to Assess Processes of Change in Treatment

Building on the idea that points of discontinuous change are important therapy markers, in the last two decades psychotherapy research has used these time points when discontinuous depression symptom change occurs to examine client processes of change and therapist strategies in therapy, as well as try to understand how depression treatments lead to beneficial treatment outcomes. This thesis focuses on two types of discontinuous change identified within the psychotherapy research as being associated with depression outcomes; sudden gains (Tang & DeRubeis, 1999) and depression spikes (Hayes, Feldman, Beevers, et al., 2007). In the following sections the evidence for these two patterns of change will be reviewed and the gaps within the literature will be highlighted.

1.5.1 Sudden Gains

Perhaps the most widely examined pattern of discontinuous depression change in psychotherapy research is a sudden gain. Sudden gains were first identified in CBT for depression to further understand the role that cognitive processes play in the alleviation of depression symptoms in response to a review by Ilardi and Craighead (1994). In the review of eight CBT studies Ilardi and Craighead (1994) observed that the majority (between 60-80%) of depression symptom reduction occurs within the first four weeks of therapy. They suggested this rapid early response of depression symptoms in treatment contradicted the cognitive mediation hypothesis of CBT as the symptom reductions occurred at a time in therapy when few cognitive strategies are employed (early in therapy, between sessions 1-4). Rather than depression change being the result of cognitive change they suggested that nonspecific factors such as the therapeutic relationship, being in

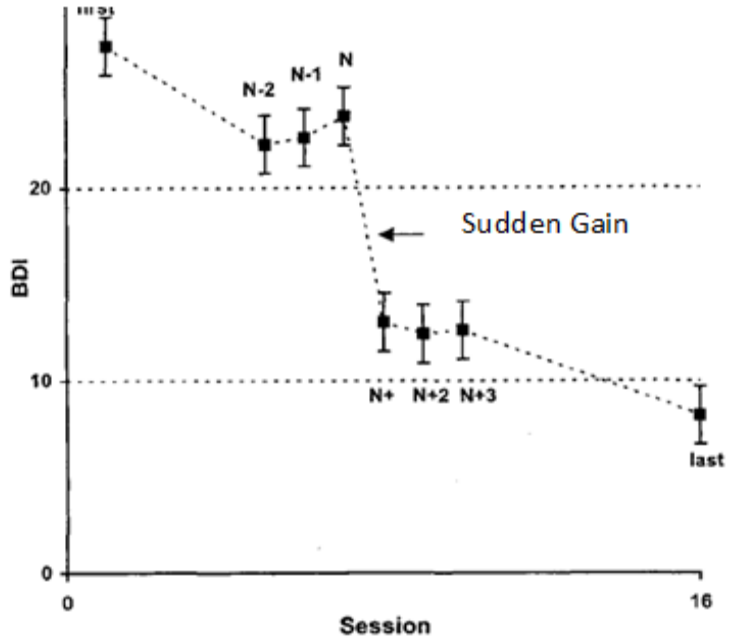
a healing setting, having a rationale for the patients' symptoms and the treatment procedure brought about depression symptom reductions (Ilardi & Craighead, 1994). In response to the suggestion that cognitive mediation cannot explain the rapid depression symptom improvement in CBT, Tang and DeRubeis (1999) argued that cognitive strategies can occur as early as session two in CBT (DeRubeis & Feeley, 1990; Feeley et al., 1999). Additionally, Tang and DeRubeis highlighted that the methodology used by Ilardi and Craighead was flawed because they made inferences about mechanisms by examining averaged symptom courses, rather than looking at individual patient symptom courses. Consequently, Tang and DeRubeis went on to examine individual time courses and identified sudden improvements in depressive symptoms, or 'sudden gains' and developed specific criteria to identify these symptom improvements.

Sudden gains are characterised as large, stable depression symptom improvements which occur between two consecutive therapy sessions. Using time course plots of 61 patients' depressive symptoms over a course of CBT for MDD, Tang and DeRubeis (1999) observed large depression symptom improvements in single between-session intervals, which accounted for a large percentage of patients' total depression symptom improvement (See Figure 1.3; Tang & DeRubeis, 1999). The authors defined sudden gains as occurring (a) if there is a single between-session reduction of at least seven Beck Depression Inventory (BDI) points or more, (b) if the magnitude of the gain was a least 25% of the pregain (session immediately prior to the drop in scores) BDI score, and (c) if the mean of the three BDI scores preceding the gain were significantly higher than the three BDI scores for the therapy sessions following the gain, using an *t*-test. Using this criteria, Tang and DeRubeis observed 29 sudden gains in 24 patients (out of a sample of 61 patients), with the

majority occurring in session 5 (out of 20 sessions) and four experiencing reversals of the sudden gain ('lost' 50% of the symptom improvement). Despite the two groups not differing in their depression levels at baseline, individuals who experienced a sudden gain had better post-treatment depression outcomes, than those who had not experienced a sudden gain, and the effect of the sudden gain was also reflected in the recovery rates of individuals. Individuals who did not experience a gain (41%) had significantly lower recovery rates than those who did have a sudden gain (79%). Crucially, the effects of the sudden gains were maintained at 6- and 18-month follow up demonstrating the longevity of the effects on depression symptoms.

Figure 1. 3

Illustration of a Sudden Gain from Tang and DeRubeis (1999)



Subsequent research has replicated and extended Tang and DeRubeis' (1999) sudden gains findings in CBT and other treatments for depression. Using the same criteria as Tang and DeRubeis (1999) sudden gains have generally been found to be associated with favourable treatment outcomes in CBT for individuals with MDD. For instance, in a replication study 29% of individuals who experienced a sudden gain early (median = session eight) in CBT were found to be associated with lower end of treatment depressions scores in 50 patients with MDD (Tang et al., 2005). Further, sudden gains in CBT for adolescents (39%; Gaynor et al., 2003), individuals with treatment resistant depression (54%; Abel et al., 2016), and in outpatient samples (32-42%; Lemmens et al., 2016; Wucherpfennig, Rubel, Hollon, et al., 2017) have been found to be associated with better depression treatment outcomes than individuals who did not have a sudden gain. There is some suggestion that sudden gains also have benefits on long term outcomes. In one study individuals who experienced sudden gains (40%) early in CBT (median = session 5) had lower relapse risks than those who did not experience a sudden gain (Tang et al., 2007). However, in another study although early sudden gains (41.9%) in CT were associated with beneficial treatment outcomes, over a two year follow up there was no advantage of experiencing a gain compared to no gain (Vittengl et al., 2005). Two exceptions to these studies that find sudden gains are advantageous are Stiles et al. (2003) and Busch et al. (2006). In the study by Stiles et al. (2003) sudden gains were experienced by 17% of individuals with diverse disorders (including depression) in a routine clinical setting. Individuals with early sudden gains did not differ on post-treatment depression scores (BDI), compared to individuals who did not experience sudden gains or those who experienced later sudden gains. This may be due to the setting of the study and that in naturalistic settings, where

therapy protocols may not be as tightly controlled as RCT settings. However, subsequent research examining sudden gains in naturalistic settings have found them to be beneficial (Greenfield et al., 2011; Wucherpfennig, Rubel, Hollon, et al., 2017). In Busch et al.'s (2006) study although the rates of sudden gains in individuals receiving CBT for depression were comparable to Tang and DeRubeis' (1999) study (38%) there were no differences in depression scores of individuals who did or did not experience a sudden gain at the end of treatment or recovery rates. The author's note that sudden gains occurred later in treatment (median session 10, range 3-18) compared to Tang and DeRubeis' (1999) sample, suggesting that early treatment sudden gains may be important for treatment outcome. Outside of CBT using the original sudden gains criteria, O'Mahen et al. (2017) examined sudden gains in women receiving online BA treatment for postpartum depression. A total of 51% (*n* = 18/32) experienced a sudden gain which were associated with better depression outcome. In a separate study of a sample of adults with recurrent MDD receiving group BA treatment, sudden gains were experienced by 34%, and although individuals with a sudden gain were significantly more likely to have clinical improvement than those without a gain, sudden gains did not predict lower dimensional posttreatment depression scores (O'Mahen et al., 2019). This may be because those with more severe, recurrent depression may require individualised treatments to produce the type of shifts associated with sudden gains.

Compared to the examination of sudden gains in CBT, substantially less research has examined them in the context of other therapies. Diverging from traditional CBT approaches, over the past 20 years so called 'third wave' therapy approaches, which focus more on the function of cognitions and emphasise

emotions, acceptance, values, goals and mindfulness (Hayes, 2004), have been used to treat depression. Therapies such as Dialectical Behaviour Therapy (DBT), Acceptance and Commitment therapy (ACT) and Mindfulness approaches are used in the treatment of depression, but sudden gains have been explored in these therapies to a lesser extent. In a recent meta-analysis of sudden gains across therapies and disorders, Shalom and Aderka (2020) highlight that there is limited research of sudden gains in DBT and ACT. Currently group-based mindfulness therapies are recommended only for relapse prevention in patients with a history of recurrent depression (NICE, 2009), and the limited research that has been conducted suggests that sudden gains are not common in this treatment (Ietsugu et al., 2015). Although this is beyond the focus of the current thesis, it is important to highlight this gap in our understanding of sudden gains in these third-wave therapies, and whether they occur at the same rate and have positive associations with treatment outcomes as has been seen in the sudden gain CBT literature. Research of this nature could also help to elucidate the role of common versus therapy-specific factors in the instigation of sudden gains.

1.5.1.1 Sudden Gains Criticisms

Although much of the research that examines sudden gains using Tang and DeRubeis' (1999) original criteria finds they are associated with beneficial short and long-term depression treatment outcomes, there has been debate around the criteria used to identify sudden gains and discussion about other aspects of sudden gains. Here the discussion of each sudden gains criteria, the research that uses altered sudden gains criteria, and discussion about stability and tautology of sudden gains will be outlined.

1.5.1.1.1. Sudden Gains Criteria.

With regards to the first criterion, Tang and DeRubeis (1999) acknowledged that the seven point drop in BDI scores between consecutive sessions was an arbitrary cut off. Previous studies by Hollon et al. (1992) and Murphy et al. (1984) observed between session BDI score peaks of seven and eight BDI points respectively. Tang and DeRubeis (1999) found no qualitative differences in sudden gains when using BDI cut offs of six and eight points and they suggested the seven-point criterion was large enough to not detect smaller fluctuations in symptoms, and in combination with sudden gain criterion two and three, short-lived reductions in depressive symptoms. The seven-point reduction in BDI symptoms has been largely used by subsequent research examining sudden gains and justified further by the fact that the BDI's reliable change index (RCI; Jacobson & Truax, 1991) is close to Tang and DeRubeis' seven point criterion; with Barkham et al. (1996) find an RCI of 6.18 and Hardy et al. (2005) finding the RCI for the BDI-II is 7.16. Therefore, most studies retain Tang and DeRubeis' first sudden gains criterion. Research using other measures of depression such as the Patient Health Questionnaire (e.g. Masterson et al., 2014) and the Hamilton Rating Scale for Depression (e.g. Vittengl et al., 2005) have used reliable change of the measure for the first sudden gains criterion.

The second criterion specifies that the magnitude of the gain in depression symptoms is at least 25% of the pregain BDI score, which was not justified by Tang and DeRubeis (1999). The authors later note that the criterion "… addresses the concern that more severely depressed patients tend to have more volatile BDI scores."(Tang et al., 2007, p.406). Hardy et al. (2005) note that this criterion is problematic because it assumes that the scale used to measure depressive symptoms (e.g. BDI) are ratio scales rather than interval scales, thus 25%

improvements on one scale may be different to another scale (Koffmann, 2019). When dropping this criterion Stiles et al. (2003) found that only one more sudden gain was detected. Similarly when Tang et al. (2007) dropped this criterion it made a difference to the classification of one individual but not to any results, and Tang et al. (2005) found the second criterion affected the classification of two individuals but not any conclusions, therefore in both studies the criterion was retained. Although Hardy et al. removed this criterion when defining gains within their study, most have kept this criterion to aid comparisons with other studies that have retained Tang and DeRubeis' second criterion.

The majority of the debate has been generated around the third sudden gains criterion, which specifies that the three scores prior to the sudden gain should be significantly higher than the three BDI scores following a sudden gain assessed using a *t* test (Tang & DeRubeis, 1999), because it violates the assumption of independence between scores within a *t* test. Additionally using this criterion means that very early or late sudden gains in therapy are not identifiable when depression scores for three sessions preceding or following the drop in depression symptoms are not available. However, this criterion seems integral to the definition of a sudden gain as when Stiles et al. (2003) removed it they detected over four times as many sudden gains. Subsequently the third criterion was re-worded by Tang et al. (2005) to better follow statistical convention and stated that the pre-and post-gain mean BDI scores should be at least 2.78 times greater than the pooled standard deviations of the two groups of BDI scores, where $p = .05$ when $t(4) = 2.78$, for 'normal' sudden gains which have three pre-and post-gain sessions available. For early and later sudden gains where only two sessions are available Hardy et al. (2005) suggest using a modified *t* distributions of ≥ 2.50 for normal sudden gains and $t \geq 3.00$ for

early and late gains, insofar as $p = .05$ when $t(3) \geq 3.19$. However, this still does not capture very early or late gains. While, Kelly et al. (2005) proposed the gain should exhibit an improvement of at least a 1.5 standard deviation from the individual mean of session-by-session scores, this makes these gains incomparable to those defined using a *t* distribution. In order to capture very early or very late gains, Harries (unpublished thesis, 2016) used $t(4) \geq 2.78$ was used for 'normal' sudden gains, $t(3) \geq 3.19$ for early and late gains, and $t(2) \geq 4.30$ for very early and very late gains. This has also been used by Wucherpfennig, Rubel, Hofmann, et al. (2017) and O'Mahen et al. (2021) to identify sudden gains.

1.5.1.1.2. Research that Uses Modified Sudden Gains Criteria.

A recent meta-analysis of sudden gains across ages, disorders and treatment types found modifications to the sudden gains criteria yielded significantly greater effect sizes (Hedges's $g = 0.72$) compared to the original criteria (Hedges's $g = 0.63$) (Shalom & Aderka, 2020). As the authors note altered sudden gains criterion raise the possibility that different phenomena are being compared. Alternatively, because the original criteria does not allow for the detection of very early sudden gains (i.e. in the first and second therapy session), and early sudden gains have been found to be particularly beneficial on treatment outcomes (Gilboa-Schechtman & Shahar, 2006), this may explain the bigger effect sizes in the modified criteria studies (Shalom & Aderka, 2020). While there is no consensus on the preferred sudden gains criterion, research still tends to use the original sudden gains criteria. Caution should be exercised when comparing sudden gains between studies using different criteria and further research is needed to compare sudden gains between treatments using a consistent sudden gains criterion.

Despite the debate and alterations to sudden gain criteria, studies that use modified sudden gains criteria still find sudden gains are associated with beneficial depression post-treatment and follow up outcomes. In naturalistic, clinic settings, sudden gains (median session = five) were observed in 41% (*n* = 31/76) of patients receiving CBT for depression and were associated with lower depression scores at the end of treatment than those who did not have a sudden gain (Hardy et al., 2005). In 644 adults with an affective disorder receiving treatment based on the cognitive behavioural model in partial hospitalisation setting, Drymalski and Washburn (2011) found 40.7% experienced a sudden gain and they were associated with significantly better treatment outcome than not experiencing a gain. However, in an outpatient sample receiving a 12 session cognitive-behavioural, psychoeducation group treatment for MDD, Kelly et al. (2005) found 41.9% experienced a sudden gain and early gains occurring in the first third of treatment had greater symptom reduction over the course of treatment compared to those who did not have a gain, but there were no statistically significant difference on end of treatment depression scores. Further, in a 12 session BA treatment for adults with MDD, sudden gains occurred in 42.5% (*n* = 17/40) of participants and were associated with better outcome than those who did not have a sudden gain (Masterson et al., 2014). Similarly, in cancer patients with MDD who received BA, 50% (*n* = 13/26) experienced a sudden gain which was associated with lower post-treatment depression scores than those who did not experience a sudden gain (Hopko et al., 2009) and in a 16 week course of BA, 35.7% (15/42) experienced sudden gains and had better outcomes than those who did not have a sudden gain (Hunnicutt-Ferguson et al., 2012). Moreover, in a six-eight session adapted BA treatment delivered by lay counselors in an Indian sample, Singla et al. (2019), found most participants who experienced sudden gains

in their sample (87/150; 58%) also met the criteria for an early response (50% reduction of depressive symptoms by session three) (87/95; 91.58%). This early response and sudden gains were associated with better depression outcomes at three and 12 months post-treatment. Overall, the literature suggests that sudden gains are beneficial on depression treatment outcomes, but relatively few studies have examined how depression sudden gains are associated with other treatment outcomes. Further research is needed to examine this.

1.5.1.1.3 Stability of Sudden Gains.

In addition to criticisms of the sudden gains criteria, there has also been concerns related to the stability of sudden gains. In Tang and DeRubeis' (1999) seminal study they reported that 17% of sudden gains reversed, defined as 50% or more reduction of their depression improvement following the sudden gain. Although fewer sudden gains reversals may demonstrate their stability, reversal rates vary significantly between studies. For example, in CBT based studies reversal rates of sudden gains vary between 19-53% (Abel et al., 2016; Gaynor et al., 2003; Kelly et al., 2005; Lemmens et al., 2016; O'Mahen et al., 2021; Tang et al., 2007; Vittengl et al., 2005). In BA, lower reversal rates have been observed (11.8-13.3%; Hopko et al., 2009; Hunnicutt-Ferguson et al., 2012; Masterson et al., 2014), but this may be due to less reporting of reversal rates in some BA studies (O'Mahen et al., 2017; O'Mahen et al., 2019). To understand why sudden gain reversals may occur, Manning et al. (2010) explored whether reversals were a consequence of within therapy session activity or outside therapy life events in 20 (10 of whom experienced a reversal) patients receiving CBT for MDD. To do so they transcribed the first and last 10 minutes of the therapy tapes for the pre-sudden gain and pre-reversal session for those who experienced a sudden gain reversal. Individuals who did not

experience a reversal were matched by session number to those who experienced a reversal, and the first and last 10 minutes of the matched sessions were transcribed. Independent judges then assessed client (client resistance scale, Mahalik, 1994) and therapist responses (helping skills system, Hill, 2004). Contrary to their expectations, there were no differences between clients who did or did not experience sudden gain reversals in their levels of resistance or therapist response before the sudden gain or the reversal session. However individuals who exhibited a reversal experienced more positive life events in the pre-reversal, compared to the pre-gain session, and there were no group differences between those who experienced a reversal and individuals who did not. The authors note that few life events were reported and thus replication is needed. Following a reversal, six out of the 10 reversals went on to recover the depression symptom improvement they had previously reached during the sudden gain by the end of therapy. This suggests that sudden gain reversals may not be stable, but this may be due to Tang and DeRubeis' reversal criterion. Instead, Wucherpfennig, Rubel, Hofmann, et al. (2017) used a modified criterion and instead identified reversals as 'sudden loss', which is the reverse of a sudden gain (Lutz et al., 2013). In their study, Wucherpfennig et al. (2017) observed 51 (73.9%) patients had a sudden gain reversal, but only 26 (37.7%) of those experienced a sudden loss. It is unclear the best criterion to use to understand reversals of sudden gains and much of the sudden gains research (Shalom & Aderka, 2020) still uses Tang and DeRubeis' original reversal criterion.

Related to this, there has also been concerns that sudden gains may be a snapshot of random fluctuations in symptoms rather than a distinct pattern of change. Thomas and Persons (2013) suggested that the sudden gain pattern may be the largest drop in symptoms occurring within a more gradual pattern of change

over treatment. In simulated data of 88 patients with mood disorders (86% unipolar major depression) receiving CBT treatment they found within a gradual course of symptom improvements sudden gains occurred, suggesting that this rapid pattern of change is an artefact of linear change. Further, they found that sudden gains did not uniquely predict treatment outcome beyond the symptom variation in the first six sessions. To further examine the sudden gains criteria compared to random symptom fluctuations, Vittengl et al. (2015) used three sudden gains criterions and Monte Carlo simulations to examine how often each criterion would produce Type I errors. They found that random fluctuations in the simulated data were not distinct from sudden gains reported in the literature, suggesting that false positive sudden gains are highly likely to be reported in the literature. To address this issue, Andrews et al. (2020) examined whether sudden gains were an artefact of linear change in therapy as suggested by Thomas and Persons (2013), and they also examined whether sudden gains were distinct from defined and undefined trajectories of change as identified by Vittengl et al. (2013) (linear, log-linear, one-step and undefined). They found that sudden gains were associated with better treatment outcomes than linear trajectories of symptom change in a treatment resistant sample receiving CBT as an adjunct to pharmacotherapy, suggesting that sudden gains are a robust pattern of change over and above general symptom variability within treatment. Consistent with Vittengl et al. (2013) they found defined, compared to undefined, trajectories of symptom change across treatment predicted better depression outcomes at 6- and 12-months post-treatment. This evidence suggests that sudden gains are a meaningful pattern of change, above and beyond general symptom variability within treatment.

1.5.1.1.4 Tautology of Sudden Gains.

Lastly, in addition to discussion and critiques about the definition of sudden gains, there are also concerns about the tautology of sudden gains in relation to outcome. It has been suggested that comparing end of treatment outcomes in individuals who have a sudden gain to those who do not signifies a tautology because we know that those who experience a sudden gain have had a large reductions in symptoms and therefore are likely to have more improvement (Kelly, Roberts, et al., 2007; Koffmann, 2019). Kelly, Roberts, et al. (2007) note that the lack of a sudden gain does not mean that individuals in the no sudden gain group have not experienced significant but steady symptom improvements across the course of treatment. It has been suggested that by using a different clinical measure to examine depression treatment outcomes than that of the measure used to identify sudden gains may reduce this tautology (Kelly, Cyranowski, et al., 2007). However, it is still the case that depression is being measured and different measures are assessing similar symptoms. Other research has found that in the presence of other depression change patterns that sudden gains are robust pattern of change (Andrews et al., 2020) and therefore suggests we should be less concerned about the tautology.

Despite the debate surrounding the sudden gains criterion, a wealth of evidence demonstrates that sudden gains are seen across disorders and treatments, and are associated with beneficial treatment outcomes (Shalom & Aderka, 2020). Sudden gains were originally identified as times at which to explore potential mechanisms of change and the current thesis will focus on the processes of change in sudden gains in CBT and BA.

1.5.1.2 Using Sudden Gains to Examine Processes of Change in CBT and BA

Overall, research suggests that sudden gains between treatments, including CBT and BA, are analogous in terms of their relationship to depression treatment outcomes. However, the majority of this research compares sudden gains between studies where there may be sample, setting and sudden gain criterion differences. A recent meta-analysis of sudden gains showed that sudden gains in CBT (Hedge's *g* = 0.72) did not differ from sudden gains in non-CBT (Hedge's *g* = 0.57) treatments in terms of the association with outcome. However BA was classified as a CBT treatment and therefore it is unclear whether CBT sudden gains differ to BA sudden gains in terms of their association with treatment outcome (Shalom & Aderka, 2020). Only one study using data from a large RCT has directly compared sudden gains in CBT and BA, using a consistent definition of sudden gains. In this study, O'Mahen et al. (2021) found 29% (86/300) of individuals experienced a sudden gain across CBT and BA, with there being no difference in the rates or timings between treatments. There was a significant main effect of sudden gain. Individuals in either treatment who had a sudden gain were more likely to have lower depression scores at 6-, 12-, and 18-months than those who had not had a sudden gain during treatment. However, treatment type moderated that relationship such that those who experienced a sudden gain in CBT, compared to sudden gain in BA, had significantly lower depression scores at 6- and 18-month treatment outcome. This suggests that perhaps there might be an advantage of sudden gains experienced within CBT on longer term treatment outcomes. Because CBT and BA are closely related treatments, comparing sudden gains between these treatments can enable us to further understand whether cognitive processes may instigate a sudden gain.

A three-stage model of how cognitive change may preceded a sudden gain, as well as how cognitive processes may help enhance the sudden gain and lead to beneficial treatment outcomes was outlined by Tang and DeRubeis (1999). They proposed within the first stage of the model, known as the 'preparation stage', therapists cultivate alliance with the patients and teach the cognitive model of depression and cognitive techniques, but few cognitive changes occur. In the second stage, the 'critical session', belief and schema changes are posited to occur and these are hypothesised to lead to the large decrease in depression symptoms (the sudden gain). Within the last stage of the model, the drop in depression symptoms is hypothesised to result in further improvements in the therapeutic alliance and additional cognitive changes. This is then posited to create a positive feedback loop of further positive cognitive change and depression symptom improvements, coined the 'upward spiral', to lead to sustained depression recovery following treatment. The preparation stage was supported by the findings that individuals who experienced a sudden gain had greater cognitive change (Cohen's *d* = 0.65) in the pregain session compared to a control session (two sessions prior to a sudden gain, known as the 'prepregain session') (Tang & DeRubeis, 1999). Other process such as therapist application of CBT concrete and abstract techniques and therapeutic alliance were not found to change prior to a sudden gain (Tang & DeRubeis, 1999) suggesting that cognitive changes are the facilitators of sudden gains. Further, they observed greater therapeutic alliance after the gain (Cohen's *d* = 0.75), compared to the pregain session, and greater cognitive changes in the postgain compared to the prepregain session (Cohen's *d* = 0.77) in individuals who experienced a sudden gain in CBT, supporting the 'upward spiral' hypothesis.

1.5.1.3 Predictors of Sudden Gains.

Subsequently process research has explored potential client processes of change that may help to facilitate sudden gains in treatment with the view that therapy strategies could then be used to help generate sudden gains. In support of Tang and DeRubeis' sudden gains model in CBT, Tang et al. (2005) found greater cognitive change in the pregain compared to a control session (two sessions prior to the drop in depression symptoms) in both automatic thought (behavioural activation and automatic thought interventions) treatment and CBT. Further, in group-based CBT for anxiety disorders changes in anxiety related cognitions were found to change prior to depression sudden gains (Norton et al., 2010; Vincent & Norton, 2019). Similarly in CBT for PTSD, change in cognitive processes (negative trauma-related appraisals) from the prepregain to the pregain session were found to be associated with sudden gains. However, other research has failed to find change in cognitive processes precede sudden gains in CBT. Changes in hope were not found to be associated with sudden gains in individuals receiving CBT for MDD (Lemmens et al., 2016). In CBT for individuals with treatment resistant depression, individuals with sudden gains expressed significantly higher levels of hope than those who did not have a gain, but levels of hope and emotional processing did not change between the control and pregain sessions for individuals who experienced a sudden gain (Abel et al., 2016). Similarly, in a recent study exploring change in processes surrounding a sudden gain, Lemmens et al. (2021) also did not find cognitive changes preceded sudden gains in individuals with MDD receiving CT in outpatient settings. This is despite using the same cognitive rating scale (PCCS) as research that has found change in cognitions prior to a sudden gain (Tang & DeRubeis, 1999; Tang et al., 2005). Outside of depression research, in two separate studies examining sudden gains in CT for social anxiety disorder cognitive change did not

precede sudden gains (Bohn et al., 2013; Hofmann et al., 2006). It is noteworthy that all the studies to date exploring cognitive processes preceding a sudden gain examine *change* in cognitive processes from a control and the pregain session, rather than examining whether levels of pregain process variable directly predict a sudden gain (Aderka & Shalom, 2021). It is possible that by examining directly whether processes in the pregain session predict having a sudden gain may elucidate important mechanisms involved in sudden gains. Other methodological differences between studies such as the use of observer ratings compared to self-report ratings of cognitive process, altered sudden gains criteria, and when cognitive change is examined (before or during the therapy session) (Aderka & Shalom, 2021) make comparisons between studies difficult.

Further, it is unclear whether client cognitive processes reliably precede the onset of sudden gains and are uniquely present in CBT, which explicitly utilises cognitive change strategies, or whether cognitive change also happens in treatments where cognitive change strategies are proscribed (Lorenzo-Luaces et al., 2015), like BA. In non-cognitive therapies cognitive processes such as change in hope in interpersonal therapy (Lemmens et al., 2016), and in psychotherapy (Adler et al., 2013) have not been found to precede depression sudden gains. However, change in processing was found to precede sudden gains in mental health in routine clinical settings (Adler et al., 2013). To date few studies have examined whether cognitive processes predict a sudden gain in BA. In a study examining sudden gains in Jacobson et al.'s (1996) BA sample, cognitive changes measured on the PCCS (Tang & DeRubeis, 1999) did not significantly differ between the pregain and control sessions (Andrusyna, 2007). Additionally, baseline dysfunctional attitudes did not predict sudden gains in BA (Hunnicutt-Ferguson et al., 2012). However, whether

within therapy cognitive processes may be associated with a sudden gain in non-cognitive therapies is still poorly understood and further research is needed to explore this.

The mixed findings regarding the role cognitive processes have in triggering a sudden gain may indicate that, contrary to Tang and DeRubeis' (1999) hypothesis, other processes may also be important for the generation sudden gains CBT and BA. Integral to the therapeutic framework of both CBT and BA are behavioural strategies to activate an individual and reduced avoidance which can maintain low mood. Yet little research has examined the role of behavioural factors preceding sudden gains in CBT and BA. One study found baseline levels of brooding rumination and activation did not predict experiencing a sudden gain in group BA treatment for individuals with recurrent depression (O'Mahen et al., 2019). To our knowledge only one study has examined within treatment behavioural processes and the association with sudden gains in BA. Andrusyna (2007) found significantly greater agreement to behaviour change in the pregain compared to control sessions in Jacobson et al.'s (1996) BA sample. Additionally, Andrusyna (2007) also found individuals engaged in a greater number of positive activities between the prepregain and pregain sessions, compared to control sessions. Outside of BA in a recent study, Lemmens et al. (2021) observed approaching significant change in behavioural processes between a control and the pregain session in CBT. The authors rated within session behavioural changes in individuals receiving CBT for MDD in an outpatient setting. Although not statistically significant there was greater preparation for change including acceptance of new behaviours, making plans for and increasing pleasurable activities, observed in the pregain, compared to a control session. This suggests that behavioural processes may also contribute to the

instigation of sudden gains, but further exploration of the role behavioural processes and sudden gains is needed within CBT and BA.

There has also been considerable interest in examining whether individuals with certain baseline clinical characteristics or demographic factors are more likely to experience a sudden gain. If there were particular characteristics that individuals with sudden gains possess then it may be possible for therapists to be aware and anticipate this pattern of change to maximise treatment outcomes. Further, this may also be helpful to understand which individuals may not be likely to experience a sudden gain, whom we know may have worse treatment outcomes. Because sudden gains have also been identified outside psychological therapy (Kelly, Roberts, et al., 2007), in pill placebo treatment (Vittengl et al., 2005), and prior to treatment commencement (Busch et al., 2006; Gaynor et al., 2003) this suggests rather than therapy factors, particular individual characteristics may also be involved in the instigation of sudden gains, or help individuals to harness sudden gains that then lead to beneficial treatment outcomes. The majority of this research has tended to explored baseline levels of clinical characteristics or demographic factors and assess whether they predict sudden gains, but across therapies and disorders no robust predictors have been identified. A range of patient factors have been explored in relation to the association with sudden gains including age, gender, relationship status, education level, income, baseline depression severity, social support, functioning, and number of treatment sessions (Hunnicutt-Ferguson et al., 2012; Keller et al., 2014; Kelly et al., 2005; Masterson et al., 2014; O'Mahen et al., 2021; O'Mahen et al., 2017; O'Mahen et al., 2019), but have not been found to differentiate between individuals who do and do not experience a sudden gain across treatments for depression. Recent research has used machine learning methods to assess

predictors of sudden gains. In 547 individuals receiving psychotherapy in an outpatient mental health setting in Chile, Zilcha-Mano et al. (2019) found no robust predictors (age, gender, education, baseline symptom severity, previous psychiatric hospitalisation, baseline tendency to self-conceal, first treatment alliance) of sudden gains using machine learning methods. Similarly, Aderka et al. (2021) in a sample of 1514 individuals receiving depression treatment in a partial hospital setting also failed to find robust baseline demographic and clinical characteristic predictors (age, gender, marital status, education level, employment status, previous hospitalisation, agoraphobia, panic disorder, GAD, social anxiety disorder, posttraumatic stress disorder, OCD, pre-treatment depression and anxiety levels) of sudden gains. This suggests that there may not be possible to predict who may have a sudden gain at the beginning of treatment based on these characteristics.

Another line of exploration is whether certain demographic or baseline clinical factors moderate the association between a sudden gain and treatment outcomes. It is possible that specific baseline characteristics or demographic factors may impact an individuals' ability to capitalise on a sudden gain and to enhance the upward spiral to lead to favourable treatment outcomes. In a meta-analysis of sudden gains across ages (children, adolescents, and adults) psychotherapies for a range of disorders, including depression, Shalom and Aderka (2020) did not find that pre-treatment severity levels of the disorder, gender or age moderated the association between sudden gain and post-treatment outcome, but greater number of therapy sessions resulted in smaller effects of the sudden gain at treatment outcome. However, it is unclear whether in a sample of adults with a primary presenting problem of depression, whether particular baseline clinical factors of demographic characteristics would moderate the impact of a sudden gain on treatment outcomes.

The absence of robust predictors of sudden gains has led to a revised theory of how sudden gains occur (Aderka & Shalom, 2021). This theory suggests that rather than treatment directly causing sudden gains, they are primarily the result of continual natural fluctuations of depression symptoms which occur both prior to and within treatment. In the context of treatment these natural fluctuations become gradual symptom reductions and this is when a sudden gain occurs. The therapy helps to harness the sudden gain and lead to beneficial treatment outcomes (Aderka & Shalom, 2021). This theory is supported by research from Shalom et al. (2018) who found in 260 patients from three different datasets (RCT of prolonged exposure of children and adolescents with PTSD; RCT of cognitive, behavioural and pharmacological treatment for OCD; psychodynamic treatment for adults) that within treatment (prior to a sudden gain) intraindividual variability in symptoms predicted sudden gains even when controlling for change occurring prior to and after a gain. Extending this research, in 101 adults who received internet CBT for social anxiety disorder, Shalom et al. (2020) found both within treatment variability prior to a sudden gain and pre-treatment variability both predicted sudden gains. Although this might suggest that variability in symptoms are important determinants of sudden gains, it is perhaps premature to stop examining other within therapy processes that may instigate sudden gains when little research has examined cognitive factors in non-cognitive therapies and behavioural processes in both cognitive and behavioural therapies, like CBT and BA, in relation to sudden gains.

1.5.1.4 Examination of Postgain Processes.

It is also important to elucidate how sudden gains lead to beneficial treatment outcomes and long term follow up. If, as Aderka and Shalom (2020) suggest, sudden gains are not directly caused by treatment and instead are times of symptom

fluctuation that interact with treatment to produce some process that leads to beneficial treatment outcomes, it is important to understand what processes are occurring following a sudden gain and whether these processes are associated with positive treatment outcomes. However, in comparison to the research examining processes that precede a sudden gain relatively few studies have explored what occurs after a sudden gain and whether specific processes are associated with treatment outcomes. This is despite the potential clinical benefits of employing therapeutic strategies within treatment after a sudden gain to maximise adaptive and minimise maladaptive processes, and further cultivate the benefits of a sudden gain.

There has been some evidence which supports Tang and DeRubeis' (1999) hypothesis that improved therapeutic alliance occurs after the sudden gain and may contribute to the upward spiral. In a sample of 211 patients with MDD who received CBT in a routine care sample in Germany, Wucherpfennig, Rubel, Hofmann, et al. (2017) used propensity score matching (PSM) to match individuals who did and did not have a sudden gain by baseline characteristics, treatment length or time point of pregain session. Client reported therapeutic alliance and coping skills in the postgain session was significantly higher in those who had a sudden gain, compared to no gain. Further, greater postgain alliance in those who had a sudden gain was associated with lower depression scores at the end of treatment (Wucherpfennig, Rubel, Hofmann, et al., 2017). In a large German outpatient sample who received CBT and interpersonal therapy across disorders (depression, anxiety, and other disorders), Lutz et al. (2013) found after a sudden gain individuals reported greater therapeutic alliance compared to after a sudden loss (sudden upward shifts of depression symptoms that do not return). In further support, Zilcha-Mano et al. (2019) found experiencing a sudden gain strengthened alliance after a gain which

then predicted improved functioning and greater life satisfaction two sessions following the critical sudden gains session in an outpatient trial of primarily depressed individuals.

There has been less research that has examined changes in cognitive processes in the postgain session. In a trial sample of individuals receiving CT or IPT for social anxiety disorder, Bohn et al. (2013) observed reductions in the frequency and the strength of belief of negative cognitions in the postgain session compared to the pregain session. A recent study used an observational coding system to examine a range of cognitive, behavioural and interpersonal processes in sessions around a sudden gain in depressed patients receiving CBT (Lemmens et al., 2021). Contrary to Tang and DeRubeis' sudden gains hypothesis, there were no differences between the pregain and postgain session in the levels of cognitive processes. There is a need to further examine whether change occurs in cognitive and/or behavioural processes following a sudden gain in CBT, or in therapies that proscribe cognitive strategies, like BA, and assess whether they may be involved in the upward spiral and lead to beneficial treatment outcomes.

1.5.2 Summary of Sudden Gains Research to Date and Future Directions

In sum, the literature suggests that sudden gains are robust patterns of discontinuous change and have beneficial effects both at the end of treatment, but also in the longer term across treatments for MDD. Most of this research has been conducted in trial samples and less may be known about how sudden gains are associated with treatment outcomes, such as depression but also other problems that are commonly associated with depression like anxiety and functioning, in everyday clinic-based settings. Additionally, further research is needed to examine whether baseline clinical and demographic factors may influence the effects of a

sudden gain on treatment outcomes. Although there has been a lot of exploration of the processes that may be involved in the generation of a sudden gain there are still no robust predictors of sudden gains, and less is known about what occurs following a sudden gain. Some literature suggests cognitive processes both facilitate and follow a sudden gain and are responsible for an upward spiral which ultimately leads to beneficial treatment outcomes in cognitive based treatments like CBT. However, little research has explored whether cognitive processes precede and follow a sudden gain in non-cognitive based treatments like BA. Given that both therapeutic frameworks encompass behavioural strategies it is also surprising little research (Lemmens et al., 2021) has explored the role of behavioural processes in both instigating a sudden gain but also the upward spiral in both therapies. Additional research is needed to understand the processes surrounding sudden gains in CBT and BA which may help to understand how treatments lead to depression symptom reductions.

1.5.3 Depression Spikes

Another pattern of discontinuous change identified in the depression literature is a depression spike. The identification of depression spikes stemmed from complex systems theory which hypothesises that discontinuous patterns are an indication of tipping points, known as order transitions, which signal an imminent transition from one state to another (Hayes & Andrews, 2020). Depression spikes were first observed in an Exposure-Based Cognitive Therapy (EBCT) treatment for depression (Hayes et al., 2005). EBCT treatment[2] was specifically designed to generate

[2] EBCT therapy consists of three phases to increase patients' health behaviours and enhance resilience, directly target rumination and avoidance, as well as to address cognitive, behavioural, emotional and interpersonal issues (Hayes & Harris, 2000). The first (sessions 1-8) phase, the 'stress management phase', focuses on helping patients increase healthy behaviours and habits. The

destabilisation in the depression network by encouraging clients to approach and explore distressing content such as describing core negative views of the self and feelings of hopelessness, as well as content that was previously avoided, during the 'exposure phase' of treatment (Hayes & Harris, 2000). Consistent with this and other research that has observed early rapid changes in therapy (Shalom & Aderka, 2020; Tang & DeRubeis, 1999; Tang et al., 2005; Vittengl et al., 2005), Hayes et al. 2007 observed a cubic pattern of depression symptom change in treatment, beginning with an initial early, rapid drop in depressive symptoms followed by a depression spike in the middle of treatment (as seen in Figure 1.4). Depression spikes during the exposure phase of treatment were experienced by 62% of patients and associated with lower depression levels and higher rates of remission, compared to those who did not experience a depression spike, suggesting this discontinuous pattern of change can have beneficial effects on depression outcomes in this treatment. Examining processes during the depression spike more cognitive emotional processing was found to occur during the spike session than those who did not experience depression spikes, whereas no differences in levels of hope were found. Subsequently, this cubic pattern of depression change has been identified in EBCT in other samples. In 21 Swiss outpatients with MDD receiving EBCT, Holtforth et al. (2012) found a cubic pattern of change was associated with lower post-treatment avoidance and depression symptoms. Levels of cognitive-emotional processing were higher during this cubic pattern of change in treatment. Further in an RCT of ECBT compared to CBT for MDD, Holtforth et al. (2014) found a cubic

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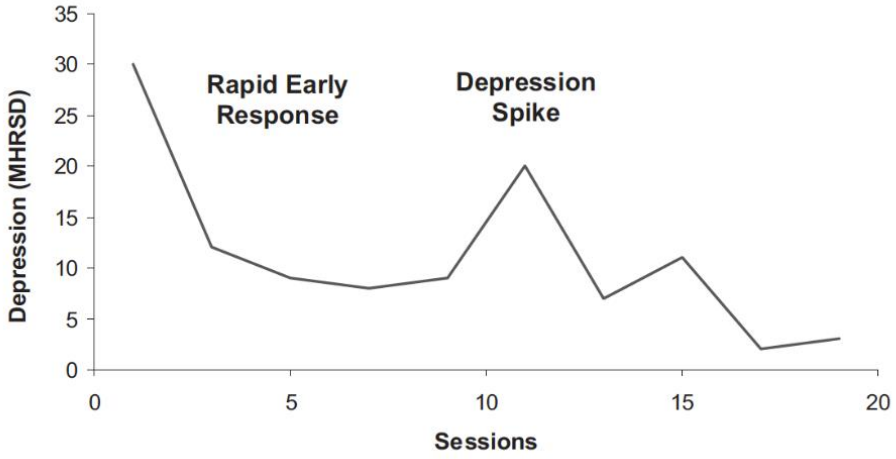
second (sessions 9-18), the 'exposure phase', aims to activate the depressive network by actively approaching previously avoided, depressive content with the aim to disrupt the depressive patterns. The third phase (sessions 9-posttreatment), known as the 'consolidation and positive growth phase' aims to solidify the new learning and develop a balanced view of the self.

pattern of change in ECBT and a quadratic pattern of change in CBT, characterised by a decrease in symptoms in early therapy with less symptom change following the initial decrease. Greater emotional processing was reported by patients in ECBT than CBT and predicted more improvement in depression scores and wellbeing outcomes. This suggests that cubic patterns of change, which include a depression spike are beneficial in EBCT.

Figure 1. 4

Depiction of a Depression Spike from Hayes, et al. (2007)



However, depression spikes have also been identified in treatments that do not employ therapeutic strategies to encourage destabilisation in the depression network to induce a depression spike and in treatments which proscribe cognitively exploring depressing content. In these treatments the association between depression spikes and treatment outcomes are varied. For example, in 200 individuals with PTSD, 54% of whom met the criteria for MDD, receiving prolonged exposure therapy or ADM, Keller et al. (2014) found 22.5% experienced depression spikes. There were no difference in rates of spikes between treatments, and spikes were not associated with post-treatment depression or PTSD severity. Similarly, in

CBT adjunct to pharmacotherapy, Abel (2014) found 50% of individuals with treatment resistant depression experience a spike, but they were not associated with depression treatment outcome. In group BA for MDD, 10% of patients experienced a depression spike (O'Mahen et al., 2019) and in internet BA for postpartum depression 19% experienced a depression spike (O'Mahen et al., 2017); in both studies there was no significant differences in individuals who did and did not have a depression spike on depression treatment outcome. Conversely in a recent study, O'Mahen et al. (2021) assessed depression spikes in an RCT sample of 300 adults with MDD. Across both therapies, 86 (29%) individuals experienced a depression spike. At 6-, 12- and 18-month follow up individuals who experienced a depression spike had significantly higher depression scores in both CBT and BA. Although non-significant, individuals who experienced a depression spike in CBT, compared to BA, had higher depression scores at 18 months post-randomisation (O'Mahen et al., 2021), suggesting in non-exposure CBT depression spikes may be more harmful. Compared to the sudden gains literature, the research examining depression spikes is limited, especially across different therapy approaches, including third-wave therapies, and further replication and extension is needed to understand depression spikes in treatments which do not purposefully bring them about.

One possible reason for the differences in rates and association with treatment outcome between Hayes et al.'s (2007) original study and subsequent research may be the criteria used to identify depression spikes. The original study specified a depression spike must decrease by seven depression points or more within the same phase of therapy (Hayes, Feldman, Beevers, et al., 2007). However, studies examining depression spikes in non-EBCT therapies often do not have distinct treatment phases and there are several modifications to the criteria in non-

EBCT treatment. For instance, Keller et al. (2014) identified depression spikes as occurring at any point during treatment with depression scores decreasing by at least seven depression points during the remaining sessions of the 10 week PTSD treatment. On the other hand, O'Mahen et al. (2017) and O'Mahen et al. (2019) examined depression spikes occurring after session three in BA when participants began to engage in approach-related behaviour, which is in line with Hayes et al.'s (2007) reasoning that spikes may be brought about during periods of intensive change. They defined decrease of symptoms in a depression spike as a reduction of depression by four points or more on the Hamilton Rating Scale for Depression within three sessions. Two other studies (Abel, 2014; O'Mahen et al., 2021) defined depression spikes as an increase of seven or more depression points followed by a decrease by at least seven depression points within a six session period. Although conceptually similar to Hayes et al.'s original depression spikes, the different operationalisation of depression spike definitions may suggest that different concepts are being measure in studies that do not use the original criterion. Nevertheless, O'Mahen et al. (personal communications) assessed the average number of sessions it took for a depression spike to recover in a trial dataset of non-exposure CBT and BA for depression, and found this to be three sessions (M = 2.52, SD = 2.11). A comparison of the modified and original depression spike criterions yielded the same number of depression spikes identified (O'Mahen et al., personal communications), suggesting altering the criteria does not affect the rate of spikes identified. Another consideration is the timing in therapy at which depression spikes occur. Hayes et al. (2007) observed depression spikes in the middle of treatment, when there is an opportunity for corrective processing, and they associated with positive treatment outcomes. Although research examining depression spikes

outside of EBCT treatment have not limited to exploring spikes only in the middle of treatment, the majority of depression spikes tend to occur midway through treatment in non-EBCT treatments (session 9/18, Abel, 2014; session 5/12, O'Mahen et al., 2017; session 3 or 4/10, O'Mahen et al., 2019; session 5/10 Keller et al., 2014). Nevertheless because of depression spikes criteria modifications caution is needed when comparing depression spikes across studies that use different definitions and there is a need to examine why depression spikes may occur in non-EBCT therapies.

Currently, in therapies that do not use purposeful therapeutic strategies to bring about a temporary worsening of depression symptoms, it is unclear what a depression spike represents. To assess why depression spike may occur, some research has examined baseline predictors of depression spikes. Demographic variables including age, education, ethnicity, relationship status (Keller et al., 2014; O'Mahen et al., 2021) and baseline clinical variables such as behavioural activation, dysfunctional cognitions (O'Mahen et al., 2021) have not been found to be associated with depression spikes in treatment. Other studies could not examine baseline clinical and demographic characteristics because of the low frequency of depression spikes identified (O'Mahen et al., 2017; O'Mahen et al., 2019). Only PTSD specific predictors (negative trauma related support) in pre-treatment has been found to be associated with having a depression spike during therapy for PTSD (Keller et al., 2014). While baseline demographic and clinical characteristics may not be associated with depression spikes, it is possible that these characteristics may influence the relationship between depression spikes and treatment outcomes and this is yet to be explored.

With regards to in-treatment processes around the depression spike pattern of change, reductions in avoidance (Hayes et al., 2005) and greater levels of processing (Hayes et al., 2005; Hayes, Feldman, Beevers, et al., 2007) have been found to occur with a depression spike. Additionally, levels of processing significantly mediated the association between depression spikes and reduced depression levels at treatment outcome (Hayes, Feldman, Beevers, et al., 2007). These findings are consistent with Hayes et al.'s (2007) theory that depression spikes represent a time of processing and re-organisation of depressionogenic material. Outside EBCT treatment, in CBT and BA it is theoretically possible that similar processes occur during a depression spike and could be associated with treatment outcomes. Alternatively, other theoretically relevant processes in CBT and BA may drive depression spikes and their association with treatment outcomes. However, because CBT and BA do not purposefully bring about a transient worsening of depression symptoms in therapy it is also possible that depression spikes represent other, out of treatment influences. Although speculative, life stressors or events may instigate a depression spike. Alternatively, therapist influences, such as negative therapeutic relationship (Safran & Muran, 2000) may contribute to depression symptom worsening, or depression spikes may even represent the iatrogenic effects of therapy. The current available research into process of change surrounding depression spikes in CBT and BA for depression is limited and further investigation is needed.

1.5.4 Summary of Depression Spikes Research to Date and Future Directions

In summary, depression spikes in EBCT therapy have been found to be associated with beneficial clinical outcomes. However, depression spikes also occur in treatments where there are no intended therapeutic processes to bring them

about, like in CBT and BA, and it is less clear whether they are associated with advantageous or detrimental treatment outcomes and what they represent. Further research is needed to explore depression spikes in treatments that do not purposefully bring them about to assess how they influence treatment outcomes, and to examine the client and therapist predictors of depression spikes to elucidate what they represent.

1.6 Summary and Aims of the Thesis

In summary both CBT and BA treatment for adult depression have been found to be effective at reducing depression symptoms. Despite the range of evidence examining processes of change in each treatment, it is unclear whether cognitive, behavioural or other processes of change are important for depression symptom change. Elucidating the processes of change in both therapies could help us to further enhance the treatments and clinical outcomes. One way to identify the optimal times in therapy to examine processes of change is to look at times when rapid, discontinuous depression change occurs. The psychotherapy literature has identified a number of different patterns of depression symptom change, and this thesis will focus on two of these patterns; sudden gains and depression spikes. The thesis aims to answer several broad questions:

1. What are the rates and timings of sudden gains and depression spikes in every day clinical practice, and what is their relationship with treatment outcomes in CBT and non-CBT therapies?
2. What are the key client processes or therapist strategies preceding and following discontinuous change in depression symptoms, and are they moderated by treatment type (CBT/BA))
3. Do key client processes or therapist strategies preceding and following discontinuous change predict treatment outcomes, and are these moderated by treatment type (CBT/BA)?

The first study (chapter two) aimed to address the first thesis question and examines both sudden gains and depression spikes in a large, clinic based naturalistic dataset. The aim of this study was to address some important gaps in the

literature that has examined discontinuous patterns of change in psychotherapies for depression. The majority of research examining these patterns of depression change has done so in RCT samples where individuals and therapy characteristics (e.g. session length) may differ compared to those who receive psychological therapies for depression in everyday clinical based settings. Therefore this study assessed the rates and timing of sudden gains and depression spikes, and their association with treatment outcomes in four therapies for depression (LiCBT, HiCBT, counselling and group therapy) in a large clinic based dataset. Furthermore, this study extends the current literature to assess how sudden gains and depression spikes are associated with anxiety and functioning as well as depression outcomes at the end of treatment, and explored whether any baseline client demographic or clinical characteristics may moderate these associations.

The second study (chapter three) addressed thesis questions two and three and used data from a RCT of CBT and BA for adult depression. The study examined whether client cognitive processes predicted a sudden gain in CBT, as suggested by Tang and DeRubeis' (1999) theory of sudden gains. Additionally we investigated whether cognitive processes may contribute to sudden gains in BA. This study also examined whether therapeutically important behavioural processes were associated with a sudden gain in CBT or BA. To explore processes that may contribute to the 'upward spiral' hypothesis (Tang & DeRubeis, 1999) we examined whether experiencing a sudden gain predicted cognitive or behavioural processes following a sudden gain. Lastly, pregain and postgain cognitive and behavioural processes in individuals who do and do not experience a sudden gain were examined to see if they were associated with treatment outcomes, and whether treatment type (CBT/BA) moderated these relationships.

The third study (chapter four) aimed to address thesis questions two and three and used the same RCT dataset as study two to examine therapeutically important client processes of change and therapist strategies to understand why depression spikes occur in CBT and BA. Additionally, as the literature examining depression spikes and their relation to treatment outcomes has been mixed, this study examines whether therapeutically important processes during a spike may influence treatment outcomes, and if these associations are moderated by treatment type (CBT/BA).

The fourth and final study (chapter five) addresses thesis question two and is a prospective study focusing on a key depressionogenic process variable (avoidance) over depression change. Behavioural processes at key junctures for individuals have been understudied relative to cognitive processes and this study aimed to further understand the reciprocal relationship between cognitive and behavioural avoidance, and depression symptoms. To understand this, the study examined the relationship between avoidance and depression during a stressful period of time when we would expect some depression mood variability- during final year undergraduate examinations. This stressful life event was used as a proxy for naturally occurring discontinuous depression change and we examined how behavioural and cognitive avoidance influences prospective depressed mood or vice versa (how depressed mood influence future avoidance). This study aimed to help understand the evolvement of avoidance and mood.

The final chapter (chapter six) discusses how the results of each study within the thesis relates to the wider literature and considers the methodological, theoretical and clinical implications of the thesis findings.

Chapter One Appendices

Appendix 1

BA Session Chart from the COBRA Trial BA Protocol

<table>
<tr><td colspan="6">PHASE I</td><td>TRANSITION</td><td colspan="8">PHASE II</td><td>TRANSITION</td><td colspan="4">PHASE III</td><td colspan="4">BOOSTER</td></tr>
<tr><td>1</td><td>2</td><td>3</td><td>4</td><td>5</td><td>6</td><td>7</td><td>8</td><td>9</td><td>10</td><td>11</td><td>12</td><td>13</td><td>14</td><td>15</td><td>16</td><td>17</td><td>18</td><td>19</td><td>20</td><td>21</td><td>22</td><td>23</td><td>24</td></tr>
<tr><td colspan="2">Assessment/rationale Formulation diagram</td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td></td><td>Goal setting and introduction of valued activities</td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td><td></td></tr>
<tr><td colspan="19">Self-monitoring leading to activity scheduling</td><td></td><td></td><td></td><td></td></tr>
<tr><td></td><td></td><td></td><td colspan="17">Avoidance-Functional analysis/TRAP and TRAC; developing formulation-diagram</td><td></td><td></td><td></td><td></td></tr>
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<tr><td></td><td></td><td></td><td></td><td></td><td></td><td colspan="14">Carry on Activating (up your hierarchy….) including grading and stress testing</td><td></td><td></td><td></td><td></td><td></td></tr>
</table>

						Additional module choices guided by functional analysis (**mandatory**/optional) **Rumination**; **Problem solving**; Functional equivalence (including values); Anxiety; Punishment; Communication; Alcohol and Substance Use													
															Relapse Prevention/ Maintaining Progress					

Appendix 2

CBT Session Chart from the COBRA Trial CBT Protocol

PHASE I						TRANSITION	PHASE II								TRANSITION	PHASE III				BOOSTER			
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24
Assessment/rationale/ agreed presenting issues/shaping towards goals. Descriptive case formulation diagram																							
	Goal setting and first interventions					Progress to goals reviewed									Progress to goals reviewed								
Homework																							
Behavioural experiments																							
	Behavioural interventions: Activity and mastery and scheduling pleasurable /rewarding activities																						
		Identifying and responding to automatic thoughts																					
						Identifying conditional assumptions and using cognitive and behavioural strategies to reframe conditional assumptions and articulate and test out more adaptive beliefs. Cross-sectional case formulation.																	
															Longitudinal case formulation, only if necessary, identifying and working with core beliefs, again only if necessary.								
																Relapse Prevention/ Maintaining Progress							

Chapter Two: Sudden gains and depression spikes in a large primary mental healthcare sample

Study 1

Asha Ladwa [a], Heather O'Mahen [a], Adele Hayes [b], Kim Wright [a]

[a] Mood Disorders Centre, University of Exeter, Washington Singer Building, Exeter, EX4 4QG, UK

[b] University of Delaware, 226 Wolf Hall Newark, DE 19716 302-831-0484

2.1 Preface

The first study aimed to address some gaps within the current literature examining discontinuous change in psychological treatments for depression. In the current literature, sudden gains (Tang & DeRubeis, 1999) and depression spikes (Hayes, Feldman, Beevers, et al., 2007) in the most part have been examined within relatively small, randomised controlled trial (RCT) samples. It is important to understand whether they occur at the same rate, timing and association with treatment outcomes in every day, clinic settings. The current study sought to a) assess the rates and timings of sudden gains and depression spikes in four therapies for depression (low- and high-intensity CBT, counselling, and group treatment) in naturalistic, clinic based settings in a sample of individuals with depression as a primary presenting problem, b) assess the association of sudden gains and depression spikes on depression treatment outcomes, c) extend the current literature and assess the association of these two patterns of change on anxiety and functioning outcomes which are often related to depression, and d) to examine whether the association between these two patterns of discontinuous depression change and treatment outcomes are moderated by treatment and baseline clinical and demographic characteristics.

The current study used data from the Improving Access to Psychological Therapies (IAPT) service, which is a stepped-care service that provides psychological therapies for mental health problems in England. The main body of this chapter consists of a paper that is currently being prepared for publication and the intention is to submit to the journal of Behaviour Research and Therapy.

2.2 Abstract

Background: Discontinuous depression change is associated with treatment outcome in trials. This study examined whether two types of discontinuous change, sudden gains and depression spikes, were associated with depression, anxiety and functioning outcomes in a large clinic-based sample, and whether these were moderated by treatment and baseline clinical and demographic factors.

Method: Data from 9,444 individuals with depression in UK primary care mental health services were examined. Within this stepped care model individuals received “low” (LiCBT) or “high” intensity cognitive behavioural therapy (HiCBT), counselling, or group treatment.

Results: 19% (*n* = 1836) experienced a sudden gain and 24% (*n* = 2265) experienced a depression spike. Rates of discontinuous change were highest in HiCBT compared to other treatments. Both patterns of discontinuous changes were associated with improved depression, anxiety, and functioning at treatment end. Treatment type did not moderate these relationships. Individuals with higher baseline depression, anxiety, and functioning severity benefitted the most from experiencing discontinuous change across all treatments.

Conclusion: Rates of discontinuous change varies between treatments, but both sudden gains and depression spikes are associated with beneficial treatment outcome across therapies. This suggests that discontinuous change may be more likely to occur in some therapies compared to others and further research is needed to understand what brings about discontinuous change. Replication and examination over longer follow-up periods is needed.

Keywords: depression, sudden gains, depression spikes, Improving Access to Psychological Therapies (IAPT)

2.3 Introduction

Depression is a highly debilitating disorder that is an international public health concern (World Health Organisation, 2017). Meta-analyses of trials demonstrate there are effective treatments for depression (Cuijpers et al., 2014; Cuijpers, Sijbrandij, et al., 2013) but less is known about how these treatments lead to improvement. Trajectory research often shows that symptom change in therapy is non-linear and patterns of discontinuous change are common, robust (Shalom & Aderka, 2020), and distinct from other patterns of symptom change (Andrews et al., 2020). Within psychotherapy research rapid, discontinuous fluctuations in depressive symptoms have been found to occur across therapies and are associated with both better and worse treatment outcomes (Hayes, Feldman, Beevers, et al., 2007; O'Mahen et al., 2021; Shalom & Aderka, 2020; Tang & DeRubeis, 1999). However, there have been few studies that have directly compared rates and outcomes of non-linear change between different types of treatments.

Non-linear patterns of change in depression treatment have been theorised to represent moments of destabilisation in the depressive network (Hayes & Strauss, 1998), which may represent changes in existing patterns of behaviours and thinking (Andrews et al., 2020). Further examining these patterns of change may elucidate points during psychotherapy associated with better or worse outcomes. To date, the majority of the evidence for the effects of symptom discontinuities has been from trial datasets. Although individuals who receive evidence-based treatments in clinical practice have similar outcomes to those in trials (Cuijpers et al., 2009), it is unclear if regular clinical practice is associated with the same rates of discontinuous change, and if these changes are related to similar outcomes as in trial data. This is important as there are concerns that individuals seen in regular clinical practice may differ from

those in trials. Further, the way that treatments are implemented and supervised in clinical practice may be less rigorous or intensive than trials (Castonguay, Barkham, Lutz, & McAleavey, 2013). Existing studies of discontinuous change are typically conducted with smaller sample sizes and fewer therapists, and are usually examined within a single type of treatment (Kelly, Cyranowski, et al., 2007; Kelly et al., 2005; Masterson et al., 2014; O'Mahen et al., 2017; O'Mahen et al., 2019; Tang et al., 2005) (for exceptions see Lemmens et al., 2016; O'Mahen et al., 2021). Therefore, research examining different types of discontinuous change in treatments provided to a broad clinical sample of individuals is needed.

Perhaps the most widely studied pattern of discontinuous change in depression therapy research is a 'sudden gain' (Tang & DeRubeis, 1999), defined as a rapid improvement of depressive symptoms in a single-session interval, that is large in magnitude and relative to previous depression scores prior to the drop in symptoms. In their seminal study Tang and DeRubeis (1999) observed 39% of individuals experienced a sudden gain in cognitive behavioural therapy (CBT) and sudden gains were associated with better depression treatment outcomes, compared to those who did not experience a sudden gain. Subsequently sudden gains have been found to occur in 40% (range = 25.7-53.8%) of individuals in treatment for depression in RCT studies (Shalom & Aderka, 2020) and are associated with better outcomes across a range of different treatments, including CBT (Abel et al., 2016; Tang et al., 2005), behavioural activation (BA) (Masterson et al., 2014) and group therapies (O'Mahen et al., 2019). In contrast, in regular clinical practice sudden gains occur in 32.7% (range = 9- 42%) of individuals (Koffmann, 2019; Lutz et al., 2013; Stiles et al., 2003; Wucherpfennig, Rubel, Hofmann, et al., 2017). There is also some variability in the effects of sudden gains in clinical settings

on treatment outcomes with some studies finding positive associations between sudden gains and later symptoms (Hardy et al., 2005; Wucherpfennig, Rubel, Hollon, et al., 2017) and others failing to do so (Stiles et al., 2003). It is unclear whether the differences in these effects are due to the ways in which treatment was administered. Within RCT studies there is strong adherence to treatment protocols and there are stringent inclusion and exclusion criteria. However, in routine clinical settings treatments may not be applied in a highly adherent fashion (Koffmann, 2019; Stiles et al., 2003) and may therefore not be standardised. Treatment standardisation may be an important factor associated with the effects of sudden gains on outcomes, particularly when attempting to compare patterns of sudden gains across different types of treatments. Clinical practices that emphasise the adherent application of treatment principles improve treatment standardisation and the ability to compare treatments.

There is also a lack of research investigating the effects of depression sudden gains on other outcomes, particularly those that have high rates of co-morbidity with depression, like anxiety (Lamers et al., 2011) and work and social functioning (Rizvi et al., 2015; Woodhead et al., 2020). The majority of research examines the association of depression sudden gains on depression outcomes, although a recent meta-analysis suggests that depression sudden gains may have small (Hedges's g = 0.38) effects on other, secondary measures. However studies used in this meta-analysis sometimes included depression as a secondary outcome and sample sizes tended to be small, reducing confidence in these effect size estimations.

In comparison to the sudden gains literature fewer studies have examined depression spikes, which are characterised by a rapid worsening in symptoms that subsequently improves (Hayes, Feldman, Beevers, et al., 2007) (see Lutz et al.,

2013 for a rapid worsening of symptoms that does not improve). Depression spikes were first identified in an exposure-based cognitive therapy (EBCT) for depression. EBCT was developed to disrupt the depressive network in-order to process and embed more adaptive thoughts and behaviours, by approaching previously avoided distressing content (Hayes et al., 2007). During the middle of treatment, where this opportunity for processing occurred, 62% of individuals experienced a depression spike which were associated with lower depression scores at the end of treatment (Hayes, Feldman, Beevers, et al., 2007). However, depression spikes have also been identified in non-exposure therapies where there are not intended therapeutic procedures which deliberately bring about a depression spike in treatment. Depression spikes may therefore occur at different rates and may not have the same relationship with treatment outcome. In support of this, previous studies have found that depression spikes in non-exposure based CBT occurred in fewer individuals (26-50%; Abel, 2014; O'Mahen et al., 2021) than in Hayes et al.'s (2007) seminal depression spikes study, but they have also been found to predict worse depression outcomes at 6-, 12- and 18-month follow-up (O'Mahen et al., 2021). Conversely in a trial of CBT adjunctive to pharmacotherapy in a treatment resistant sample depression spikes were unrelated to 12-month outcome (Abel, 2014). Other, smaller studies examining depression spikes outside of CBT have identified too few depression spikes to assess associations with treatment outcomes (10-19%; O'Mahen et al., 2017; O'Mahen et al., 2019). There is a need to understand the frequency of depression spikes outside EBCT, and how depression spikes influence end of treatment depression and other outcomes, like anxiety and funcitioning, between treatments in a large natuaralistic sample.

It is also possible that differences observed in both the rates and association of discontinuous change and treatment outcomes lies with the intensity with which the treatment is delivered. In England psychological care for common mental health problems in Improving Access to Psychological Therapies (IAPT, Clark, 2011) is organised according to a stepped care model. Some individuals will receive group psychoeducation or guided self-help forms of treatment known as “low-intensity” treatments, whilst others will receive more intensive forms of treatment (“high-intensity”) either on their own or following low-intensity treatment. There is at least some research showing that there are similar rates and effects of sudden gains in trials of group (42%; Kelly et al. 2005) and guided internet-based self-help interventions for depression (51%, O’Mahen et al. 2017) to those in high-intensity face-to-face treatments. Some research in this area suggest that early improvements in low-intensity treatments for depression are associated with small (OR = 1.33; Tadić et al., 2010) to large (OR = 12.60; Delgadillo et al., 2014) effects on outcomes. However, there has been very little research examining rates and effects of sudden gains, and none of depression spikes on treatment outcomes, across low- and high-intensity treatments in regular clinical practice. Further research that directly compares sudden gains and depression spikes across low- and high-intensity treatments may provide some support for whether intensity of treatment makes a difference.

Also deserving attention is the exploration of whether individual client characteristics influence the way sudden gains or depression spikes relate to treatment outcomes in clinic settings. Patient demographic and clinical characteristic differences have been shown to influence treatment outcomes (Barber, 2007; Delgadillo et al., 2016), but it is unclear whether these may also impact on the ability

to capitalise upon discontinuous changes in therapy and effect treatment outcomes. Examining baseline characteristics and the influence of sudden gains on treatment outcomes in an outpatient sample, Wucherpfennig, Rubel, Hollon, et al. (2017) used a range of propensity score approaches to match the baseline characteristics of the clinical sample with the characteristics of those in the RCT sample in Tang and DeRubeis (1999) seminal sudden gains study. They found that as the propensity scoring approach brought the clinical sample closer to the trial sample, so did the effects of sudden gains more closely match those in the original trial. This suggests that sudden gains may not be as powerful in producing positive outcomes in a broader range of individuals suffering from depression, and that patients with specific characteristics may be more likely to sustain and utilise a sudden gain. In recent meta-analysis of sudden gains in both RCT and clinical settings in a range of diagnostic mental health problems, Shalom and Aderka (2020) found greater number of therapy sessions moderated the association between a sudden gain and post-treatment outcomes, but other factors such as treatment modality (CBT vs non-CBT treatments), being female, age, or pre-treatment severity levels did not. Additionally, no research has examined moderators of depression spikes in non-exposure based therapies. In clinic-based samples where there is greater diversity amongst patients, and often shorter treatment duration (e.g. 6.9-7.6 sessions; National Health Service (NHS) digital, 2020) due to prompt discharge practices it may be particularly important to examine additional patient-level variables such as socioeconomic factors and number of sessions attended as moderators of discontinuous change and treatment outcomes.

2.3.1 The Current Study and Hypotheses

The current study aimed to examine i) the rates and timings of sudden gains and depression spikes in a large primary care, clinic-based sample who had depression as their primary presenting problem and received either low- (group or low-intensity CBT (LiCBT)) or high- (counselling or high-intensity CBT (HiCBT)) intensity treatments, ii) whether these patterns of change were associated with improved depression (primary outcome), anxiety and functioning at the end of treatment, and iii) if baseline clinical characteristics and treatment variables moderated the relationship between symptom discontinuities and treatment outcomes.

Rates and timing of discontinuous change between treatments. Consistent with previous research (e.g. Tang & DeRubeis, 1999; Tang et al., 2005) we expected sudden gains would occur early in treatment (Shalom & Aderka, 2020). Comparisons between rates of sudden gains in low- and high-intensity treatments were treated as exploratory. In contrast, we expected more depression spikes to occur during the middle of high-, rather than low-intensity treatment. This is because Hayes et al. (2007) found depression spikes that were associated with treatment outcome were more likely to occur during the middle of treatment, where distressing, previously avoided content was approached in an intensive fashion. We note that this may be more so in HiCBT which is based on standard CBT treatment and is guided by a therapist, rather than LiCBT which utilises guided self-help materials, with limited intensive exploration of content with a practitioner (Shafran et al., 2021).

Outcomes. In keeping with previous literature, we hypothesised that individuals with sudden gains would have a greater reduction in depression, anxiety, and work and social adjustment (functioning) symptoms and higher rates of reliable

improvement, clinically significant change (CSC), and reliable recovery in depressive, anxiety, and functioning symptoms, compared to those without a sudden gain. Similarly, we expected individuals with depression spikes would have greater reductions in depression symptoms and higher rates of reliable improvement, CSC, and reliable recovery in depressive symptoms. We explored the association between depression spikes and anxiety and functioning treatment outcomes as this has not been previously examined.

Moderators. We explored whether treatment type moderated the relationship between sudden gains and depression spikes and depression, anxiety, and functioning outcome. Further, we explored whether baseline depression, anxiety and functioning, number of treatment sessions, and socio-economic level moderated the relationship between sudden gain or depression spike status and depression, anxiety and functioning outcomes at the end of treatment.

2.4 Method

2.4.1 Data Source: Improving Access to Psychological Therapies (IAPT)

This retrospective analysis used anonymised data collected between 2008 and 2011 as part of the IAPT Evaluation Project in the South-West of England (Byng et al., 2011) across 14 primary care trusts, which were NHS administrative bodies responsible for primary, community and secondary care health services until 2013. The service gathers weekly assessments of each client's mood, symptoms and functioning as part of standard practice. The therapies offered for depression were LiCBT, group therapy, HiCBT, and counselling. In IAPT's step-care model, LiCBT and group therapy are delivered by Psychological Wellbeing Practitioners (PWP) who have completed a one-year training course. LiCBT employs guided self-help

CBT materials (Shafran et al., 2021), whereas group therapy utilises strategies from CBT and psychoeducation materials about depression. Individuals complete six-eight sessions of low-intensity treatments with up to 30 minutes of weekly contact with a PWP to support them with the materials. These interventions are typically the first offered in IAPT.

High-intensity interventions (counselling and HiCBT) are often provided to individuals who do not respond to low-intensity treatments and who can be “stepped up” to higher-intensity treatment. In some cases individuals may directly go to high-intensity treatment without first having received low-intensity treatment. HiCBT is provided by therapists who have completed at least two years of high-intensity therapy training and consists of standard CBT. Individual HiCBT sessions are 60 minutes in duration and between 12-20 sessions long. Counselling is provided by trained counsellors and sessions are typically 60 minutes long for between six and twelve sessions. The aim of counselling is to help the client understand themselves better to find solutions and coping strategies to cope with their problems. The role of the counsellor is to take an impartial but understanding role and listen, empathise but also challenge thinking, behaviour, or emotions to enable to client to see their issues in a different way (BACP, 2021).

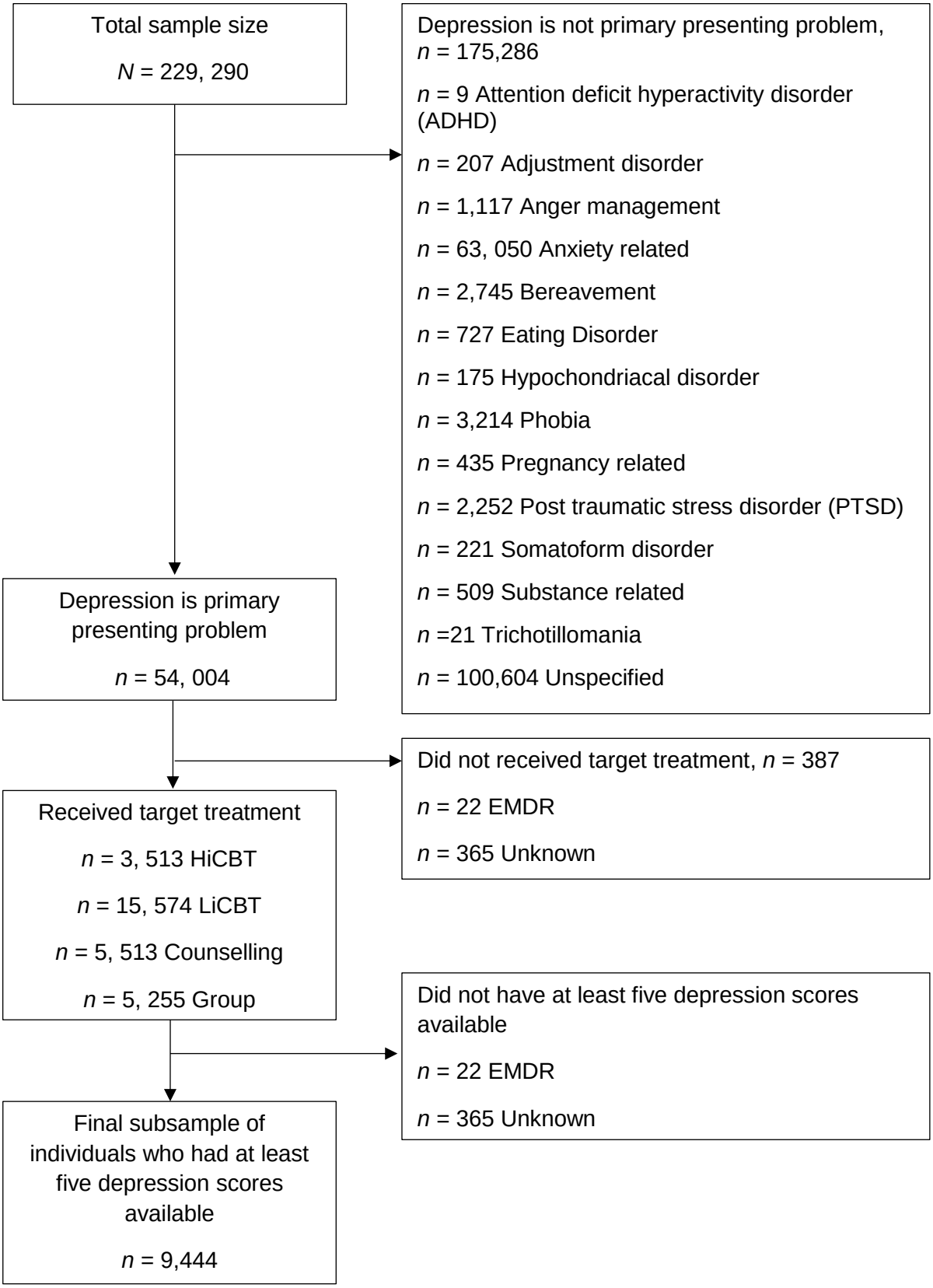
Ethical approval for this secondary analysis was received from the local University departmental ethics committee and through addition of an amendment to the National Research Ethics Service (NRES) approval for the original IAPT evaluation project (Ref: 09/H0203/91).

2.4.2 Sample

The original dataset contained data from 229,290 individuals. Figure 2.1 gives an overview of the sample used in the current analysis. Individuals were included in the current analysis if they had a primary diagnosis of depression and had at least five sessions of Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) depression scores available, the minimum needed to identify a sudden gain or depression spike and to have the last depression score separate from the sudden gain or depression spike identification. This resulted in a sample of 9,444 individuals seen by 3,512 therapists.

Figure 2. 1

STROBE Diagram of the Sample Used in the Current Study



A sensitivity power calculation in G*Power indicated that a sample size of 9,444 participants, with 95% power, an alpha of 0.05 and 17 predictors (all main effects and interactions) would allow us to detect a small effect ($f^2 = 0.0013$).

2.4.3 Measures

2.4.3.1 Depression. The Patient Health Questionnaire (PHQ-9; Kroenke et al., 2001) is a nine item self-report measure of depression symptoms over the previous two weeks. Items are scored on a 0-4 Likert scale where higher scores indicate greater depression severity. The PHQ-9 is a valid and reliable measure (Kroenke et al., 2001) and is sensitive to depression change over time (Löwe et al., 2004). The PHQ-9 was measured at every session and was used to identify sudden gains and depression spikes. Each individual's final PHQ-9 score was their outcome depression score.

2.4.3.2 **Anxiety.** The Generalised Anxiety Disorder (GAD-7; Spitzer et al., 2006) questionnaire is a reliable, seven item self-report assessment of worry and general anxiety over the last two weeks. Items are scored on a 0-3 Likert Scale where higher scores indicate severe anxiety. The GAD-7 is reliable and valid measure making it the most widely used measure of anxiety in both clinical practice and research (Dear et al., 2011). The GAD-7 is also sensitive to anxiety change over the course of treatment, even in populations with comorbid depression (Toussaint et al., 2020). In the current study individuals' outcome GAD-7 score was their final session anxiety score.

2.4.3.3 Functioning. The Work and Social Adjustment Scale (WSAS; Mundt et al., 2002) is a five item self-report scale measuring how an individual's problem impacts on their perceived daily functioning. The scale covers work, home

management, social and private leisure activities, and family relationships, measured on a 0-8 Likert scale where lower scores indicate better functioning. The WSAS has good psychometric properties and high internal reliability in an IAPT sample (Zahra et al., 2014).

2.4.3.4 **Moderators.** The moderating variables included treatment modality (counselling, group treatment, LiCBT or HiCBT), number of treatment sessions, baseline PHQ-9, GAD-7 and WSAS score. Additionally, the index of multiple deprivation (IMD) score, a measure of the relative deprivation in areas of England was measured at baseline. The IMD total score is a composite variable which consists of seven domains; deprivation, health, education level, crime, employment, income, and quality of housing and living environment (Payne & Abel, 2012). Scores range between 0 and 100, with higher scores indicating greater deprivation.

2.4.4 Procedure

2.4.4.1 Identification of Sudden Gains. Sudden gains are large improvements in depression symptoms defined as being (1) a decrease in at least seven Beck Depression Inventory (BDI; Beck et al., 1961) points between two sessions of therapy, (2) which is at least 25% of the magnitude of the pre-gain (session immediately prior to the sudden drop in depression scores) BDI score, and (3) where the mean of the three scores preceding the gain are significantly higher than the three scores after the sudden gain, using an independent samples *t*-test (Tang & DeRubeis, 1999). In line with research which has used the PHQ-9 to identify sudden gains (Masterson et al., 2014; Singla et al., 2019) the PHQ-9's reliable change index of ≥ 5 (McMillan et al., 2010) was used for the first sudden gains criterion. Tang and DeRubeis' second sudden gains criterion was retained, but the current study used a modified third criterion. This criterion still uses the *t*-distribution

approach, but modifies the *t* values to allow for the identification of very early and very late (occurring in the second or penultimate therapy session) in treatment (O'Mahen et al., 2021; Wucherpfennig, Rubel, Hofmann, et al., 2017). For normal sudden gains with three sessions prior to and following the drop in symptoms Tang and DeRubeis' criteria of $t(4) \geq 2.78$ was retained, for two available sessions either side of the gain, $t(3) \geq 3.18$ was used, and for one session before and after the gain, $t(2) \geq 4.30$ was used. Individuals who 'lost' 50% or more of their symptom improvement are defined as having a reversal of the sudden gain (Tang & DeRubeis, 1999).

2.4.4.2 Identification of Depression Spikes. Depression spikes were originally defined by Hayes, Feldman, Beevers, et al. (2007) as an increase of seven depression points or more between two consecutive sessions of therapy, which returns (spike recovery) by seven or more depression points within the same phase of therapy in EBCT. As with the sudden gain criteria a ≥5 PHQ-9 depression point difference was used. As treatment phases are arbitrary and treatments in IAPT do not have defined phases, O'Mahen et al. (personal communications) assessed the average number of sessions it took for a depression spike to recover in a trial dataset of non-exposure CBT and BA for depression and observed the average number of sessions it took for a depression to return was three sessions ($M = 2.52$, $SD = 2.11$). A comparison of this modified depression spike criterion and depression spikes identified using Hayes et al.'s original criterion yielded the same number of depression spikes identified (O'Mahen et al., personal communications). Therefore, in the current study we identified depression spikes as occurring if there was an increase of five or more PHQ-9 depression points between two consecutive sessions

that returned by five or more PHQ-9 depression points within three treatment sessions.

It is important to note a depression spike, a transient worsening in depression that subsequently drops, is different to a 'sudden loss' (Lutz et al., 2013) which by definition is a symptom worsening that does not drop.

2.4.5 Outcome Measures

We assessed the association of sudden gain or depression spike status on dimensional PHQ-9, GAD-7 and WSAS outcomes, reliable change, clinically significant change (CSC) and reliable recovery. Standard IAPT reporting examines reliable change (improved or deteriorated) and reliable recovery. Individuals have reliably improved/deteriorated if their PHQ-9, GAD-7 or WSAS scores have reduced/increased (respectively) more than the measurement error of the scale (PHQ-9 ≥ 5; GAD-7 ≥ 4; WSAS minimally clinically significant change of 8 (Zahra et al., 2014)). Individuals are deemed to have reliably recovered in IAPT if they score above the clinical cut off on the PHQ-9 (≥ 10) and/or the GAD-7 (≥8) during the assessment session, they show reliable improvement during treatment and at the end of treatment they score below the clinical cut off on *both* the PHQ-9 and GAD-7.

Clinically significant change (CSC) is defined as improvement from clinical to non-clinical ranges of symptoms. Jacobson and Truax (1991) provided three criterions to measure CSC. For the current study CSC on the PHQ-9 was assessed using criterion c (for a comparison of the different criterions on the PHQ-9 see McMillan et al., 2010), which uses data from the clinical and non-clinical populations and requires individuals to be above the clinical cut off prior to treatment and below subsequent to treatment (Jacobson & Truax, 1991). The pre-treatment mean (16.3)

and standard deviation (6.1) from the current sample (n = 9,444) was used to assess PHQ-9 CSC. The non-clinical mean (3.3) and standard deviation (3.8), as well as the internal reliability estimate for the PHQ-9 (α = 0.89) was used from the original validation study of the PHQ-9 by Kroenke et al. (2001). To examine GAD-7 CSC the pre-treatment mean from the current sample was 13.07 and standard deviation was 5.26. The non-clinical mean (4.9) and standard deviation (4.8), as well as the reliability (α = 0.92) were derived from Spitzer et al.'s (2006) validation study of the GAD-7. As there are little comparisons of the WSAS in clinical and non-clinical samples, the minimally clinically significant change of 8 points was used (Zahra et al., 2014). Reliable change and CSC were calculated using the Leeds Reliable Change calculator (Morley & Dowzer, 2014).

2.4.6 Analytical Strategy

Analyses were performed using IBM SPSS Statistics version 25 (IBM Corp, 2017) and R version 3.6.0 (R Core Team, 2016). Sudden gains were identified using the 'suddengains' R package (Wiedemann et al., 2020) which automates the detection of sudden gains. Cases were selected using "pattern", which selects the minimum number of available data (Wiedemann et al., 2020). Where individuals had multiple sudden gains the earliest sudden gain was used in the analysis. Depression spikes were identified using code in R. Where there were multiple depression spikes the spike closest to the middle of treatment was used in the subsequent analyses. Missing data was not imputed to be comparable to sudden gains and depression spikes in the wider literature, but also to ensure gains or spikes were not falsely detected due to imputed depression scores.

To compare baseline clinical and demographic characteristics between treatments Chi-Square and Analysis of Variance (ANOVA) analyses were

conducted. To assess the rates of the discontinuities between treatments and the timing of sudden gains and depression spikes Chi-Square tests were used.

Separate hierarchical multiple linear regression models were conducted to assess the association of sudden gain (0,1) or depression spike (0,1) status on PHQ-9, GAD-7 and WSAS continuous outcomes[3], and whether baseline PHQ-9, GAD-7, WSAS, IMD score, number of treatment sessions, and treatment type (counselling, group, LiCBT and HiCBT) moderated these relationships. Each of the moderator variables were entered into the first step. Treatment type was dummy coded and HiCBT was used as the reference group because this is considered the gold standard of depression treatment (David et al., 2018) and is where most of the literature has examined sudden gains. Sudden gain (0, 1) or depression spike (0, 1) status was entered in step two. In the final step the two-way interactions between sudden gain or depression spike status and the moderators were entered. The dependent variables were either outcome PHQ-9, GAD-7 or WSAS continuous scores. Significant two-way interactions were explicated to understand how low and high levels of the moderator influenced sudden gain or depression spike status on continuous treatment outcomes.

To assess how sudden gain or depression spike status was related to reliable change (improvement, deterioration, no change) a series of multinomial logistic regression analyses were conducted. In order to be consistent with the other models,

[3] Out of the 1836 who experienced a sudden gain, 271 individuals had their last session PHQ-9 score as part of the sudden gain identification. Out of the 2265 who experienced a depression spike 406 had their last PHQ-9 score as part of the depression spike identification. Therefore the outcome analyses include only the 1565 who had a sudden gain and 1859 who had a depression spike and their last session score was not part of the gain or spike identification.

sudden gain (0,1) or depression spike (0,1) status, baseline PHQ-9, GAD-7, WSAS, IMD score, number of treatment sessions, and dummy coded treatment variables were entered as independent variables, with HICBT being the reference group. Either PHQ-9, GAD-7 or WSAS reliable change (improve (1), deteriorate (2) or no change (3)) was entered as the dependent variable. 'Improve' was the reference category to see if individuals were more or less likely to deteriorate or experience no change compared to improvement on depression, anxiety and functioning reliable change.

Separate binary logistic regression analyses were used to assess the association between sudden gain or depression spike status on CSC (0, 1) or reliable recovery (0, 1) outcome. In all logistic regression models, baseline PHQ-9, GAD-7, WSAS scores, IMD score, number of treatment sessions, and dummy coded treatment variables were entered as independent variables, with HICBT being the reference group. Next sudden gain (0, 1) or depression spike status (0, 1) was entered in step two. In the final step the two-way interactions between sudden gain or depression spike status and the moderating variables were entered. The dependent variable was either whether individuals had CSC (0, 1) or experienced reliable recovery (0, 1).

2.5 Results

2.5.1 Participants

A comparison of the baseline demographic and clinical characteristics of the sample across the therapies (Table 2.1) showed there was a greater number of females compared to males in all the therapies. Most individuals in the sample were Caucasian. Individuals in HiCBT had significantly more treatment sessions than

those in both low-intensity interventions (group, LiCBT) and counselling. Those in LiCBT had more sessions than individuals in group treatment. Baseline depression, anxiety and functioning were lowest in individuals receiving counselling or group treatment. Individuals in LiCBT had intermediary levels of baseline depression, anxiety and functioning symptomatology, and those in HiCBT had the highest levels of baseline depression and anxiety symptoms and poorest functioning. Additionally, individuals receiving group therapy had significantly less baseline deprivation scores than individuals in the other treatments. There were no other baseline differences between treatment conditions.

Table 2. 1

Descriptive Statistics of the Current Sample (n = 9444)

	LiCBT (*n* = 4604; 48.8%)		Group (*n* = 1293; 13.7%)		HiCBT (*n* = 1865; 19.7%)		Counselling (*n* = 1682; 17.8%)						
	n(%)	*M*(*SD*)	*n*(%)	*M*(*SD*)	*n*(%)	*M*(*SD*)	*n*(%)	M(*SD*)	χ^2	*df*	*F*	*p*	ηp2
Sex									14.580	3		.002**	
Male	1528		398		630		481						
	(33.2%)		(30.8%)		(33.8%)		(28.6%)						
Female	3075		882		1232		1196						
	(66.8%)		(68.2%)		(66.1%)		(71.1%)						
Unknown	1		13		3		5						
	(<.1%)		(1%)		(0.2%)		(0.3%)						
Ethnicity									66.692	3		<.001***	
Caucasian	3766		1146		1475		1451						
	(81.8%)		(88.6%)		(79.1%)		(86.3%)						
Other	838		147		390		231						
	(18.2%)		(11.4%)		(20.9%)		(13.7%)						

Number of treatment sessions	8.53 (4.23)	8.20 (4.30)	10.74 (5.08)	8.32 (3.66)	3, 9440	144.81	<.001***	.044
					Counselling = Group <LiCBT < HiCBT			
Baseline PHQ-9	16.28 (5.89)	15.31 (6.12)	17.44 (5.77)	15.65 (6.64)	3, 9440	40.252	<.001***	.013
					Counselling = group < LiCBT < HiCBT			
Baseline GAD-7	13.03 (5.12)	12.39 (5.49)	14.00 (4.98)	12.70 (5.58)	3, 9439	29.939	<.001***	.009
					Counselling = Group < LiCBT< HiCBT			
Baseline WSAS	19.77 (8.84)	18.63 (8.72)	21.43 (9.16)	17.36 (9.50)	3, 9432	65.488	<.001***	.020
					Counselling < Group< LiCBT < HiCBT			

Baseline	19.56	15.43	20.04	19.48	3, 8980	52.045	<.001***	.017
IMD	(11.05)	(11.26)	(11.52)	(10.78				
					Group<	LiCBT, HiCBT, Counselling		

Note. HiCBT = High-intensity cognitive behavioural therapy; LiCBT = Low-intensity cognitive behavioural therapy; < significantly less than; PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; IMD = Index of Multiple Deprivation

2.5.2 Rates and Timings of Discontinuous Change between Treatments

2.5.2.1 Sudden Gains.

Across all the treatments a total of 1836/9444 (19.44%) individuals experienced a sudden gain. Of these 1585/1836 (86.3%) had one, 248 (13.5%) had two, and three (0.2%) individuals experienced three sudden gains. Of the 1836 sudden gains, 396 (22%) experienced a reversal of a sudden gain. Consistent with expectations, sudden gains were more likely to occur early (62.9%; n = 1154/1836) rather than midway (21.9%, n = 403/1836), χ^2 (1) = 40.39, p = <.001, or in late treatment (15.2%, n = 279/1836), χ^2 (1) = 6.20, p = .012. There was no significant difference in the number of sudden gains occurring in the middle compared to late treatment, χ^2 (1) = 0.63, p = .663.

There were significant differences between rates of sudden gains in low- and high-intensity treatments, χ^2 (1) = 13.89, p = <.001. Individuals in low-intensity treatments (LiCBT, Group) had significantly fewer sudden gains (n = 1077/5897; 18.3%) than those in high-intensity (HiCBT, counselling) treatments (n = 759/3547; 21.4%). In post-hoc analyses we compared whether there were differences in rates of sudden gains within the two different types of treatment intensities. Within the low-intensity treatments significantly more individuals had a sudden gain in LiCBT (n = 911/4604; 19.8%) than group treatment (n = 166/1293; 12.8%), χ^2 (1) = 515.34, p <.001. Within the high-intensity treatments individuals in HiCBT (n = 441/1865; 23.6%) had significantly more sudden gains than those in counselling (n = 318/1682; 18.9%), χ^2 (1) = 19.93, p <.001.

2.5.2.2 Depression Spikes.

Across all treatments 2265/9444 (24%) experienced a depression spike. Of these 1862/2265 (82.2%) had a single depression spike, 326 (14.4%) had two, 65 (2.9%) had three, 11 (0.4%) had four and 1 (<1%) had five depression spikes. Contrary to expectations, spikes were more likely to occur in the early stages of treatment (*n* = 1291/2265; 57%) than in the middle (*n* = 429/2265; 19%), χ^2 (1) = 138.91, *p* = <.001; or late stage (*n* = 545/2265; 24%), χ^2 (1) = 15.63, *p* = <.001; and in the late than middle stage of treatment, χ^2 (1) = 6.44, *p* = .016.

There were also different rates of depression spikes between high- and low-intensity treatments, χ^2 (1) = 29.05, *p* = <.001. Consistent with predictions, those in high- (*n* = 959/3547; 27%) rather than low-intensity (*n* = 1306/5897; 22.1 %) treatments were more likely to experience a depression spike. Post-hoc analyses revealed that within the low-intensity treatments there was no significant difference in rates of depression spikes between LiCBT (*n* = 1020/4604; 22.2%) and group treatment (*n* = 286/1293; 22.1%), χ^2 (1) = 0.001, *p* = .978. In the high-intensity treatments individuals in HiCBT (*n* = 543/1865; 29.1%) were more likely to have depression spikes than those in counselling (*n* = 416/1682; 24.7 %), χ^2 (1) = 8.61, *p* = .004.

2.5.3 Sudden Gain and Depression Spikes Association with Depression, Anxiety and Functioning Outcomes

2.5.3.1 Depression Outcomes.

Consistent with expectations, having a sudden gain (*M* = 6.72, *SD* = 5.50) was associated with significantly lower end of treatment PHQ-9 scores than those who did not have a sudden gain (*M* = 9.69, *SD* = 7.03) (Table 2.2). The effect size

for the association of sudden gains on depression outcome was Hedges's *g* = 0.44 indicating a medium effect. Individuals who experienced a sudden gain, compared to no gain, were significantly more likely to experience reliable improvement than deterioration (OR = 5.88) or no change (OR = 3.70) in PHQ-9 depression scores (Table 2.3), and were three times as likely (OR = 3.02) to have clinically significant change (CSC; moving from clinical to non-clinical range of symptoms) in PHQ-9 depression scores (Table 2.4).

In line with hypotheses, experiencing a depression spike (*M* = 8.64, *SD* = 6.48) was associated with significantly lower end of treatment PHQ-9 scores, than those who did not have a depression spike (*M* = 9.33, *SD* = 6.99) (Table 2.5). This was a small effect, Hedges's *g* = 0.10. However, the picture regarding clinical improvement was more complex. Although individuals who had a depression spike, compared to no spike, were more likely to improve (OR= 1.25) than experience no change, they were also more likely to have deterioration (OR= 1.46) in PHQ-9 scores than improve (Table 2.6). Individuals who experienced a depression spike, compared to no depression spike, were more likely to experience CSC in PHQ-9 scores (OR = 1.16; Table 2.4).

Table 2. 2

Hierarchical Regression Results for Sudden Gain Status on PHQ-9 Depression Outcome, GAD-7 Anxiety Outcome and WSAS Functioning Outcome

	PHQ-9 Outcome			GAD-7 Outcome			WSAS Outcome		
	B(*Se*)	95% CI	R^2 , Δ R^2	*B*(*Se*)	95% CI	R^2 , Δ R^2	*B*(*Se*)	95% CI	R^2 , Δ R^2
Step 1			.16, .16***			.16, .16***			.18, .18***
Constant	0.42 (0.32)	-0.20, 1.04		0.38 (0.27)	-0.15, 090		1.224**	0.36, 2.09	
Baseline PHQ-9	0.32(0.02) ***	0.29, 0.35		0.11(0.01)***	0.09, 0.14		0.21***	0.17, 0.26	
Baseline GAD-7	0.07(0.02) ***	0.03, 0.10		0.28(0.01)***	0.25, 0.30		-0.02	-0.07, 0.03	
Baseline WSAS	0.09(0.01) ***	0.07, 0.10		0.06(0.01)***	0.04, 0.07		0.35***	0.32, 0.37	
Number of treatment sessions	0.05(0.02) **	0.02, 0.08		0.03(0.10)*	0.001, 0.05		0.10***	0.05, 0.14	
IMD score	0.03(0.01) ***	0.02, 0.04		0.03(0.01)***	0.02, 0.04		0.04***	0.02, 0.05	
Counselling vs HiCBT	-0.14(0.22)	-0.58, 0.30		-0.07(0.19)	-0.44. 0.29		-1.02**	-1.63, -0.41	
Group vs HiCBT	0.82(0.24) **	0.35, 1.29		0.59(0.20)**	0.19, 0.99		1.80***	1.14, 2.46	

LiCBT vs HiCBT	-0.15(0.18)	-0.51, 0.20		-0.29(0.15)	-0.59, 0.01		-0.68**	-1.18, -0.18	
Step 2			.26, .05***			.20, .04***			.21, .03***
Sudden Gain (Ref group No Sudden Gain)	-4.04(0.18)***	-4.39, -3.69		-3.17(0.15)***	-3.47, -2.88		-4.52***	-5.01, -4.02	
Step 3			.26, .01***			.21, .004***			.22, .003***
Baseline PHQ-9 x Sudden Gain	-0.24(0.05)***	-0.33, -0.15		-0.12(0.04)**	-0.19, -0.40		-0.24***	-0.36, -0.11	
Baseline GAD-7 x Sudden Gain	0.01(0.05)	-0.08, 0.10		-0.08(0.04)*	-0.16, -0.01		0.04	-0.09, 0.16	
Baseline WSAS x Sudden Gain	-0.01(0.02)	-0.05, 0.04		-0.01(0.02)	-0.05, 0.03		-0.06	-0.012, 0.01	
Number of treatment sessions x Sudden Gain	0.06(0.04)	-0.01, 0.14		0.03(0.03)	-0.03, 0.10		0.06	-0.04, 0.17	
IMD x Sudden Gain	-0.03(0.02)	-0.06 ,0.01		-0.01(0.01)	-0.04, 0.02		-0.01	-0.06, 0.03	

Counselling vs HiCBT x Sudden Gain	0.01(0.57)	-1.11, 1.12	-0.12(0.48)	-1.06, 0.82	1.33	-0.23, 2.90
Group vs HiCBT x Sudden Gain	0.04(0.67)	-0.38, 2.26	0.52(0.57)	-0.60, 1.65	0.97	-0.90, 2.83
LiCBT vs HiCBT x Sudden Gain	0.56(0.45)	-0.31, 1.44	0.19(0.38)	-0.56, 0.93	0.70	-0.53, 1.94

Note. CI = confidence interval; PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; IMD = Index of Multiple Deprivation; Ref group = reference group.

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

Table 2. 3

Logistic Regression Results of Sudden Gain and Treatment on PHQ-9, GAD-7 and WSAS Reliable Clinical Change

	PHQ-9 Reliable Change Outcome		GAD-7 Reliable Change Outcome		WSAS Reliable Change Outcome	
Sudden Gains	OR	95% CI	OR	95% CI	OR	95% CI
Deteriorate vs improvement						
Baseline PHQ-9	0.748***	0.72, 0.77	1.11***	1.08, 1.14	1.11***	1.09, 1.15
Baseline GAD-7	1.041**	1.01, 1.07	0.69***	0.67, 0.71	1.01	0.97, 1.04
Baseline WSAS	1.043***	1.03, 1.06	1.02*	1.00, 1.03	0.77***	0.76, 0.79
Number of treatment sessions	1.050**	1.02, 1.08	1.04**	1.01, 1.06	1.07***	1.04, 1.09
IMD score	1.014*	1.00, 1.03	1.01**	1.00, 1.02	1.02**	1.01, 1.03
Counselling vs HiCBT	1.309	0.86, 1.99	1.21	0.91, 1.86	1.40	0.92, 2.13
Group vs HiCBT	0.90	0.59, 1.37	0.99	0.69, 1.44	0.47***	0.31, 0.72
LiCBT vs HiCBT	1.36	0.97, 1.92	1.50**	1.12, 2.01	1.15	0.81, 1.64
Sudden Gain (Ref group no sudden gain)	0.17***	0.09, 0.29	0.16***	0.10, 0.25	0.12***	0.07, 0.19
Sudden Gain* Baseline PHQ-9	0.86	0.73, 1.01	0.93	0.84, 1.03	0.91	0.81, 1.02

Sudden Gain* Baseline GAD-7	1.08	0.95, 1.23	0.94	0.84, 1.06	1.02	0.90, 1.16
Sudden Gain* Baseline WSAS	0.99	0.92, 1.07	1.04	0.97, 1.10	0.99	0.92, 1.07
Sudden Gain* Number of treatment sessions	1.07	0.95, 1.21	1.09*	1.00, 1.18	1.09	1.00, 1.19
Sudden Gain* IMD score	0.97	0.90, 1.03	1.00	0.96, 1.04	0.98	0.93, 1.03
Sudden Gain* Counselling vs HiCBT	1.42	0.14, 14.26	0.54	0.13, 2.25	0.24	0.05, 1.14
Sudden Gain* Group vs HiCBT	0.62	0.09, 3.92	0.41	0.10, 1.62	0.68	0.09, 4.75
Sudden Gain* LiCBT vs HiCBT	0.83	0.21, 3.39	1.05	0.33, 3.36	0.55	0.12, 2.43
No change vs improvement						
Baseline PHQ-9	0.87***	0.86, 0.88	1.06***	1.04, 1.07	1.03***	1.02, 1.05
Baseline GAD-7	1.01*	1.00, 1.03	0.81***	0.79, 0.82	0.99	0.97, 1.00
Baseline WSAS	1.02***	1.01, 1.03	1.02***	1.01, 1.03	0.89***	0.88, 0.89
Number of treatment sessions	1.02***	1.01, 1.04	1.01	0.99, 1.01	1.03***	1.01, 1.04
IMD score	1.01**	1.00, 1.01	1.01**	1.00, 1.01	1.01*	1.00, 1.01
Counselling vs HiCBT	0.88	0.75, 1.04	0.95	0.97, 1.12	1.26*	1.05, 1.51
Group vs HiCBT	0.88	0.74, 1.05	0.78**	0.65, 0.93	0.71**	0.58, 0.87

LiCBT vs HiCBT	1.11	0.97, 1.28	1.06	0.92, 1.22	1.17*	1.00, 1.35
Sudden Gain (Ref group no sudden gain)	0.27***	0.23, 0.32	0.36***	0.32, 0.42	0.45***	0.39, 0.51
Sudden Gain* Baseline PHQ-9	0.92***	0.89, 0.96	0.98	0.94, 1.02	0.98	0.95, 1.01
Sudden Gain* Baseline GAD-7	1.03	0.99, 1.07	0.94**	0.90, 0.98	1.01	0.98, 1.04
Sudden Gain* Baseline WSAS	1.01	0.99, 1.03	0.99	0.98, 1.02	0.97**	0.95, 0.99
Sudden Gain* Number of treatment sessions	1.03*	1.00, 1.07	1.02	0.99, 1.05	1.00	0.97, 1.03
Sudden Gain* IMD score	1.00	0.99, 1.02	1.00	0.98, 1.01	1.00	0.98, 1.01
Sudden Gain* Counselling vs HiCBT	1.13	0.68, 1.87	1.38	0.86, 2.21	0.92	0.60, 1.39
Sudden Gain* Group vs HiCBT	0.83	0.47, 1.47	1.04	0.61, 1.77	0.89	0.54, 1.49
Sudden Gain* LiCBT vs HiCBT	0.87	0.59, 1.28	1.21	0.85, 1.74	0.97	069, 1.35

Note. PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; OR = Odds Ratio; 95% CI = 95% Lower and Upper Confidence Intervals; HiCBT = High intensity cognitive behavioural therapy; LiCBT = Low intensity cognitive behavioural therapy; Ref group = reference group. * p<.05, ** p<.010, *** p<.001

Some in text odd ratios are inverse of the odds ratio presented in the table for ease of explanation.

Table 2. 4

Logistic Regression Results of Sudden Gain and Depression Spike on PHQ-9, GAD-7 and WSAS Clinically Significant Change

	PHQ-9 CSC		GAD-7 CSC		WSAS CSC	
	OR	95% CI	OR	95% CI	OR	95% CI
Sudden Gains						
Baseline PHQ-9	1.06***	1.04, 1.07	0.99	0.98, 1.00	0.99	0.98, 1.01
Baseline GAD-7	0.99*	0.98, 0.99	1.00	0.99, 1.01	1.00	0.99, 1.02
Baseline WSAS	0.98***	0.97, 0.98	1.00	0.99, 1.00	0.99	0.99, 1.00
Number of treatment sessions	0.98**	0.97, 0.99	0.98*	0.97, 0.99	0.99	0.97, 1.00
IMD score	0.99***	0.98, 0.99	1.00	0.99, 1.00	1.00	0.99, 1.01
Counselling vs HiCBT	0.99	0.85, 1.14	0.97	0.84, 1.12	0.92	0.77, 1.08
Group vs HiCBT	1.26**	1.08, 1.48	1.03	0.88, 1.19	1.12	0.92, 1.35
LiCBT vs HiCBT	0.91	0.81, 1.02	0.99	0.89, 1.11	1.00	0.87, 1.15
Sudden Gain (Ref group no sudden gain)	3.02***	2.68, 3.40	0.81***	0.72, 0.91	0.75***	0.64, 0.86
Sudden Gain* Baseline PHQ-9	0.99	0.97, 1.02	1.00	0.97, 1.03	0.98	0.95, 1.02
Sudden Gain* Baseline GAD-7	0.98	0.95, 1.02	0.99	0.96, 1.03	1.01	0.97, 1.05

Sudden Gain* Baseline WSAS	0.99	0.97, 1.01	0.99	0.98, 1.01	0.99	0.97, 1.02
Sudden Gain* Number of treatment sessions	0.97*	0.94, 0.99	0.99	0.97, 1.02	1.01	0.97, 1.04
Sudden Gain* IMD score	1.00	0.99, 1.01	0.99	0.98, 1.01	0.99	0.98, 1.01
Sudden Gain* Counselling vs HiCBT	0.91	0.61, 1.33	1.03	0.72, 1.51	1.08	0.67, 1.75
Sudden Gain* Group vs HiCBT	1.45	0.93, 2.27	0.92	0.59, 1.43	0.59	0.34, 1.02
Sudden Gain* LiCBT vs HiCBT	1.27	0.94, 1.71	1.06	0.78, 1.42	1.04	0.71, 1.52
Depression Spikes						
Baseline PHQ-9	1.06***	1.04, 1.06	0.99	0.98, 1.01	0.99	0.98, 1.01
Baseline GAD-7	0.98*	0.97, 0.99	1.00	0.99, 1.01	1.00	0.99, 1.02
Baseline WSAS	0.98***	0.97, 0.98	1.00	0.99, 1.01	0.99	0.99, 1.01
Number of treatment sessions	0.98**	0.97, 0.99	0.98*	0.97, 0.99	0.99	0.97, 1.00
IMD score	0.99***	0.98, .099	1.00	0.99, 1.01	1.00	0.99, 1.01
Counselling vs HiCBT	0.98	0.85, 1.14	0.97	0.84, 1.12	0.92	0.77, 1.08
Group vs HiCBT	1.26**	1.08, 1.47	1.03	0.88, 1.19	1.12	0.92, 1.35
LiCBT vs HiCBT	0.90	0.81, 1.02	0.99	0.89, 1.12	1.00	0.87, 1.15

Depression Spike (Ref group no depression spike)	1.16**	1.03, 1.29	0.93	0.84, 1.05	0.99	0.87, 1.14
Depression Spike * Baseline PHQ-9	1.02*	1.00, 1.06	1.01	0.98, 1.03	0.98	0.95, 1.02
Depression Spike * Baseline GAD-7	1.03*	1.00, 1.06	1.02	0.98, 1.04	1.02	0.98, 1.05
Depression Spike * Baseline WSAS	1.01	0.99, 1.03	0.99	0.98, 1.01	0.99	0.98, 1.02
Depression Spike * Number of treatment sessions	1.00	0.98, 1.03	0.98	0.96, 1.01	1.00	0.97, 1.03
Depression Spike * IMD score	0.99	0.98, 1.001	1.00	0.99, 1.01	1.00	0.99, 1.02
Depression Spike * Counselling vs HiCBT	1.19	0.84, 1.69	1.26	0.89, 1.77	1.44	0.95, 2.19
Depression Spike * Group vs HiCBT	0.83	0.56, 1.23	1.06	0.72, 1.55	0.96	0.61, 1.54
Depression Spike * LiCBT vs HiCBT	1.15	0.86, 1.52	1.14	0.87, 1.50	1.19	0.85, 1.67

Note. CSC= Clinically Significant Change; PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; IMD = Index of Multiple Deprivation; OR = Odds Ratio; 95% CI = 95% Lower and Upper Confidence Intervals; HiCBT = High intensity cognitive behavioural therapy; LiCBT = Low intensity cognitive behavioural therapy; Ref group = reference group. * p<.05, ** p<.010, *** p<.001

Some in text odd ratios are inverse of the odds ratio presented in the table for ease of explanation.

Table 2. 5

Hierarchical Regression Results for Depression Spike Status on PHQ-9 Depression Outcome, GAD-7 Anxiety Outcome and WSAS Functioning Outcome

	PHQ-9 Outcome			GAD-7 Outcome			WSAS Outcome		
	B(*Se*)	95% CI	R^2, Δ R^2	*B*(*Se*)	95% CI	R^2, Δ R^2	*B*(*Se*)	95% CI	R^2, Δ R^2
Step 1			.16, .16***			.16, .16***			.18, 1.8***
Constant	0.42(0.32)	-0.20, 1.04		0.38(0.27)	-0.15, 0.90		1.225(0.44)**	0.36, 2.09	
Baseline PHQ-9	0.32(0.02)***	0.29, 0.35		0.11(0.01)***	0.09, 0.14		0.21(0.02)***	0.17, 0.26	
Baseline GAD-7	0.07(0.02)***	0.03, 0.10		0.28(0.01)***	0.25, 0.30		-0.02(0.02)	-0.07, 0.03	
Baseline WSAS	0.09(0.01)***	0.07, 0.10		0.06(0.01)***	0.04, 0.07		0.35(0.01)***	0.32, 0.37	
Number of treatment sessions	0.05(0.02)**	0.02, 0.08		0.03(0.01)*	0.01, 0.05		0.10(0.02)***	0.05, 0.14	
IMD score	0.03(0.01)***	0.02, 0.04		0.03(0.01)***	0.02, 0.04		0.04(0.01)***	0.02, 0.05	

Counselling vs HiCBT	-0.14(0.22)	-0.58, 0.29		-0.07(0.19)	-0.44, 0.29		-1.02(0.31)**	-1.63, -0.41	
Group vs HiCBT	0.82(0.23)**	0.35, 1.29		0.59(0.20)**	0.19, 0.99		1.80(0.33)***	1.14, 2.46	
LiCBT vs HiCBT	-0.15(0.18)	-0.51, 0.20		-0.29(0.15)	-0.59, 0.01		-0.68(0.25)**	-1.18, -0.18	
Step 2			.17, .001**			.16, .001**			.19, .002***
Depression Spike (Ref group No Depression Spike)	-0.56(0.18)**	-0.91, -0.22		-0.51(0.15)**	-0.79, -0.21		-1.07(0.25)***	-1.55, -0.59	
Step 3			.17, .01***			.17, .007***			.19, .005***
Baseline PHQ-9 x Depression Spike	-0.16(0.04)***	-0.23, -0.08		-0.11(0.03)**	-0.18, -0.04		-0.18(0.06)**	-0.29, -0.07	
Baseline GAD-7 x Depression Spike	-0.09(0.04)*	-0.17, -0.01		-0.10(0.04)**	-0.17, -0.03		-0.13(0.06)*	-0.25, -0.02	
Baseline WSAS x Depression Spike	-0.04(0.02)	-0.08, 0.01		-0.02(0.02)	-0.06, 0.02		-0.01(0.03)	-0.07, 0.05	

Number of treatment sessions x Depression Spike	-0.03(0.04)	-0.09, 0.04	-0.01(0.03)	-0.07. 0.05	-0.03(0.05)	-0.12, 0.07
IMD x Depression Spike	0.01(0.02)	-0.02, 0.04	0.08(0.01)	-0.19, 0.04	0.04(0.02)	-0.003, 0.08
Counselling vs HiCBT x Depression Spike	0.49(0.54)	-0.56, 1.54	-0.08(0.45)	-0.98, 0.81	0.16(0.75)	-1.31, 1.63
Group vs HiCBT x Depression Spike	0.41(0.59)	-0.77, 1.58	0.48(0.51)	-0.51, 1.47	0.09(0.84)	-1.55, 1.73
LiCBT vs HiCBT x Depression Spike	0.34(0.43)	-0.51, 1.19	0.06(0.37)	-0.66, 0.78	-0.09(0.61)	-1.27, 1.10

Note. CI = confidence interval; PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; IMD = Index of Multiple Deprivation; Ref group = reference group.

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

Table 2. 6

Multinomial Logistic Regression Results of Depression Spike and Treatment on PHQ-9 and GAD-7 Reliable Clinical Change and WSAS Minimally Clinically Significant Change

	PHQ-9 Reliable Change Outcome		GAD-7 Reliable Change Outcome		WSAS Reliable Change Outcome	
Depression Spikes	OR	95% CI	OR	95% CI	OR	95% CI
Deteriorate vs improvement						
Baseline PHQ-9	0.74***	0.72, 0.77	1.11***	1.08, 1.14	1.12***	1.08, 1.15
Baseline GAD-7	1.06**	1.02, 1.09	0.69***	0.67, 0.71	1.03	0.99, 1.06
Baseline WSAS	1.05***	1.03, 1.07	1.02*	1.01, 1.04	0.77***	0.76, 0.78
Number of treatment sessions	1.04*	1.00, 1.08	1.04*	1.01, 1.07	1.08***	1.05, 1.12
IMD score	1.02**	1.00, 1.03	1.01*	1.00, 1.02	1.02**	1.01, 1.03
Counselling vs HiCBT	1.33	0.81, 2.19	1.18	0.79, 1.76	1.30	0.94, 2.03
Group vs HiCBT	0.78	0.48, 1.26	0.93	0.62, 1.39	0.41***	0.26, 0.65
LiCBT vs HiCBT	1.31	0.87, 1.96	1.36	0.99, 1.87	1.21	0.83, 1.77
Depression Spike (Ref group no depression spike)	1.46**	1.11, 1.93	1.07	0.83, 1.39	0.71*	0.52, 0.98

Depression Spike * Baseline PHQ-9	0.97	0.90, 1.05	0.95	0.89, 1.00	0.93*	0.87, 0.99
Depression Spike * Baseline GAD-7	0.95	0.88, 1.01	0.97	0.90, 1.04	0.94	0.87, 1.01
Depression Spike * Baseline WSAS	0.99	0.95, 1.03	0.99	0.95, 1.02	1.01	0.96, 1.06
Depression Spike * Number of treatment sessions	0.97	0.92, 1.03	0.97	0.91, 1.02	0.95	0.89, 1.01
Depression Spike * IMD score	0.99	0.96, 1.01	1.00	0.98, 1.03	0.99	0.96, 1.02
Depression Spike * Counselling vs HiCBT	0.94	0.39, 2.30	1.46	0.65, 3.26	0.87	0.33, 2.35
Depression Spike * Group vs HiCBT	1.01	0.41, 2.46	0.69	0.30, 1.56	1.10	0.39, 3.14
Depression Spike * LiCBT vs HiCBT	1.03	0.50, 2.11	1.57	0.79, 3.09	0.71	0.31, 1.65
No change vs improvement						
Baseline PHQ-9	0.87***	0.86, 0.88	1.05***	1.04, 1.07	1.03***	1.02, 1.05
Baseline GAD-7	1.02**	1.01, 1.03	0.81***	0.80, 0.83	0.99	0.98, 1.01
Baseline WSAS	1.02***	1.02, 1.03	1.02***	1.01, 1.03	0.89***	0.88, 0.89
Number of treatment sessions	1.02**	1.01, 1.04	1.01	0.99, 1.03	1.03***	1.02, 1.05
IMD score	1.01**	1.00, 1.01	1.00**	1.00, 1.01	1.00	0.99, 1.01
Counselling vs HiCBT	0.89	0.76, 1.06	0.96	0.81, 1.14	1.21*	1.01, 1.46
Group vs HiCBT	0.81*	0.68, 0.97	0.78*	0.65, 0.94	0.65***	0.53, 0.79

LiCBT vs HiCBT	1.12	0.97, 1.28	1.14	0.98, 1.31	1.12	0.97, 1.30
Depression Spike (Ref group no depression spike)	0.80***	0.71, 0.91	0.82**	0.72, 0.93	0.73***	0.64, 0.82
Depression Spike * Baseline PHQ-9	0.95**	0.92, 0.98	0.97*	0.94, 0.99	0.96**	0.93, 0.99
Depression Spike * Baseline GAD-7	0.98	0.95, 1.10	0.97	0.95, 1.01	0.98	0.95, 1.02
Depression Spike * Baseline WSAS	0.99	0.97, 1.00	0.99	0.98, 1.01	0.99	0.98, 1.02
Depression Spike * Number of treatment sessions	0.99	0.97, 1.01	0.99	0.97, 1.02	0.98	0.96, 1.01
Depression Spike * IMD score	1.00	0.99, 1.101	1.00	0.99, 1.02	1.01	0.99, 1.02
Depression Spike * Counselling vs HiCBT	0.98	0.67, 1.46	1.19	0.80, 1.78	0.99	0.67, 1.48
Depression Spike * Group vs HiCBT	1.05	0.68, 1.62	0.78	0.51, 1.21	1.12	0.71, 1.76
Depression Spike * LiCBT vs HiCBT	0.91	0.66, 1.25	0.83	0.61, 1.14	1.09	0.80, 1.50

Note. PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; OR = Odds Ratio; 95% CI = 95% Lower and Upper Confidence Intervals; HiCBT = High intensity cognitive behavioural therapy; LiCBT = Low intensity cognitive behavioural therapy; Ref group = reference group. * p<.05, ** p<.010, *** p<.001

Some in text odd ratios are inverse of the odds ratio presented in the table for ease of explanation.

2.5.3.2 Anxiety Outcome

In line with expectations, experiencing a sudden gain (*M* = 5.72, *SD* = 4.85) was associated with significantly lower GAD-7 anxiety scores at treatment outcome than those who did not have a sudden gain (*M* = 8.08, *SD* = 5.91) (Table 2.2) with a medium effect size (Hedges's *g* = 0.41). Those who had a sudden gain were more likely to experience reliable improvement than deterioration (OR= 6.25) or no change (OR= 2.78) in GAD-7 scores (Table 2.3). However, individuals who had a sudden gain, compared to no sudden gain, were significantly *less* likely to experience CSC in GAD-7 scores (OR= 0.81; Table 2.4).

Having a depression spike (*M* = 7.29, *SD* = 5.55) was associated with lower anxiety scores at the end of treatment, compared to those who did not have a depression spike (*M* = 7.79, *SD* = 5.87) (Table 2.5) and this was a small effect (Hedges's *g* = 0.09), and experiencing a depression spike was associated with reliable improvement, compared to no change (OR= 1.22) in GAD-7 scores (Table 2.6). Depression spike status was not associate with CSC in GAD-7 scores (Table 2.4).

2.5.3.3 Reliable Recovery

Reliable recovery considers the change in both PHQ-9 and GAD-7 scores from baseline to the end of treatment. The results of how sudden gain or depression spike status are associated with reliable recovery are shown in Table 2.7. Individuals who experienced a sudden gain were nearly three times more likely to have reliable recovery at the end of treatment than those who did not experience a sudden gain. Those who experienced a depression spike, compared to no depression spike, were more likely (OR = 1.14) to experience reliable recovery.

Table 2. 7

Logistic Regression Results of Sudden Gain and Depression Spike and Treatment on Reliable Recovery

	OR	95% CI
Outcome- Reliable Recovery (0,1)		
Step 1		
Baseline PHQ-9	1.00	0.99, 1.01
Baseline GAD-7	1.02**	1.01, 1.03
Baseline WSAS	0.99***	0.97, 0.99
Number of treatment sessions	0.98***	0.97, 0.99
IMD score	0.98***	0.98, 0.99
Counselling vs HiCBT	1.03	0.89, 1.18
Group vs HiCBT	1.36***	1.16, 1.58
LiCBT vs HiCBT	0.87*	0.77, 0.98
Step 2		
Sudden Gain (Ref group no sudden gain)	2.93***	2.59, 3.03
Step 3		
Sudden Gain* Baseline PHQ-9	1.03*	1.01, 1.07
Sudden Gain* Baseline GAD-7	0.97	0.94, 1.00
Sudden Gain* Baseline WSAS	0.98*	0.96, 0.99
Sudden Gain* Number of treatment sessions	0.97*	0.94, 0.99
Sudden Gain* IMD score	0.99	0.98, 1.01
Sudden Gain* Counselling vs HiCBT	0.92	0.63, 1.36
Sudden Gain* Group vs HiCBT	1.52	0.97, 2.38

Sudden Gain* LiCBT vs HiCBT	1.09	0.80, 1.47
Outcome- Depression Spike (0,1)		
Step 1		
Baseline PHQ-9	1.00	0.99, 1.01
Baseline GAD-7	1.02**	1.01, 1.03
Baseline WSAS	0.98***	0.97, 0.99
Number of treatment sessions	0.98***	0.97, 0.99
IMD score	0.98***	0.98, 0.99
Counselling vs HiCBT	1.03	0.89, 1.18
Group vs HiCBT	1.36***	1.16, 1.58
LiCBT vs HiCBT	0.87*	0.77, 0.98
Step 2		
Depression Spike (Ref group no depression Spike)	1.14*	1.02, 1.27
Step 3		
Depression Spike * Baseline PHQ-9	1.02	0.99, 1.05
Depression Spike * Baseline GAD-7	1.03*	1.00, 1.06
Depression Spike * Baseline WSAS	1.01	0.99, 1.02
Depression Spike * Number of treatment sessions	1.00	0.97, 1.02
Depression Spike * IMD score	0.99	0.98, 1.00
Depression Spike * Counselling vs HiCBT	1.07	0.76, 1.51
Depression Spike * Group vs HiCBT	1.15	0.77, 1.69
Depression Spike * LiCBT vs HiCBT	1.09	0.83, 1.45

Note. PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; IMD = Index of Multiple Deprivation; OR = Odds Ratio; 95% CI = 95% lower and upper confidence intervals;

HiCBT = High intensity cognitive Behavioural Therapy; LiCBT = Low intensity cognitive behavioural therapy; Ref group = reference group. * p<.05, ** p<.010, *** p<.001

Some in text odd ratios are inverse of the odds ratio presented in the table for ease of explanation.

2.5.3.4 Functioning Outcome

Consistent with hypotheses experiencing a sudden gain (*M* = 9.74, *SD* = 8.29) was associated with significant functioning improvement on the WSAS at the end of treatment, compared to individuals who did not experience a sudden gain (*M* = 13.04, *SD* = 9.87) (Table 2.2) with a medium effect size (Hedges's *g* = 0.34). Additionally, individuals with sudden gains, compared to no sudden gains, were significantly more likely to reliably improve than deteriorate (OR= 8.33) or have no change (OR= 2.22) in WSAS scores (Table 2.3). However, individuals with sudden gains, compared to no gains, were significantly less likely to experience CSC in WSAS functioning scores (OR = 0.75; Table 2.4).

Experiencing a depression spike (*M* = 11.74, *SD* = 9.22) was associated with significantly lower WSAS scores at the end of treatment, compared to no depression spike (*M* = 12.68, *SD* = 9.81) (Table 2.5), with a small effect size (Hedges's *g* = 0.10). Individuals who experienced depression spikes, compared to no spikes, were more likely to reliably improve than deteriorate (OR= 1.41) or have no change (OR= 1.37) in WSAS scores (Table 2.6). Depression spike status was not associated with CSC in WSAS scores (Table 2.4).

2.5.4 Moderators of the Relationship between Sudden Gain or Depression Spike Status and Outcome

We examined six moderators (treatment type, baseline PHQ-9, GAD-7, WSAS score, number of treatment sessions and IMD score) of the relationship between sudden gain or depression spike status in relation to continuous depression, anxiety and functioning outcomes, as well as reliable recovery outcomes.

2.5.4.1 Depression Outcome.

Contrary to expectations, treatment type (HiCBT, LiCBT, group or counselling) did not moderate the relationship between sudden gain (continuous, Table 2.2; reliable recovery, Table 2.7) or depression spike (continuous, Table 2.5; reliable recovery, Table 2.7) status and depression outcomes on any of the measures.

Baseline depression score moderated the association between sudden gain status and depression outcome (Table 2.2). The effect of having a sudden gain on PHQ-9 outcome was more beneficial for individuals who had higher baseline depression severity than those with lower baseline depression scores (Table 2.8) (see Figure 1.1a). No other significant moderations were found for the relationship between sudden gain status and PHQ-9 continuous depression outcome (Table 2.2).

Table 2. 8

Explication of the Significant Sudden Gain and Depression Spike Moderations

	Low baseline (-1*SD*)		High baseline (+1*SD*)	
	B(*Se*)	95% CI	*B*(*Se*)	95% CI
Sudden gains				
Baseline PHQ x Sudden Gain on PHQ-9 outcome	-1.98(0.30)***	-2.57, -1.39	-5.10(0.23)***	-5.54, -4.65
Baseline PHQ x Sudden Gain on GAD-7 outcome	-1.61(0.26)***	-2.12, -1.10	-3.96(0.20)***	-4.35, -3.57
Baseline PHQ x Sudden Gain on WSAS outcome	-2.21(0.44)***	-3.086 -1.34	-5.72(0.34)***	-6.38, -5.06
Baseline GAD-7 x Sudden Gain on GAD-7 outcome	-1.65(0.24)***	-2.12, -1.18	-3.79(0.20) ***	-4.18, -3.39
Depression spikes				
Baseline PHQ x Depression Spike on PHQ-9 outcome	0.96(.22)***	0.52, 1.39	-1.88(0.24)***	-2.35, -1.41
Baseline PHQ x Depression Spike on GAD-7 outcome	0.73(.19)***	0.35, 1.10	-1.39(0.21)***	-1.79, -0.98
Baseline PHQ x Depression Spike on WSAS outcome	0.74(0.32)**	0.11, 1.37	-2.06(0.35)***	-2.74, -1.38
Baseline GAD-7 x Depression Spike on PHQ-9 outcome	0.53(0.24)*	0.06, 0.99	-1.85(.24)***	-2.32, -1.38

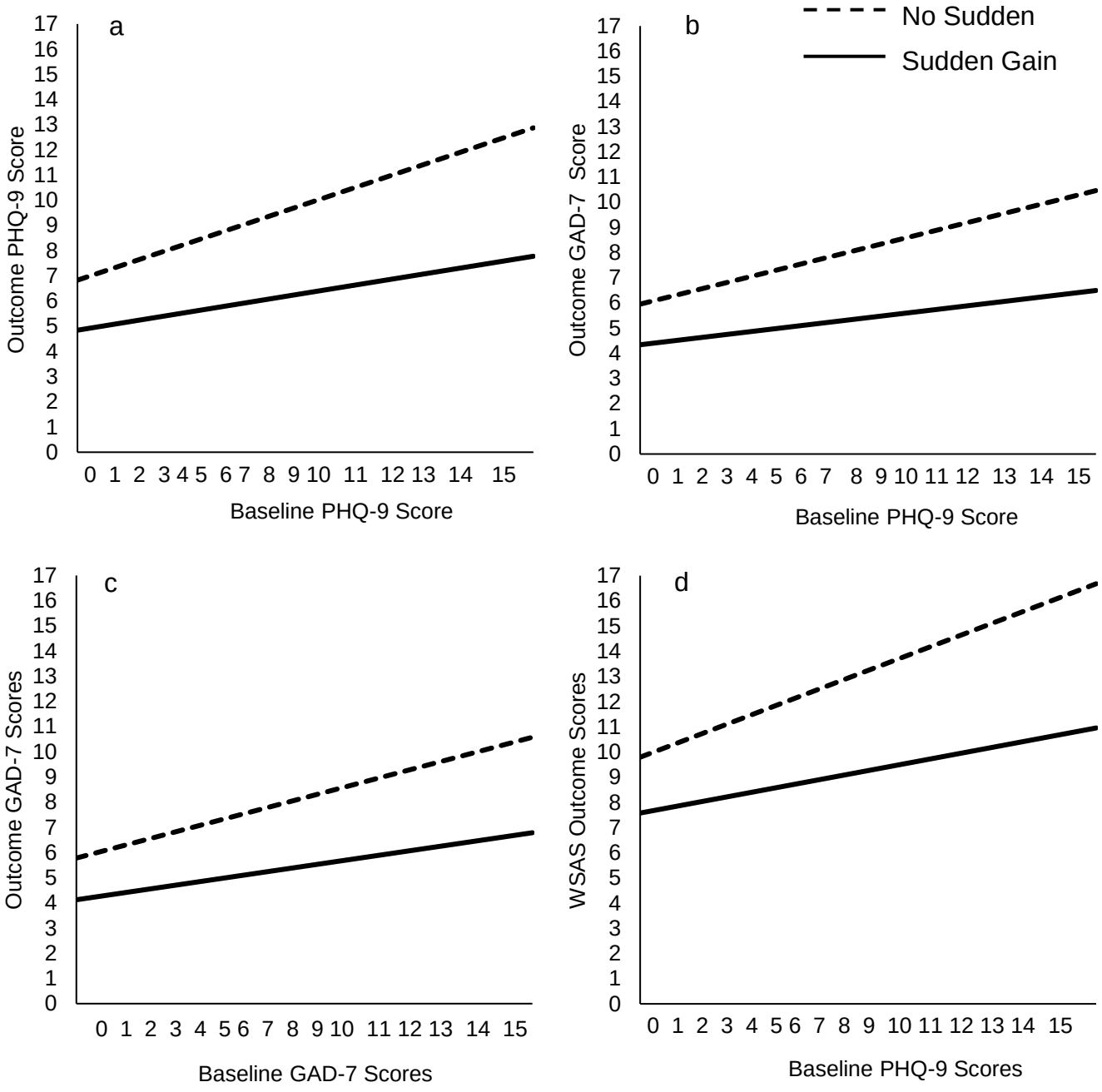
Baseline GAD-7 x Depression Spike on GAD-7 outcome	0.51(.19)**	0.12, 0.89	-1.44(0.20)***	-1.82, -1.05
Baseline GAD-7 x Depression Spike on WSAS outcome	0.31(0.34)	-0.352, 0.988	-2.131(0.346)***	-2.810, -1.452

Note. CI = confidence interval; PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Work and Social Adjustment Scale; IMD = Index of Multiple Deprivation.

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

Figure 2. 2

Graphs of Sudden Gain Moderations



Note. PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Working and Adjustment Scale.

a. Relationship between sudden gain status and outcome PHQ-9 score moderated by baseline PHQ-9 score

b. Relationship between sudden gain status and outcome GAD-7 score moderated by baseline PHQ-9 score

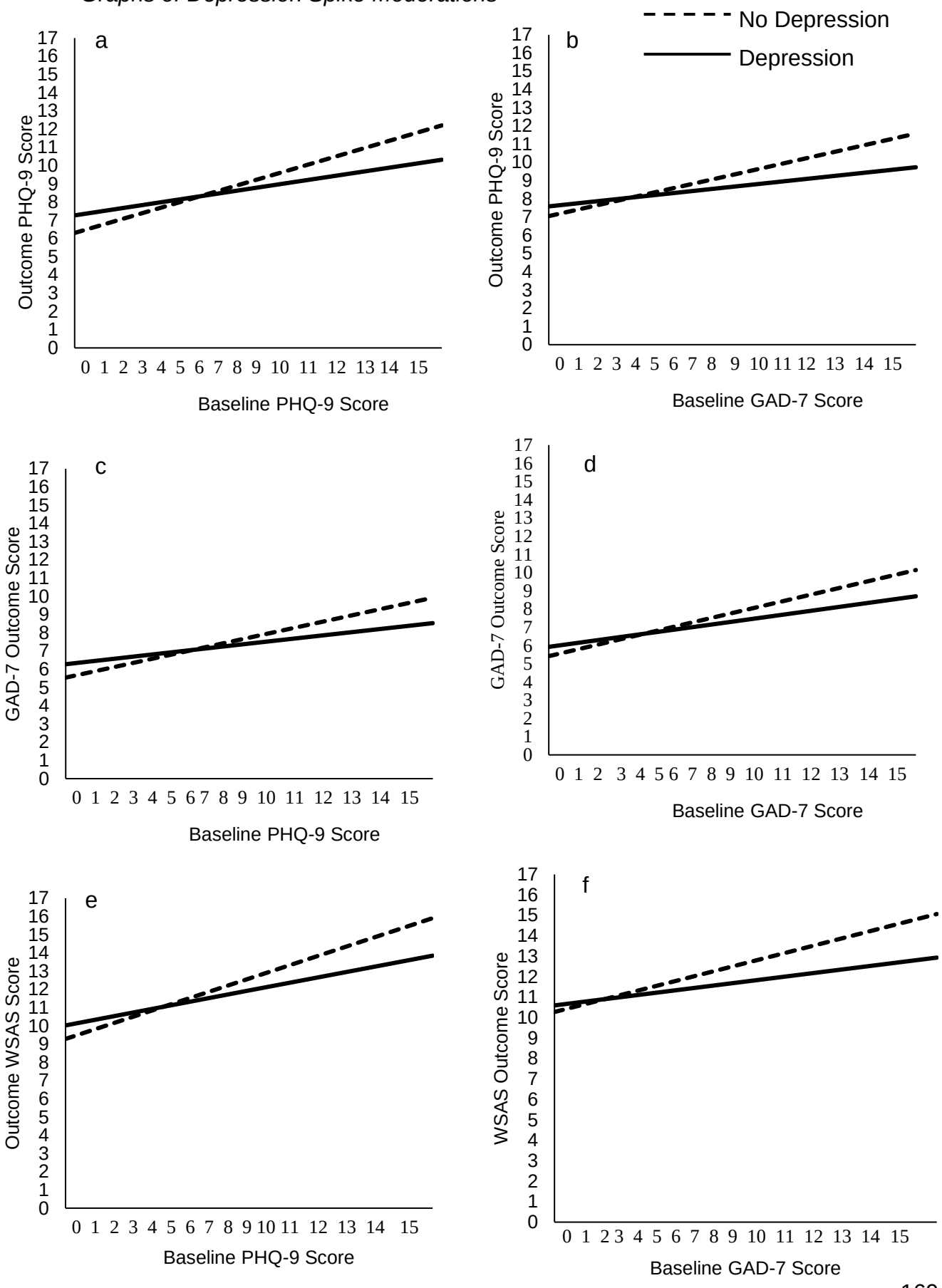
c. Relationship between sudden gain status and outcome GAD-7 score moderated by baseline GAD-7 score

d. Relationship between sudden gain status and outcome WSAS score moderated by baseline PHQ-9 score

The relationship between depression spike status and PHQ-9 depression outcome was moderated by baseline PHQ-9 and GAD-7 score (Table 2.5). As baseline depression scores increased, presence of a depression spike, compared to no depression spike (Table 2.8), was more likely to be associated with favourable outcomes post-treatment (Figure 2.2a). Similarly, as baseline GAD-7 scores increased, experiencing a depression spike compared to no spike (Table 2.8), was significantly associated with lower PHQ-9 treatment outcome scores (Figure 2.2b). No other significant moderations were found for the relationship between depression spike status and PHQ-9 continuous treatment outcome (Table 2.5).

Figure 2. 3

Graphs of Depression Spike Moderations



Note. PHQ-9 = Patient Health Questionnaire; GAD-7 = Generalised Anxiety Disorder; WSAS = Working and Adjustment Scale.

a. Relationship between depression spike status and outcome PHQ-9 score moderated by baseline PHQ-9 score

b. Relationship between depression spike status and outcome PHQ-9 score moderated by baseline GAD-7 score

c. Relationship between depression spike status and outcome GAD-7 score moderated by baseline PHQ-9 score

d. Relationship between depression spike status and outcome GAD-7 score moderated by baseline GAD-7 score

e. Relationship between depression spike status and outcome WSAS score moderated by baseline PHQ-9 score

f. Relationship between depression spike status and outcome WSAS score moderated by baseline GAD-7 score

2.5.4.2 Anxiety Outcome.

Contrary to expectations, treatment type (HiCBT, LiCBT, group or counselling) did not moderate the relationship between sudden gain (continuous, Table 2.2; reliable recovery, Table 2.7) or depression spike (continuous, Table 2.5; reliable recovery, Table 2.7) status and anxiety outcomes on any of the measures.

Baseline PHQ-9 and GAD-7 moderated the association between sudden gain status and anxiety outcome (Table 2.2). The effect of experiencing a sudden gain, compared to no gain on GAD-7 outcome scores (Table 2.8), was more beneficial for individuals who had higher baseline depression severity (Figure 2.2b). Similarly the

presence of a sudden gain was associated with lower post-treatment GAD scores at all levels of baseline GAD (Table 2.8), and this effect was greatest for those with higher GAD scores at baseline (Figure 2.2c). No other significant moderations were found for the relationship between sudden gain status and GAD-7 continuous outcome (Table 2.2).

Baseline PHQ-9 and GAD-7 moderated the relationship between depression spike status and anxiety outcome (Table 2.5). As both baseline PHQ-9 (Figure 2.3c) and GAD-7 (Figure 2.3d) increased, presence of a depression spike was more likely to be associated with favourable anxiety outcome at the end of treatment (Table 2.8). No other significant moderations were found for the relationship between depression spike and GAD-7 continuous treatment outcome (Table 2.5).

2.5.4.3 Functioning Outcome.

Contrary to expectations, treatment type (HiCBT, LiCBT, group or counselling) did not moderate the relationship between sudden gain (continuous, Table 2.2; reliable recovery, Table 2.7) or depression spike (continuous, Table 2.5; reliable recovery, Table 2.7) status and functioning outcomes on any of the measures.

The relationship between sudden gain status and WSAS outcome was moderated by baseline PHQ-9 score (Table 2.2). Experiencing a sudden gain, compared to no sudden gain, was associated with significantly lower WSAS outcome scores at all levels of baseline PHQ-9 scores (Table 2.8) and this effect was greatest for those with higher PHQ-9 scores at baseline (Figure 2.2d). No other significant moderations were found for the relationship between sudden gain status and WSAS continuous outcome (Table 2.2).

Baseline PHQ-9 and GAD-7 score moderated the relationship between depression spike status and WSAS treatment outcome (Table 2.5). Experiencing a depression spike, compared to no depression spike (Table 2.8), resulted in more favourable WSAS scores at the end of treatment in individuals who had higher baseline PHQ-9 scores (Figure 2.3e). Low baseline GAD-7 was not significantly associated with WSAS outcome scores (Table 2.5). For individuals with more severe baseline GAD-7 scores, experiencing a depression spike, compared to those who did not, was associated with significantly lower and WSAS outcomes (Table 2.8; Figure 2.3f). No other significant moderations were found for the relationship between depression spike and WSAS continuous treatment outcome (Table 2.5).

2.5.4.4 Reliable Recovery Outcome.

Contrary to expectations, treatment modality did not moderate the relationship between sudden gain or depression spike status on reliable recovery outcome (Table 2.7).

The relationship between sudden gain status and reliable recovery was moderated by baseline PHQ-9 and WSAS scores and number of treatment sessions (Table 2.7). Individuals who had a sudden gain, compared to no gain, and had higher baseline PHQ-9 scores were more likely to experience reliable recovery at the end of treatment. However, sudden gains individuals who had higher baseline WSAS scores or greater number of treatment sessions were significantly less likely to experience reliable recovery outcome at the end of treatment, compared to those who did not have a sudden gain.

Depression spike individuals, compared to no spike, who higher baseline GAD scores were associated with greater rates of reliable recovery. No other

significant moderations were found for sudden gain status on reliable recovery outcome.

2.6 Discussion

The current study examined sudden gains and depression spikes across four treatments in a large, primary mental health care dataset (IAPT) that drew from multiple clinics, therapists, and psychological practitioners across the Southwest of England. To our knowledge this is the first study to directly compare patterns of discontinuous change in individuals primarily presenting with depression, between multiple low- and high-intensity treatments. Here we observed that discontinuous changes also occur in low-intensity treatments and are associated with beneficial treatment outcomes despite having typically fewer therapy sessions and being delivered by less experienced psychological practitioners. The frequency of sudden gains (19%) and depression spikes (24%) in the current sample were comparable to previous research, but notably highest in HiCBT. Our findings support existing research demonstrating that sudden gains (Shalom & Aderka, 2020) and depression spikes (Grosse et al., 2012; Hayes, Feldman, Beevers, et al., 2007) are associated with beneficial depression treatment outcomes, and we extend this to show both patterns of discontinuous change are also associated with reduced anxiety symptoms and improved functioning at the end of treatment in clients presenting with depression as a primary problem. While treatment type did not moderate any of the associations between discontinuous change and outcomes, we found individuals with higher baseline depression, anxiety and functioning scores benefitted most from a sudden gain or depression spike on end of treatment outcomes in terms of their overall response to treatment. Across all treatments experiencing a sudden gain or depression spike was beneficial on end of treatment outcomes, however there were

differences in the rates of discontinuous change between treatments suggesting discontinuous change may be more likely to occur in some treatments compared to others.

The frequency of sudden gains in this sample are similar to other clinic samples (9-32.7%; Koffmann, 2019; Lutz et al., 2013; Wucherpfennig, Rubel, Hofmann, et al., 2017) but as seen across this literature they were notably lower than rates of sudden gains in trials (40%; Shalom & Aderka, 2020). It is unclear why the overall rate of sudden gains in this sample were generally lower than in trial samples, although it is worth noting that rates of sudden gains in HiCBT (24%) were within the range of rates of sudden gains in clinical trials (Shalom & Aderka, 2020). Lower rates of sudden gains in regular clinical practice could be due to sample differences, therapist experience (Deisenhofer et al., 2021), and/or the limited number of treatment sessions typically delivered in routine care samples. However, in the current study sudden gains were more likely to occur earlier in treatment and therefore might not be due to the number of therapy sessions received. Rates of sudden gains were highest in HiCBT and notably lower in group-based treatment. This finding is consistent with previous research that has found sudden gains (Norton et al., 2010; Thorisdottir et al., 2018; for an exception see Kelly et al., 2005) occur less frequently in both psychoeducational and therapeutic groups. With regards to depression spikes the rates in this sample (24%) were lower than in previous studies of depression spikes in EBCT (62%; Hayes, Feldman, Beevers, et al., 2007), but similar to rates of depression spikes in smaller samples in trials (O'Mahen et al., 2021; O'Mahen et al., 2017; O'Mahen et al., 2019). There were greater rates of depression spikes in high-intensity treatments and, consistent with Hayes et al. (2007), it is possible that this is due to greater depth processing of

depression content in higher intensity treatments. Whereas in the low-intensity treatments there were fewer spikes and no differences in the rates between the treatments. While previous research has tended to focus on the relationship between discontinuous change and treatment outcomes these findings perhaps highlight the importance of looking at rates of discontinuous change between treatments. It may also be important to consider why treatments of different intensities have varying rates of discontinuous change. In a recently revised theory of sudden gains, Aderka and Shalom (2021) suggest individuals with depression may experience fluctuating symptoms of depression, both within and outside therapy. Rather than sudden gains being brought about by treatment it is the interaction of experiencing these fluctuations of depression symptoms within a treatment context that may change the slope of the fluctuations to produce a sudden gain. Therefore, it is perhaps that some treatments may be more likely to facilitate the change of slope of depression fluctuations and increase the probability that a sudden gain will occur (Aderka & Shalom, 2021). It is also possible that this applies to depression spikes. Thus in the context of the current study high-intensity therapies may be more likely to produce a change of slope of fluctuating depression symptoms than low-intensity therapies. Further research is needed to compare the rates of discontinuous change between treatments of different intensity, as well as examine depression symptom fluctuations prior to and within treatment to see whether some treatments are more likely to encourage discontinuous change.

With regards to timing, both sudden gains and, unexpectedly, depression spikes were more likely to occur in early treatment. This is consistent with the sudden gains literature (Shalom & Aderka, 2020) and research has explored different client (Abel et al., 2016; Aderka et al., 2021; Lemmens et al., 2021; Shalom

et al., 2018; Zilcha-Mano et al., 2019) and therapist (Deisenhofer et al., 2021) factors that may help to instigate a sudden gain. For depression spikes this is contrary to the seminal study in which spikes in the middle of EBCT were related to positive treatment outcomes (Hayes, Feldman, Beevers, et al., 2007). Depression spikes in non-EBCT treatments have generally been found to occur around the middle of treatment (Abel, 2014; O'Mahen et al., 2017; O'Mahen et al., 2019) but these are in smaller samples. Understanding what depression spikes represent in non-EBCT therapies may help to explain differences in timings of spikes outside of EBCT. They were originally discussed in relation to deliberate therapeutic events which temporarily exacerbated depression symptoms (Hayes, Feldman, Beevers, et al., 2007) whereas this is not a feature of treatments delivered in IAPT for depression. Processes that instigate depression spikes have not yet been examined in non-EBCT treatments. Although speculative, depression spikes may represent iatrogenic effects of therapy, unintended therapeutic events like a therapeutic rupture and repair, or the processing of difficult material in therapy. On the other hand, they may signify the impact of external life stressors. Outside the depression spikes literature, work by Lambert and colleagues has focussed on identifying individuals who are not on track to experience favourable treatment outcomes (because they may have experienced a deterioration in symptoms, like the deterioration of symptoms in a depression spike) and to develop tools to help clinicians recognise and focus on factors that may improve treatment outcomes (Lambert, 2015; Lambert et al., 2001; Probst et al., 2020). Routine outcome monitoring (ROM) systems are used to help clinicians to recognise an individual may be on a trajectory to poor treatment response, and tools are utilised to help increase therapy collaboration in areas such as improving therapeutic alliance, client motivation, social support, or help with

stressful life events- all of which have been consistently associated with good treatment outcomes (Lambert et al., 2018). For depression spikes, using ROM systems within therapy may help to identify factors that lead to the deterioration of symptoms observed in a depression spike. Further, for individuals with depression spikes who have worse treatment outcomes (e.g. O'Mahen et al., 2021) this early identification and targeted focus on specific process may help to improve treatment outcomes. Further investigation of this and the factors which predict depression spikes in non-exposure based treatments is needed.

We found that discontinuous change was associated with better end-of-treatment depression dimensional outcomes, regardless of treatment type. With regards to sudden gains, this finding is consistent with previous meta-analyses of sudden gains (Shalom & Aderka, 2020). We also note sudden gains were associated with reliable improvements in scores at the end of treatment, reliable recovery and CSC in depression scores at the end of treatment. While sudden gains look to be beneficial, it is important to examine the association with longer term outcomes between treatments. In a recent study examining sudden gains in a trial comparing CBT and BA, although sudden gains were associated with better end of treatment outcomes in both treatments, by 18-month follow-up individuals who experienced sudden gains in CBT, compared to BA, had lower depression scores at 18 months post-randomisation (O'Mahen et al., 2021). Future research would benefit from directly comparing different types and intensities of treatment on long-term outcomes, as it may be the case that some forms of treatment more effectively embed the benefits of sudden gains. Further examination of client and therapist factors involved in both inducing and sustaining a sudden gain between treatments can help us to understand how we can utilise sudden change in therapies for

depression. Although no robust client predictors of sudden gains have been found (Aderka et al., 2021; Zilcha-Mano et al., 2019), recent research has found therapist facilitation, for example prompts of reinterpretation of problems (Schilling et al., 2020) and therapist skills (Deisenhofer et al., 2021) may be involved in the instigation of sudden gains. This suggests that a combination of therapist skill and the application of this skill at the right time within a structured treatment may be important to promoting a sudden gain. This also concurs with the revised sudden gains theory, which suggests that treatment factors may alter the slope of natural depression symptom fluctuations to increase the probability of a sudden gain occurring within treatment (Aderka & Shalom, 2021). Although speculative, this may explain the greater incidence of sudden gains in HiCBT where therapists are generally more experienced. It is also of note that sudden gains in this sample were associated with favourable anxiety and functioning treatment outcomes, but these changes were not clinically significant. Previously, Stiles et al. (2003) did not find sudden gains were associated with anxiety or functioning outcomes in a clinic-based sample, however, in their sample not all participants had a primary diagnosis of depression and they received a range of different therapies. Our results suggest that depression sudden gains have additional benefits on symptoms that are commonly associated with depression, but replication is needed.

From previous depression spike research examining their association with treatment outcomes outside of EBCT, it has been unclear whether they are advantageous or result in unfavourable outcomes. Smaller studies have been unable to examine the relationship with depression spikes and treatment outcomes (O'Mahen et al., 2017; O'Mahen et al., 2019) and another study found depression spikes were unrelated to treatment outcomes (Abel et al., 2014). In this study

depression spikes were generally associated with favourable outcomes, consistent with studies of the impact of depression spikes on outcomes at treatment end in EBCT (Grosse Holtforth et al., 2012; Hayes, Feldman, Beevers, et al., 2007; Holtforth et al., 2014), but there was substantial variability in depression outcomes in those who had a spike, with a relatively high proportion deteriorating. This result concurs with a recent RCT study which suggested depression spikes may be associated with negative treatment outcomes (O'Mahen et al., 2021). It is possible these differences could reflect subgroups amongst those who experience a depression spikes, in terms of the causes of spikes, for example recurrent external negative life stressors or intense, productive processing in therapy. Although further replication is needed, the majority of research looking at depression spikes outside EBCT has examined the occurrence with outcome in CBT (Abel et al., 2014; O'Mahen et al., 2021) and BA (O'Mahen et al., 2017; O'Mahen et al., 2019) and the current study extends this to look at group treatment and counselling.

Few studies have examined moderators of sudden gains in clinic-based samples, and none have examined moderators of depression spikes in non-exposure based therapies. In studies of sudden gains most have failed to find evidence of moderation (Shalom & Aderka, 2020). In the current study out of the five moderators explored, only baseline depression, anxiety and functioning severity moderated the association between sudden gains or depression spikes and treatment outcomes. Our results are consistent with Wucherpfennig, Rubel, Hollon, et al. (2017) who found better outcome effects for those who experienced a sudden gain and had higher intake symptom severity in a clinic-based sample. It is possible that individuals with higher scores on clinical measures at intake have greater potential to experience improvement on these measures, thus making any benefits

of discontinuous change easier to detect statistically. Our findings regarding outcomes for people who experienced a depression spike but had low symptom scores at baseline was somewhat counterintuitive. It is possible this is a spurious result and these findings need to be replicated in other samples.

The present research has some limitations to note. While we used a consistent definition of sudden gains across all four treatments in the current study, we, like others (O'Mahen et al., 2021; Wucherpfennig, Rubel, Hollon, et al., 2017) used a criterion that differed from Tang and DeRubeis' (1999) original sudden gains criterion to include very early and very late sudden gains. Shalom and Aderka (2020) found altered sudden gains criteria from Tang and DeRubeis' original criterion yielded significantly greater effects on treatment outcomes. Similarly, the depression spikes criterion used in this study is consistent with other research of depression spikes in non-exposure based studies (O'Mahen et al., 2021; O'Mahen et al., 2019) but differed from Hayes et al.'s (2007) original criterion which required spikes to return in the same phase of therapy, rather than within a session limit like the current study. However, another study has found altering the definition did not change either the rate of depression spikes or their relationship to outcome (O'Mahen et al., 2021). Some caution should still be used when comparing the rates and effects sudden gains and depression spikes to studies that used the original or other altered criterion. Further, integral to the nature of both sudden gains and depression spikes is a decrease in depression scores. Although we constrained the sample to those who had five or more sessions the average number of treatment sessions was relatively low compared to that in RCT samples. This means that the depression score drop may have had a strong influence on the outcome variables, as the final session of the sudden gain or depression spike was likely to be close in time to the

final treatment session. IAPT does not routinely follow up clients and therefore we could only examine short term treatment outcomes, but follow up would allow us to establish if the long term benefits of these patterns of change are similar to RCT findings. Finally, the non-randomised nature of the dataset means that generalised conclusions cannot be drawn about differences between treatments in terms of rates of sudden gains and depression spikes and their relationship to treatment outcomes.

Despite these limitations, this study has important implications for regular clinical services. While it is known that sudden gains repeatedly lead to beneficial treatment outcomes, should these results be replicated in other clinic based samples they also suggests that depression spikes are important because of their association with positive therapy outcomes. Although additional research examining whether and which clinician behaviours make a difference in harnessing the positive effects of discontinuous change is needed, our findings suggest that therapists should be alert to the positive prognostic value of discontinuous change. Further research should also investigate whether therapists can improve client outcome by maximising effective thinking, behaviours and emotional states during and after these changes.

In conclusion, the positive associations of sudden gains and depression spikes on clinical outcomes observed in trials were replicated in a large, clinic-based sample. Associations between sudden gains and depression spikes and outcome were consistent across treatments, whereas rates of sudden gains and depression spikes were not. This suggests experiencing discontinuous depression symptom change is beneficial and perhaps we should focus on whether and why discontinuous change is more likely to in some treatments rather than others. Replications of these findings in other large cohorts with longer term follow up are needed, as is further investigation of how client, therapist and treatment factors can

enhance these patterns of change. Ultimately information from this line of research may shed light on ways to improve treatment outcomes in therapies for depression.

Chapter Three: Windows into the process of change in therapy: an examination of cognitive and behavioural processes surrounding sudden gains in cognitive behavioural therapy (CBT) and behavioural activation (BA)

Study 2

Asha Ladwa [a], Kim Wright [a], Adele Hayes [b], Leigh Andrews [b], Elizabeth Alpert [b], David Richards [c], Heather O'Mahen [a]

[a] Mood Disorders Centre, University of Exeter, Washington Singer Building, Exeter, EX4 4QG, UK

[b] University of Delaware, 226 Wolf Hall Newark, DE 19716 302-831-0484

[c] Medical School, University of Exeter, St Luke's Campus, Exeter, EX1 2LU

3.1 Preface

The findings of the previous study (chapter two) demonstrated that sudden gains in naturalistic settings occur at similar rates, timing, and are associated with beneficial treatment outcomes across therapies, as is seen across the literature in RCT settings (Shalom & Aderka, 2020). The study in this chapter builds upon these findings and a recent study by O'Mahen et al. (2021) to examine therapeutically important client cognitive and behavioural processes that might be involved in the instigation of sudden gains and the association with treatment outcomes in CBT and BA.

Currently it is unclear whether cognitive processes instigate a sudden gain in CBT, as Tang and DeRubeis' (1999) hypothesis suggests, but also whether cognitive processes would instigate sudden gains in therapies that proscribe cognitive strategies, like BA. Further, given that both CBT and BA utilise behavioural strategies it is important to examine whether behavioural processes may bring about sudden gains. In a recent study O'Mahen et al. (2021) found differences between the associations between sudden gains in CBT and BA and depression treatment outcomes. The study found individuals who had a sudden gain, compared to those who did not, had significantly lower depression scores (PHQ-9) at 6-, 12- and 18-months post-treatment. However, at 6- and 18-months outcome, individuals who experienced a sudden gain in CBT had significantly lower PHQ-9 depression scores than individuals who had a sudden gain in BA. The current study builds upon these findings using the same dataset as O'Mahen et al. (2021) and used a psychotherapy process coding system, the 'Change and Growth Experiences Scale' (CHANGE; Hayes, Feldman, & Goldfried, 2007), to rate theoretically important cognitive and behavioural processes over a sudden gain in both therapies. The study examined

whether any of these cognitive or behavioural processes were associated with a sudden gain and differ between treatments. To further examine why there may be differences between treatments at outcome, the study assesses whether these cognitive and behavioural processes a trigger the hypothesised ‘upward spiral’ (Tang & DeRubeis, 1999) following a sudden gain and whether they are differentially related to treatment outcomes at 12- and 18-months follow up in CBT and BA.

The main body of this chapter consists of a paper that is currently being prepared for publication and the intention is to submit to the Journal of Consulting and Clinical Psychology.

3.2 Abstract

Background: This study examined client cognitive and behavioural processes that preceded and followed a sudden gain in cognitive behavioural therapy (CBT) and behavioural activation (BA). How these processes related to 12- and 18-month treatment outcomes and whether these processes varied between treatments was also assessed.

Method: Data from a randomised controlled trial comparing the effectiveness of CBT and BA for adult Major Depressive Disorder (MDD) were used. Participants were a subsample of 50 (*n* = 25 CBT) individuals who experienced a sudden gain. A yoked control group was created from 50 individuals who did not experience a sudden gain, and were matched by treatment modality, baseline depression score, and sudden gain session number to individuals who experienced a sudden gain. Sessions before (pregain) and after (postgain) the sudden gain, and the control sessions were rated for client accommodation, overgeneralisation, avoidance and positive behaviour.

Results: Pregain processes did not predict whether individuals had a sudden gain or not, in either treatment. Sudden gains were associated with reduced avoidance in the postgain session, but this was not moderated by treatment type. In individuals without a sudden gain, pregain accommodation and positive behaviour in BA was negatively associated, and postgain accommodation in CBT was positively associated with depression at 18 months. For those who experienced a sudden gain greater postgain overgeneralisation in BA, compared to CBT, was associated with worse depression at 18 month outcome.

Conclusions: This study did not replicate previous research suggesting that cognitive processes facilitate sudden gains in CBT, nor did we find this was the case

in BA, or that behavioural processes predicted sudden gains. Instead, focusing on the session following a sudden gain may allow therapists to implement treatment strategies to maximise the benefits of sudden gains in both treatments. The findings highlight the need to examine client processes and treatment outcomes for those who do not have a sudden gain, who subsequently have worse depression outcomes. Suggestions for future research and clinical implications are discussed.

Keywords: sudden gain, cognitive behavioural therapy, behavioural activation, client process

3.3 Introduction

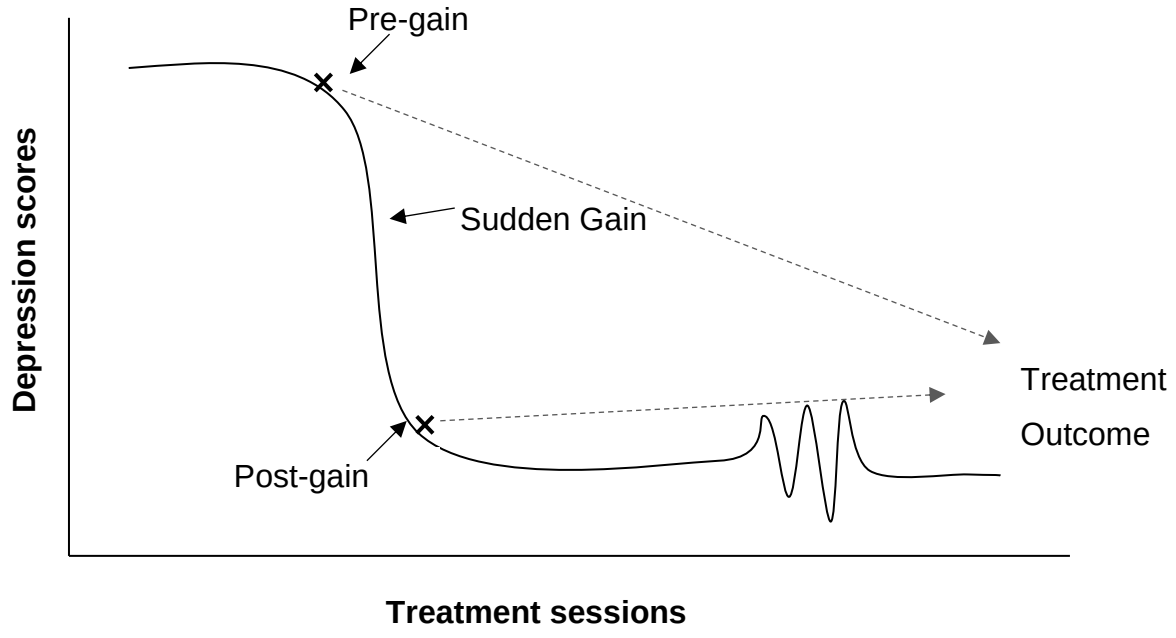
As the world's single largest contributor to global disability, depression is a significant public health concern (WHO, 2017). There is evidence that psychological interventions can effectively reduce depression symptoms and the risk of relapse (Cuijpers et al., 2011), however only around 50-60% of individuals will achieve clinical remission following treatment (Cuijpers et al., 2014; Hollon & Ponniah, 2010). Knowing how treatments work could help to improve the efficiency and efficacy of psychological interventions. For example, two widely used treatments for depression, cognitive behavioural therapy (CBT) and behavioural activation (BA) have similar levels of efficacy (Richards et al., 2016), but it is unclear if they operate through similar or unique mechanisms. Understanding this may help to pinpoint which change processes are most effective at reducing depression symptoms.

One way to examine the mechanisms through which treatments work is to focus on periods of symptom instability during therapy that are associated with positive clinical outcomes and then examine factors associated with these shifts and treatment outcomes. Periods of discontinuous change in depression symptoms are hypothesised to represent critical transition points during therapy, where old patterns of thinking and behaviours are disrupted and new, more adaptive thoughts and behaviours are utilised (Hayes & Andrews, 2020; Hayes, Laurenceau, et al., 2007). One such symptom discontinuity that has been widely studied is a 'sudden gain' (Tang & DeRubeis, 1999) (see schematic in Figure 3.1). Often occurring early in treatment, sudden gains are rapid improvements of depression symptoms that occur between consecutive therapy sessions, defined as a reduction that is (1) large in absolute terms, (2) represents a reduction of symptoms which is at least 25%, and (3) is stable (three sessions following the gain are significantly lower than the three

sessions preceding the gain, assess using a t-test)(Tang & DeRubeis, 1999). Sudden gains in depression symptoms are robustly associated with better end-of-treatment and longer-term outcomes in both CBT (Abel et al., 2016; Andrews et al., 2020; Tang & DeRubeis, 1999; Tang et al., 2005) and BA (Hunnicutt-Ferguson et al., 2012; Masterson et al., 2014; O'Mahen et al., 2017; Singla et al., 2019) for depression. However, the research that has examined which factors might be associated with the onset of sudden gains is mixed, and less research has examined processes that follow a sudden gain, as well as which processes before and after a sudden gain might be associated with long-term depression outcomes. The focus of the current study was therefore to examine whether key adaptive and maladaptive cognitive and behavioural processes hypothesised to be central to each of the treatments preceded and followed sudden gains, as well as whether they predicted treatment outcomes in a comparative trial of CBT and BA.

Figure 3. 1

Schematic of Sudden Gains and Relation of Factors Associated with Outcome



Sudden gains were originally proposed to test the cognitive mediation hypothesis in CBT. This hypothesis posits that depression change in CBT operates through changes in cognitions (Beck, Rush, Shaw, & Emery, 1979). Tang and DeRubeis (1999) proposed a three-stage model of how cognitive change supports both sudden gains and lasting improvements in depression symptoms. In the 'preparation stage', cognitive strategies are taught but little cognitive change occurs. In the second 'critical session' stage, cognitive changes occur and are hypothesised to lead to a sudden improvement (gain) in depression symptoms. The last 'upward spiral' stage occurs when the client, as a result of depression symptom improvements, forms a closer working relationship with the therapist and experiences further cognitive change and depression improvement. This theory suggests that important treatment processes both precede and follow sudden gains, and that these processes may relate directly with short and long term treatment outcomes (see Figure 3.1). Research has tended to focus on the processes associated with the onset of sudden gains in the hope that treatments can be improved to facilitate more of these beneficial periods of depression symptom instability. In their seminal study, Tang and DeRubeis' (1999) conclusions supported their sudden gains model; greater cognitive changes measured on the Patient Cognitive Change Scale (PCCS) both preceded and followed sudden gains. However, subsequent examinations of whether cognitive change occurs prior to a sudden gain has been mixed. Some research has found changes in hope (Abel et al., 2016), anxiety related cognitive change (Norton et al., 2010), and cognitive change measured on the PCCS in depressed individuals (Tang et al., 2005) between the prepregain (two sessions prior to the gain) and the pregain (session immediately prior to the drop in depression scores) occurs prior to a sudden gain in CBT.

However, others have failed to find change in cognitive processes prior to sudden gains in CBT. Changes in cognitive emotional processing (perspective shifts and meaning-making) in individuals with treatment resistant depression receiving CBT adjunct to pharmacotherapy (Abel et al., 2016), change in cognitive processes measured on the PCCS in individuals receiving CT for depression (Ryan, 2013), and change in the degree of belief in cognitions in those receiving CT or interpersonal therapy (IPT) for social anxiety disorder (Bohn et al., 2013), did not precede having a sudden gain. Further, Vittengl et al. (2005) did not find reductions of dysfunctional attitudes predicted sudden gains in a sample of depressed individuals receiving CT, and in group based CBT for phobia, cognitive changes did not precede sudden gains (Hofmann et al., 2006). Methodological differences between studies assessing cognitive changes make it difficult to compare across studies. As highlighted by Aderka and Shalom (2021) observer rating methods compared to self-reports of cognitive changes, measuring cognitive change during compared to prior the therapy session, and the use of different sudden gains criteria, all make comparisons between studies difficult. Further, rather than directly predicting sudden gains these studies compare cognitive change from the pregain session to two sessions prior to the sudden gain or a control session. Although cognitive changes may or may not precede a sudden gain, it is unclear whether they have a direct effect on experiencing a sudden gain. It is also the case that sudden gains occur outside CBT (Shalom & Aderka, 2020), in therapies that proscribe cognitive strategies like BA (Hunnicutt-Ferguson et al., 2012; Masterson et al., 2014) yet little research has looked at whether cognitive changes precede sudden gains in therapies that do not use cognitive change strategies. In addition, although it is possible for cognitive change processes to occur in the absence of cognitive change strategies (Lorenzo-

Luaces et al., 2015), it may alternatively be the case that non-cognitive processes are associated with the onset of a sudden gain. Because CBT and BA are closely related treatments, and CBT contains some of the behavioural therapeutic change procedures also found in BA, it is also important to understand the extent to which behavioural change procedures may also be related to inducing a sudden gain, both independently of cognitive change procedures (BA) and in conjunction with cognitive change procedures (CBT).

Compared to the literature examining processes associated with sudden gains, relatively few studies have looked at the processes which occur following a sudden gain, as well as their relation to treatment outcomes (Figure 3.1). This is despite the potential clinical implications of being able to harness the benefits of a sudden gain within therapy. Tang and DeRubeis (1999), as well as a recently revised theory of sudden gains (Aderka & Shalom, 2021), hypothesise that following a sudden gain increases in therapeutic alliance and further cognitive processes create a positive upward spiral which helps to embed and maintain depression symptom improvements. Some research supports this hypothesis and has found experiencing a sudden gain results in increases in positivity within the therapeutic relationship (Lutz et al., 2013; Wucherpfennig, Rubel, Hofmann, et al., 2017), and that postgain therapeutic alliance moderates the relationship between a sudden gain and treatment outcomes (Wucherpfennig, Rubel, Hofmann, et al., 2017). Contrary to this, a recent study found therapeutic alliance scores were high both prior to and after a sudden gain in depressed individuals receiving CBT (Lemmens et al., 2021), suggesting that change in alliance may not result from a sudden gain but that good therapeutic alliance may help improvements in other domains. There has been a dearth of research examining cognitive processes that follow a sudden gain. One

study by Bohn et al. (2013) found following a sudden gain in individuals with social anxiety disorder receiving CT or interpersonal therapy, there were reductions in the frequency and belief of negative cognitions compared to the pregain session. These cognitive changes following a sudden gain were not moderated by treatment modality. It is has not yet been examined whether cognitive changes occur following a sudden gain in individuals with a primary presenting problem of depression, and if this differs between CBT and BA. Another gap in the literature relates to whether specific cognitive and/or behavioural processes prior to or following a sudden gain are involved in sustaining sudden gain benefits in the long-term, and whether this differs between CBT and BA. In a recent study, O'Mahen et al. (2021) found that although sudden gains in both CBT and BA were associated with lower depression scores at 6-, 12-, and 18-month follow-up, individuals who experienced a sudden gain in CBT had significantly lower depression scores at 6- and 18-months, compared to those who had a sudden gain in BA. It is possible that cognitive processes may promote long-term sudden gain benefits as Tang and DeRubeis (1999) hypothesise, but investigation of other cognitive processes that are therapeutically important targets of CBT is needed. Similarly to processes associated with a sudden gain, investigation of behavioural processes that might sustain a sudden gain in the longer term in CBT and BA is needed.

The current study focuses on four client processes in relation to sudden gains in CBT and BA. We examine whether two cognitive processes, accommodation and overgeneralisation, and two behavioural processes, positive behaviour and avoidance, are associated with the onset of a sudden gain, and long term treatment outcomes, as well as treatment differences in each of these associations between CBT and BA.

Accommodation occurs when pre-existing negative beliefs are modified to reflect more balanced and healthy beliefs, and is the end result of cognitive-emotional processing (perspective shifts and meaning making). There is evidence that sudden gains are associated with increases in cognitive emotional processing in the postgain session compared to the pregain session, but within this study changes in cognitive emotional processing were not found to precede sudden gains in CBT (Abel et al., 2016). However, in individuals with mixed anxiety and depression in psychotherapy, increases in processing were found to precede sudden gains (Adler et al., 2013). Outside the sudden gains literature, cognitive emotional processing is also associated with improved depression outcomes in exposure-based CBT for depression (Grosse Holtforth et al., 2012; Hayes, Feldman, Beevers, et al., 2007). However, more sustained cognitive changes (i.e. accommodation) have been shown to mediate the relationship between immediate cognitive change occurring during the therapy session and session-to-session symptom change (Schmidt et al., 2019). With regards to sudden gains it is unclear if reaching more balanced and healthy beliefs is one mechanism that might instigate drops in depression symptoms, or if accommodation may be a factor that facilitates the 'upward spiral' and lasting depression improvements.

Additionally a significant body of work demonstrates that overgeneralised thinking is linked to both the onset and maintenance of depression and that it underlies other key cognitive thinking errors (Beck et al., 1979). Overgeneralised thinking describes the tendency to draw negative conclusions that become exaggerated and applied broadly across unrelated contexts and/or to ones self-worth. One aim in CBT is to modify such thinking errors. Overgeneralisation has been found to predict both concurrent (Carver & Ganellen, 1983; Ganellen, 1988;

Weeks et al., 2017) and prospective depression up to six weeks later, suggesting it is a key cognitive vulnerability for depression (Carver, 1998). In a sample of depressed adolescents who received CBT, Shirk et al. (2013) found those who experienced greater reductions in overgeneralisation subsequently experienced more symptom improvement. Importantly, improvements in depressive symptoms did not predict future changes in cognitions, suggesting overgeneralisation may be an important mediating factor. However, this key cognitive factor has not been investigated in the context of sudden gains in CBT. It follows that reductions in overgeneralisation may facilitate rapid depression drops, but it is also possible that following sudden gains reduced overgeneralisation may be associated with positive outcomes.

While accommodation and overgeneralisation represent key cognitive targets of change in CBT, in both CBT and BA individuals may experience cognitive change as a consequence of engaging in new behaviours (Lorenzo-Luaces et al., 2015). Only one study has examined cognitive processes in relation to sudden gains in BA, and found baseline dysfunctional cognitive style did not predict having a sudden gain in BA (Hunnicutt-Ferguson et al., 2012). However, no research has examined within-therapy cognitive processes in relation to sudden gains in BA. Because BA does not engage clients in direct cognitive change procedures, whereas CBT does, these processes might be more relevant to therapeutic change in CBT than in BA, both in predicting sudden gain status and treatment outcome.

With regards to the potential behavioural processes associated with a sudden gain and treatment outcomes, behavioural theory posits that change in BA occurs when clients actively engage in positively-reinforced behaviours when faced with negative mood and the urge to avoid, creating an upward spiral of positive

behaviours and mood improvement (Carvalho & Hopko, 2011). A recent study found clients preparation to engage in alternative positive behaviours in CT for depression increased following a sudden gain (Lemmens et al., 2021). However, other behavioural therapy processes such as deciding to increase or making plans for pleasurable activities, engaging in a wide range of activities, or structured daily activities were not found to change from the pregain to postgain session, nor were there any changes in behavioural processes prior to the sudden gain (between a control session and the pregain session). Little other research has examined other therapeutically important targets in BA in the context of sudden gains. In line with behavioural theory, in BA lower levels of avoidance and more positive behaviours should be associated with both experiencing a sudden gain, and fewer depressive symptoms at treatment outcome. Further, it is possible that these behavioural processes may partly explain the benefits of sudden gains in CBT, especially in the early stages of CBT where there is a behavioural focus (Beck et al., 1979).

3.3.1 The Current Study and Hypotheses.

In this study, we sought to compare these four processes (accommodation, overgeneralisation, avoidance and positive behaviour) in relation to sudden gains and treatment outcome in a non-inferiority, randomised controlled trial (RCT) of CBT and BA for adults with Major Depressive Disorder; MDD (Richards et al., 2016). We sought to determine the following in this data: (1) which processes predict sudden gains, (2) whether sudden gains were associated higher levels of positive change in these processes postgain, (3) if processes before and following the sudden gain predicted depression 12- and 18-months treatment outcome, and (4) if these relationships varied by treatment condition. To examine these questions, we used the Change and Growth Experiences Scale (CHANGE; Hayes, Feldman, &

Goldfried, 2007), a transtheoretical coding system of client processes to code therapy sessions prior to and following a sudden gain in individuals with and without sudden gains in CBT and BA.

When examining which process predicted sudden gains, we hypothesised that greater accommodation and positive behaviours ('adaptive processes'), and less overgeneralisation and avoidance ('maladaptive processes') would predict having a sudden gain. We expected treatment would moderate these relationships, such that accommodation and overgeneralisation would be more likely to be associated with presence of a sudden gain status in CBT, rather than BA, whereas positive behaviour and avoidance would be more likely to be associated with a presence of a sudden gains in BA, rather than CBT.

Next we expected that individuals who had a sudden gain would have higher levels of adaptive processes and lower levels of maladaptive processes following the sudden gain in the postgain session, than matched individuals without a sudden gain, and these would similarly be moderated by treatment as above.

Lastly when examining how the adaptive and maladaptive processes in the pregain and postgain sessions predict treatment outcomes, we expected higher levels of adaptive and lower levels of maladaptive processes before and following the sudden gain would relate to lower depressive symptoms at 12- and 18-month post-randomisation in those who had a sudden gain relative to those who did not. We also explored whether these relationships would be moderated by treatment condition. We expected it would be more likely that there would be a relationship between having a sudden gain and accommodation or overgeneralisation in CBT

than BA, whereas it would be more likely there would be a relationship between have a sudden gain and positive behaviour or avoidance in BA than CBT.

3.4 Method

3.4.1 Data Source: The COBRA Trial

This study is part of a process analysis of the 'Cost and Outcome of Behavioural Activation versus Cognitive Behavioural Therapy for Depression' (COBRA) trial, which was a non-inferiority RCT of BA compared to CBT for adults with MDD (ethical approval reference NRES/07/H1208/60). Adults aged 18 years and over, who met diagnostic criteria for MDD were recruited from primary care and psychological services at three sites in the United Kingdom (UK), and randomly allocated to BA (*n* = 221) and CBT (*n* = 219). Individuals were excluded if they were receiving psychological therapy, were alcohol or drug dependent, were acutely suicidal, cognitively impaired, or who had bipolar disorder or psychotic symptoms. In the original trial 12-months post-randomisation was the primary outcome and 18-months was the follow up time point. The main trial findings are reported by Richards et al. (2016).

3.4.2 Therapy and Therapists

The aim of BA is to disrupt the depression cycle by encouraging re-engagement with positive reinforcing behaviours in the environment, despite negative mood and a learned propensity to avoid. In the trial BA was delivered according to the National Institute for Health and Care Excellence (NICE) guidelines which recommends 16-20 sessions over 3-4 months with two sessions per week for the first 3-4 weeks (NICE, 2009). Therapists administered treatment according to a revised treatment manual of BA (Ekers et al., 2011). The revised manual followed

standard BA treatment as set out by Martell et al. (2001) and also included optional modules that provided further information about applying rumination, communication, and problem-solving strategies in addition to strategies to manage anxiety and find functionally equivalent behaviours within a BA framework.

In the CBT condition, therapists followed a CBT manual based on Beck et al.'s (1979) approach. The treatment focussed on restructuring dysfunctional, depressogenic thoughts and testing these modified thoughts in behavioural experiments in an effort to embed cognitive learning, improve opportunities for reinforcement, and improve mood.

Participants received a maximum of 20 sessions of BA (*M* = 14.3, *SD* = 5.2) or CBT (*M* = 17.1, *SD* = 5.7) over 16 weeks with the option of four booster sessions. Therapy sessions were delivered face-to-face and lasted approximately 60 minutes. Junior mental health workers (MHWs) delivered BA, whereas CBT was delivered by senior MHWs with a diploma (≥ 2 years of study) in CBT.

Prior to trial commencement both CBT and BA therapists received five days training in their respective therapies. Therapist quality was assessed by independent experts who selected a random sample of therapy audiotapes and rated competency. Both MHWs delivering BA and therapists delivering CBT met acceptable competency standards (Richards et al., 2016). To further ensure that therapy was delivered in accordance with the theoretical model in the subsample used within the current study, therapy tapes across the treatment arms were compared in terms of corrective information delivered by the therapist. Therapists in CBT provided more cognitive corrective information (*M* = 1.34, *SE* = .10) than BA (*M* = .42, *SE* = .11), (*F* (1, 77) = 36.327, *p* = <.001, η_p^2 = .321). BA therapists provided

more behavioural corrective information (*M* = 1.74, *SE* = .11) compared to CBT (*M* = 1.08, *SE* = .10), (*F* (1, 77) = 19.628, *p* = <.001, η_p^2 = .203).

3.4.3 Measures

***3.4.3.1 Baseline and Outcome Depression*.** The Patient Health Questionnaire- 9 (PHQ-9; (Kroenke et al., 2001) is a nine item self-report measure of depression severity. The items enquire about anhedonia, low mood, poor appetite, feeling tired, difficulty concentrating, and thoughts of self-harm/suicide. Higher scores indicate greater depression severity. The PHQ-9 is sensitive to detecting change over time (Löwe et al., 2004), and is a valid and reliable measure of depression severity (Kroenke et al., 2001). In the original trial the PHQ-9 was used to measure depressive symptoms at baseline, 6-, 12-, and 18-months post-randomisation.

***3.4.3.2 Weekly Depression Symptoms*.** The Beck Depression Inventory (BDI; Beck et al., 1961) is a widely used 21-item self-report measure of depression symptoms over the previous week. The measure enquires about a range of symptoms including mood, sleep, appetite, self-dislike, guilt and thoughts of suicide. Items are rated on a 0-3 scale with higher scores reflecting greater intensity of the symptom. Scores can range from 0-63, with higher scores indicating greater depressive symptoms. The BDI has good reliability and validity (Beck et al., 1988). In the trial participants completed the BDI at the beginning of each therapy session and this measure was used to identify sudden gains for the current study.

***3.4.3.3 Process Coding*.** Therapy sessions were coded using the Change and Growth Experiences Scale (CHANGE; Hayes, Feldman, & Goldfried, 2007), an observational coding system designed to examine processes of change occurring

within psychotherapy. The CHANGE has good reliability across treatments and disorders (Abel et al., 2016; Alpert et al., 2021; Cummings et al., 2012; Yasinski et al., 2019). Coded variables in the current study included accommodation, overgeneralisation, avoidance and positive behaviour. Descriptions of each process can be found in Table 3.1 and examples of each process are in Appendix 1. All CHANGE variables were rated using a four-point Likert scale from 0 (*not present*) to 3 (*high*), and processes can co-occur. During coding content within the session as well as experiences from the week prior to the therapy session were considered.

Table 3. 1

CHANGE Coding System Variables

Process Variable	Description	ICC
Accommodation	Adapting pre-existing beliefs to incorporate new information in an accurate, adaptive and healthy way	0.71
Overgeneralisation	The over application or exaggeration of depressive beliefs across time (past or future), the self, others, or situations	0.76
Avoidance	Captures efforts to protect/defend self by pulling away rather than moving towards problems or issues, e.g. social withdrawal	0.77
Positive behaviour	Adaptive behaviours an individual engages with between therapy sessions	0.68

Note. ICC = Intra Class Correlation

3.4.4 Procedure

***3.4.4.1 Defining Sudden Gains*.** Sudden gains are large, rapid symptom improvements that occur between consecutive therapy sessions. They were originally defined by Tang and DeRubeis (1999) as (1) a rapid improvement of depressive symptoms by seven or more BDI points between two consecutive sessions, (2) where the magnitude of the gain is equal to at least 25% of the pregain score, and (3) the mean of the three scores preceding the gain are significantly greater than the three scores following the gain, assessed using a *t*-test (Tang & DeRubeis, 1999). The third criterion has received the most criticism in the literature for violating the assumption of independence in a *t*-test and for not being able to detect very early or very late sudden gains where three sessions before or after the symptom drop are not available. The current study used the sudden gains identified in the trial by O'Mahen et al. (2021). They retained the original first and second sudden gains criteria, and in line with Wucherpfennig, Rubel, Hofmann, et al. (2017), the *t* distribution in the third criterion was modified to be able to identify very early or very late sudden gains. Where three sessions were available, Tang and DeRubeis' (1999) original criterion was retained, necessitating a *t* score of at least 2.78 when comparing the mean of three sessions before and after the gain. Where only two sessions before or after the gain was available $t\,(3) \geq 3.18$ was used, and for very early or very late gains $t\,(2) \geq 4.30$ was used. Individuals who exhibited reversals of gains, where 50% or more of the symptom improvement was lost following the gain, or who experienced a gain as part of a depression spike, which is a transient increase in depression symptoms between treatment sessions which then returns (Hayes, Feldman, Beevers, et al., 2007), were excluded.

3.4.4.2 Session Selection. The sample was restricted to participants who attended the per-protocol number of eight therapy sessions (Richards et al., 2016) and completed the BDI in six sessions or more, yielding a sample of 300/400 (75%). A total of 110 (37%) participants experienced a sudden gain in the trial sample (O'Mahen et al., 2021). Therapy recordings from the pregain and postgain sessions were coded. To be selected for coding using the CHANGE system, participants with sudden gains needed to have both session tapes available and to have given consent for their therapy tapes to be used in additional research.

Given the intensive nature of coding audio tapes, 25 participants from each treatment condition (CBT/BA) of those who experienced a sudden gain and 25 yoked matched controls from the same treatment condition were coded, resulting in 100 participants. A random sample with replacement strategy was used to select 25 participants who experienced a sudden gain from each therapy group. The comparison yoked control group was created from participants who did not experience a sudden gain. Participants in the yoked control group were matched to participants who experienced a sudden gain by treatment type (CBT/BA), baseline PHQ-9 band score, and sudden gain session numbers (+/-1 session).

A sensitivity power calculation revealed that a sample size of 100 participants would allow us to detect a small-medium effect (Cohen's d = 0.36). This is smaller than the effect size of d = 0.57 detected by the most similar previous study, which examined processes associated with sudden gains in adults with treatment resistant depression who received CBT (Abel et al., 2016).

***3.4.4.3 Coding and Coders*.** De-identified audio tapes of therapy sessions were coded by a team of four coders who completed three days of training in the

CHANGE manual. Out of 200 tapes total, 78 (39%) tapes were coded by two coders to assess inter-rater agreement and to prevent rater drift. Coders were blind to treatment, sudden gain status, session number, and treatment outcome. Weekly meetings with all coders were held to discuss discrepancies of two or more points on the four-point CHANGE scale for sessions that were double coded. Consensus codes replaced discrepant codes and then all ratings were averaged between the two coders. The inter-rater agreement between coders (intraclass correlations, ICC) for each process ranged from 0.68-0.77 (see Table 3.1) indicating good agreement (Koo & Li, 2016).

3.4.5 Data Analytical Strategy

Statistical analyses were performed using IBM SPSS Statistics version 25 (IBM Corp, 2017). Baseline demographic and clinical characteristic differences between those who did and did not experience a sudden gain were examined using *t*-tests and Chi-square analyses[4].

Logistic regression analyses were conducted to examine whether process variables in the pregain session predicted sudden gain status (0, 1). Prepregain (the session before the pregain session) BDI depression score was entered in the first step as a covariate in order to account for symptom severity just before the sudden gain. Each pregain process variable and treatment (CBT/BA) were entered into step two, and the interaction between the pregain process variable and treatment was entered in the third step.

[4] For additional demographic and baseline clinical characteristic comparisons between the current sample compared to the full trial sample (Appendix 2) and those who experienced a sudden gain but were not coded within the current study (Appendix 3), see the chapter appendices.

Next, to examine how sudden gain status influenced processes following the gain, Analysis of Covariance (ANCOVA) analyses examined whether sudden gain status (0, 1), treatment (CBT/BA) and the interaction between gain status and treatment was associated with postgain processes, controlling for postgain BDI depression score. Postgain BDI score was controlled for in order to examine the influence of the sudden drop in depression scores captured in a sudden gain on postgain process above and beyond the effects of the absolute depression severity score in the pregain session.

To examine how each of the processes at the pregain and postgain sessions predicted outcome at 12- and 18-months post-randomisation a series of hierarchical multiple linear regression analyses were conducted. Separate regression models were conducted for each of the four process variables at the pregain and postgain session on 12- and 18-month outcome. In the first step of each model prepregain BDI score was regressed onto 12- or 18-month PHQ-9 score. In the second step the main effects of the pregain or postgain process variable, sudden gains status (0, 1) and treatment type (CBT/BA) were entered. In the third step, the two-way interactions were entered, and in the final step the three-way interaction between the pregain or postgain process variable, sudden gains status and treatment type were entered. Depression at the prepregain session was controlled for in order to assess the interactions with the process variable at pregain or postgain, sudden gain status and treatment independently from depression severity. Three-way interactions between pregain or postgain processes, treatment type and sudden gain status were firstly explicated by sudden gain status (yes/no) and then by treatment type (CBT/BA). All assumptions were met, except the assumption of homoscedasticity. Visual examination of plots suggested there was heteroscedasticity within the data (a

funnel shape of data points). A Box-Cox transformation was applied to the 12- and 18-month PHQ-9 outcome score. Following this visual examination of a scatter plot with this transformed dependent variable showed a random scatter of data points, suggesting the transformation had corrected the heteroscedasticity and the assumption of homoscedasticity was met.

3.5 Results

3.5.1 Participants

There were no significant differences in age, antidepressant use, baseline PHQ-9 score, number of previous episodes of MDD, gender, relationship status, ethnicity, or education in individuals who experienced a sudden gain compared to those who did not ($p > .05$; Appendix 4). However, participants who experienced a sudden gain had significantly fewer treatment sessions ($M = 14.52$, $SD = 5.41$) than those who did not have a sudden gain ($M = 16.92$, $SD = 5.54$), $t(1, 98) = 2.192$, $p = .031$. The means and standard deviations of the process variables at each time point split by sudden gain status and treatment can be found in Table 3.2 and the correlations between processes are in Table 3.3.

Table 3. 2

Means (Standard Deviations) for Each Process Variable by Sudden Gain Status and Treatment

	Pregain				Postgain			
	Sudden Gain		No Sudden Gain		Sudden Gain		No Sudden Gain	
	CBT	BA	CBT	BA	CBT	BA	CBT	BA
Accommodation	0.3 (0.6)	0.2 (0.4)	0.1 (0.3)	0.2 (0.6)	0.4 (0.7)	0.5 (0.8)	0.2 (0.5)	0.2 (0.4)
Overgeneralisation	0.7 (0.8)	0.7 (0.9)	0.9 (0.9)	0.8 (0.6)	0.7 (0.6)	0.7 (0.9)	1.1 (0.9)	0.7 (0.7)
Avoidance	1.1 (0.9)	1.5 (1.0)	1.5 (0.9)	1.7 (0.9)	0.4 (0.5)	0.9 (0.9)	1.0 (1.0)	1.5 (0.9)
Positive Behaviour	1.5 (0.9)	1.5 (0.9)	1.3 (0.8)	1.3 (0.9)	1.9 (0.9)	1.7 (0.8)	1.3 (0.7)	1.4 (0.9)

Note. CBT = cognitive behavioural therapy; BA = behavioural activation.

Table 3. 3

Correlations between Pregain and Postgain Process Variables

	1.	2.	3.	4.	5.	6.	7.	8.
1. Pregain Accommodation	1	.167	-.103	-.060	-.303**	.001	.485***	.161
2. Postgain Accommodation		1	.002	-.030	.036	-.261**	.183	.462***
3. Pregain Overgeneralisation			1	.356***	.451***	.119	-.121	-.119
4. Postgain Overgeneralisation				1	.242*	.362***	-.114	-.162
5. Pregain Avoidance					1	.309**	-.425***	-.146
6. Postgain Avoidance						1	-.127	-.350***
7. Pregain Positive Behaviour							1	.342**
8. Postgain Positive Behaviour								1

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

3.5.2 Pregain Processes Associated with Sudden Gains

Contrary to expectations, neither adaptive nor maladaptive processes in the pregain session were associated with sudden gain status (Table 3.4). Further, the relationship between adaptive and maladaptive pregain processes and sudden gain status was not moderated by treatment type (Table 3.4).

Table 3. 4

Binary Logistic Regression Analyses Examining the Association between Pregain processes on Sudden Gain Status (Yes/No)

Variable	OR	95% CI
Pregain Accommodation		
Prepregain BDI	0.98	.92, 1.04
Pregain Accommodation	1.67	.69, 4.03
Treatment	0.96	.39, 2.31
Accommodation x Treatment	0.45	.07, 2.77
Pregain Overgeneralisation		
Prepregain BDI	0.98	.92, 1.03
Pregain Overgeneralisation	0.75	.40, 1.40
Treatment	0.89	.36, 2.19
Overgeneralisation x Treatment	0.84	.21, 3.32
Pregain Avoidance		
Prepregain BDI	0.98	.92, 1.03
Pregain Avoidance	0.79	.48, 1.28
Treatment	1.00	.41, 2.41

Avoidance x Treatment	1.31	.48, 3.51
Pregain Positive behaviour		
Prepregain BDI	0.98	.92, 1.03
Pregain Positive Behaviour	1.30	.77, 2.16
Treatment	0.93	.38, 2.25
Positive Behaviour x Treatment	0.83	.30, 2.30

Note. BDI = Beck Depression Inventory.

3.5.3 Sudden Gain Status Associated with Postgain Processes

In line with predictions individuals who experienced a sudden gain (*M* = 0.68, *SD* = 0.80) had significantly lower levels of avoidance in the postgain session than those who did not have a sudden gain at the matched session (*M* = 1.27, *SD* = .96) (Table 3.5). However, treatment modality did not moderate this relationship. There were no other significant relationships between sudden gain status and the other post-gain processes.

Table 3. 5

ANCOVA Comparing Postgain Processes by Sudden Gain and Treatment

Postgain process	*df*	*F*	*p*	η_p^2
DV- Postgain Accommodation				
Postgain BDI	1, 94	0.00	.988	.000
Sudden Gain	1, 94	3.25	.074	.033
Treatment	1, 94	0.02	.904	.000
Sudden Gain x Treatment	1, 94	0.43	.509	.005
DV- Postgain Overgeneralisation				
Postgain BDI	1, 94	3.33	.071	.034
Sudden Gain	1, 94	0.14	.704	.002
Treatment	1, 94	2.31	.132	.024
Sudden Gain x Treatment	1, 94	3.81	.054	.039
DV- Postgain Avoidance				
Postgain BDI	1, 94	0.50	.479	.005
Sudden Gain	1, 94	6.41	.013*	.064
Treatment	1, 94	9.93	.002**	.096
Sudden Gain x Treatment	1, 94	0.03	.854	.000
DV- Postgain Positive behaviour				
Postgain BDI	1, 94	6.51	.012*	.065
Sudden Gain	1, 94	0.44	.504	.005
Treatment	1, 94	0.29	.590	.003
Sudden Gain x Treatment	1, 94	0.62	.430	.007

Note. BDI = Beck Depression Inventory. *$p < .05$. **$p < .01$. ***$p < .001$

3.5.4 Pregain Processes as Predictors of Depression Outcome

Regression analyses showed individuals who had higher pregain accommodation levels had significantly lower depression at 12-months outcome (Table 3.6). This was moderated by treatment type in that individuals who had higher levels of pregain accommodation in BA, compared to CBT, had lower PHQ-9 scores at 12 months outcome (Table 3.7). However, this association was not moderated by sudden gain status. There were no other main effects of pregain adaptive or maladaptive processes on depression outcome at 12- or 18-months, nor were there any other two-way interactions between processes and treatment or sudden gain status on depression outcome (Table 3.6). However, there were significant three-way interactions between pregain process variable, sudden gain status, and treatment for pregain accommodation and pregain positive behaviour on 18-month outcome (Table 3.6).

Explications of the three-way interaction between pregain accommodation, sudden gain status, and treatment on 18-month PHQ-9 outcome indicated there was a pregain accommodation by treatment interaction for individuals who did not have a sudden gain (Table 3.7). There was no pregain accommodation by treatment interaction for those who experienced a sudden gain. Individuals who did not have a sudden gain in BA and who had higher levels of pregain accommodation had significantly lower PHQ-9 depression scores at 18 month treatment outcome, than their counterparts in CBT (Figure 3.2).

Table 3. 6

Regression Analyses Examining Pregain Client Processes as Predictors of 12- and 18-Month PHQ-9 Outcome

	12 Month PHQ-9						18 Month PHQ-9					
	B(*se*)	*t*	*p*	95% CI	R^2 adj	$R^2\Delta$	*B*(*se*)	*t*	*p*	95% CI	R^2 adj	$R^2\Delta$
Pregain Accommodation												
Step 1					.061	.07*					.06	.07*
Prepregain BDI Score	0.34(.01)	2.44	.017*	.006, .062			.034(.01)	2.39	.019*	.006, .062		
Step 2					.19	.17**					.09	.07
Sudden Gain	-.635(.20)	-3.133	.003**	-1.040, -.231			-.463(.21)	-2.165	.034*	-.889, -.036		
Treatment	-.069 (.20)	-.338	.736	-.472, .335			-.076(.21)	-3.53	.725	-.503, .352		
Pregain Accommodation	-.409(.19)	-2.067	.042*	-.803, -.015			-.136(.21)	-.656	.514	-.547, .276		
Step 3					.22	.05					.11	.05

Sudden Gain x Treatment	.041(.40)	.103	.919	-.762, .845			.336 (.43)	.787	.434	-.516, 1.188		
Pregain	-.908(.43)	-2.096	.040*	-1.772, -.044			-.699(.45)	-1.569	.121	-1.588, .190		
Accommodation x Treatment												
Pregain	-.595(.43)	-1.376	.173	-1.456, .267			.208(.44)	.470	.640	-.675, 1.188		
Accommodation x Sudden Gain												
Step 4					.23	.02					.15*	.05
Pregain	1.230	1.426	.158	-.491, 2.952			1.854(.87)	2.125	.037*	.112, 3.595		
Accommodation x	(.86)											
Treatment x Sudden Gain												
Pregain Overgeneralisation												
Step 1					.06	.07*					.06	.07*

Prepregain BDI Score	0.34(.01)	2.44	.017*	.006, .062			.034(.01)	2.39	.019*	.006, .062		
Step 2					.15	.12*					.13	.10*
Sudden Gain	-.664(.21)	-3.174	.002**	-1.081, -.247			-.455(.21)	-2.176	.033*	-.871, -.038		
Treatment	-.052(.21)	-.242	.809	-.477, .374			-.008(.21)	-.038	.970	-.435, .419		
Pregain Overgeneralisation	.081(.15)	.528	.599	-.225, .387			.274(.15)	1.780	.079	-.033, .581		
Step 3					.13	.02					.1	.01
Sudden Gain x Treatment	.155(.44)	.358	.722	-.712, 1.022			.211(.43)	.488	.627	-.652, 1.074		
Pregain Overgeneralisation x Treatment	.319(.35)	.912	.365	-.379, 1.017			-.032(.34)	-.096	.924	-.708, .643		
Pregain Overgeneralisation x Sudden Gain	.216(.31)	.693	.491	-.406, .839			-.236(.32)	-.745	.459	-.868, .396		
Step 4					.12	.00					.13	.04

Pregain Overgeneralisation x Treatment x Sudden Gain	-.073(.71)	-.104	.918	-1.487, 1.340			1.245(.68)	1.837	.071	-.108, 2.598		
Pregain Avoidance												
Step 1					.06	.07*					.06	.07*
Prepregain BDI Score	0.34(.01)	2.44	.017*	.006, .062			.034(.01)	2.39	.019*	.006, .062		
Step 2					.15	.12*					.09	.06
Sudden Gain	-.685(.21)	-3.261	.002**	-1.104, -.266			-.472(.22)	-2.198	.031*	-.899, -.044		
Treatment	-.074(.21)	-.352	.726	-.490, .343			-.088(.22)	-.407	.685	-.519, .343		
Pregain Avoidance	-.024(.12)	-.203	.840	-.264, .215			.028(.124)	.227	.821	-.220, .276		
Step 3					.12	.01					.11	.06
Sudden Gain x Treatment	.086(.43)	.199	.843	-.773, .944			.412(.43)	.962	.340	-.443, 1.267		
Pregain Avoidance x Treatment	.139(.25)	.564	.574	-.352, .630			.275(.25)	1.109	.271	-.220, .770		

Pregain Avoidance x Sudden Gain	-.105(.25)	-.428	.670	-.594, .384			-.407(.25)	-1.638	.106	-.902, .089		
Step 4					.14	.03					.11	.01
Pregain Avoidance x Treatment x Sudden Gain	-.738(.49)	-1.520	.133	-1.707, .231			-.523(.49)	-1.058	.294	-1.510, .464		
Pregain Positive Behaviour												
Step 1					.06	.07*					.06	.07*
Prepregain BDI Score	0.34(.01)	2.44	.017*	.006, .062			.034(.01)	2.39	.019*	.006, .062		
Step 2					.15	.13*					.09	.07
Sudden Gain	-.659(.21)	-3.147	.002**	-1.076, -.241			-.499(.21)	-2.329	.023*	-.926, -.072		
Treatment	-.069(.21)	-.330	.743	-.484, .346			-.093(.21)	-.433	.667	-.520, .335		
Pregain Positive Behaviour	-.078(.12)	-.649	.518	-.319, .162			.095(.13)	.756	.452	-.155, .344		
Step 3					.13	.01					.12	.06

Sudden Gain x Treatment	-.030(.43)	-.071	.943	-.881, .821			.343(.43)	.806	.423	-.506, 1.192		
Pregain Positive Behaviour x Treatment	.055(.25)	.222	.825	-.437, .547			-.339(.25)	-1.363	.178	-.835, .157		
Pregain Positive Behaviour x Sudden Gain	.246(.25)	.993	.324	-.249, .741			.383(.25)	1.527	.131	-.118, .884		
Step 4					.16	.04					.20	.08**
Pregain Positive Behaviour x Treatment x Sudden Gain	.930(.49)	1.918	.059	-.038, 1.898			1.332(.48)	2.792	.007**	.380, 2.285		

Note. R^2 adj = adjusted R Squared; PHQ-9 = Patient Health Questionnaire 9; BDI = Beck Depression Inventory.

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

Table 3. 7

Explications of Interactions for Linear Regression on 12- and 18-Month Outcome

	12 month PHQ-9 outcome				18 month PHQ-9 outcome			
	B(*se*)	*t*	*p*	95% CI	*B*(*se*)	*t*	*p*	95% CI
Pregain Interactions								
Pregain Accommodation x treatment								
Main effect of Accommodation for CBT	-.071(.27)	.297	.791	-.613, .470				
Main effect of Accommodation for BA	-.648(.28)	-2.287	.029*	-1.226, - .071				
Pregain Accommodation x Treatment x Sudden Gain								
Pregain Accommodation x treatment for sudden gain					.142 (.48)	.296	.769	-.831, 1.114
Pregain Accommodation x treatment for no sudden gain					-1.809 (.73)	-2.487	.018*	-3.289, -3.29

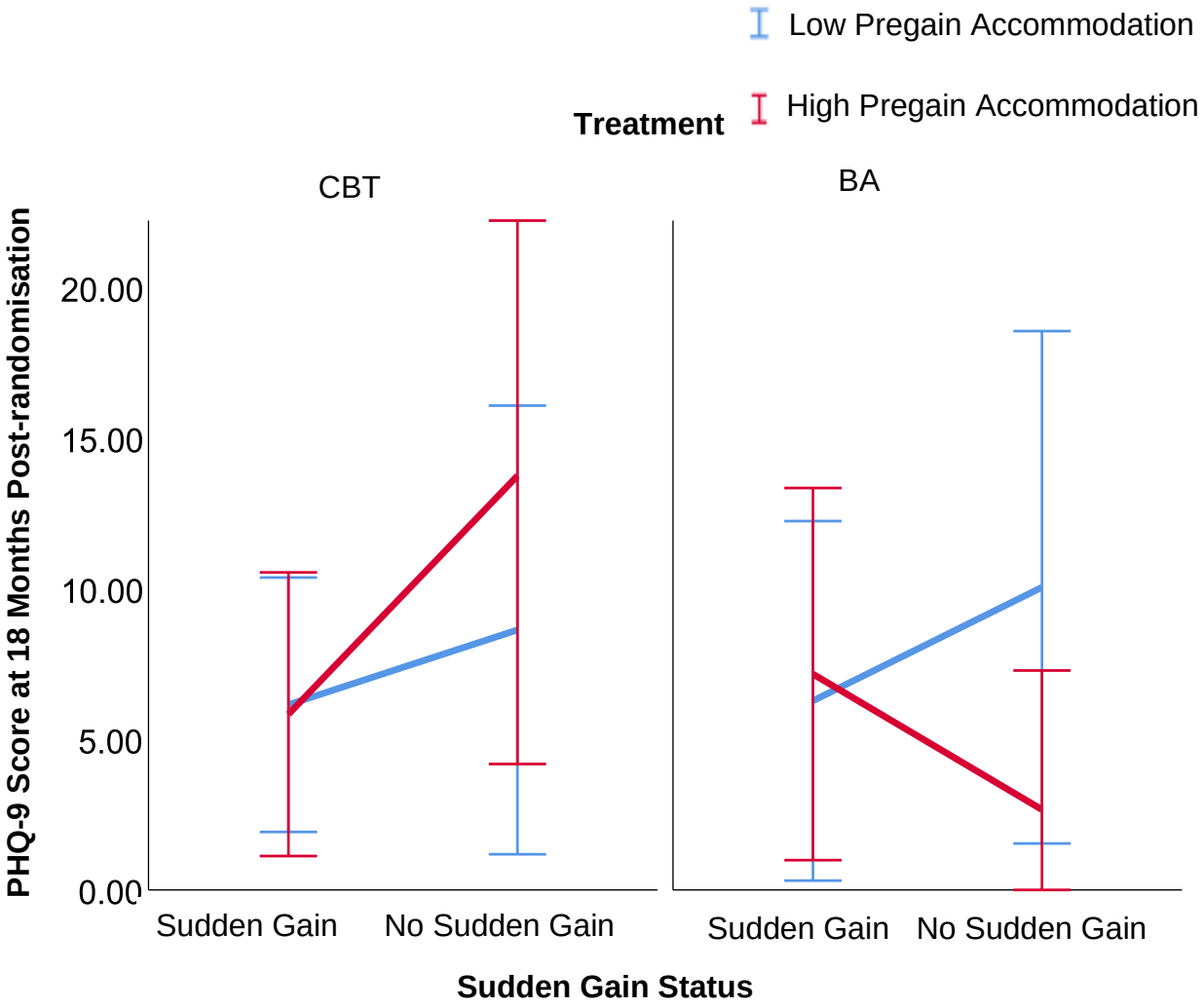
Main effect of accommodation for no sudden gain in CBT	.905(.66)	1.367	.160	-.492, 2.301
Main effect of accommodation for no sudden gain in CBT	-.900(.36)	-2.462	.026*	-1.679, -.121
Pregain positive behaviour x SG x Treatment				
Positive behaviour x treatment for sudden gain	.215(.25)	.857	.398	-.295, .725
Positive behaviour x treatment for no sudden gain	-1.076(.41)	-2.614	.013*	-1.914, -.239
Main effect of positive behaviour for no sudden gain in CBT	.486(.34)	1.412	.176	-.240, 1.213
Main effect of positive behaviour for no sudden gain in BA	-.596(.26)	-2.311	.035*	-1.145, -.046
Postgain Interactions				

Postgain avoidance x treatment				
Main effect of avoidance for CBT	-.069(.17)	-.398	.693	-.422, .284
Main effect of avoidance for BA	.445(.17)	2.659	.012*	.104, .785
Postgain accommodation x SG x treatment				
Postgain accommodation x treatment for sudden gain	.274(.29)	.941	.353	-.318, .866
Postgain accommodation x treatment for no sudden gain	-2.543(.95)	-2.665	.012*	-4.485, -.602
Main effect of postgain accommodation for no sudden gain in CBT	1.799(.72)	2.485	.024*	.271, 3.326
Main effect of postgain accommodation for no sudden gain in BA	-.734(.63)	-1.158	.265	-2.086, .617

Postgain overgeneralisation x SG x treatment				
Postgain overgeneralisation x treatment for sudden gain	.986(.31)	3.223	.003**	.363, 1.608
Postgain overgeneralisation x treatment for no sudden gain	-.516(.61)	-.844	.405	-1.759, .727
Main effect of postgain overgeneralisation for sudden gain in CBT	-.552(.24)	-2.331	.032*	-1.051, -.052
Main effect of postgain overgeneralisation for sudden gain in BA	.638(.17)	3.838	.002**	.283, .992

Figure 3. 2

Pregain Accommodation on 18-months Post-randomisation Split by Treatment Type and Sudden Gains Status



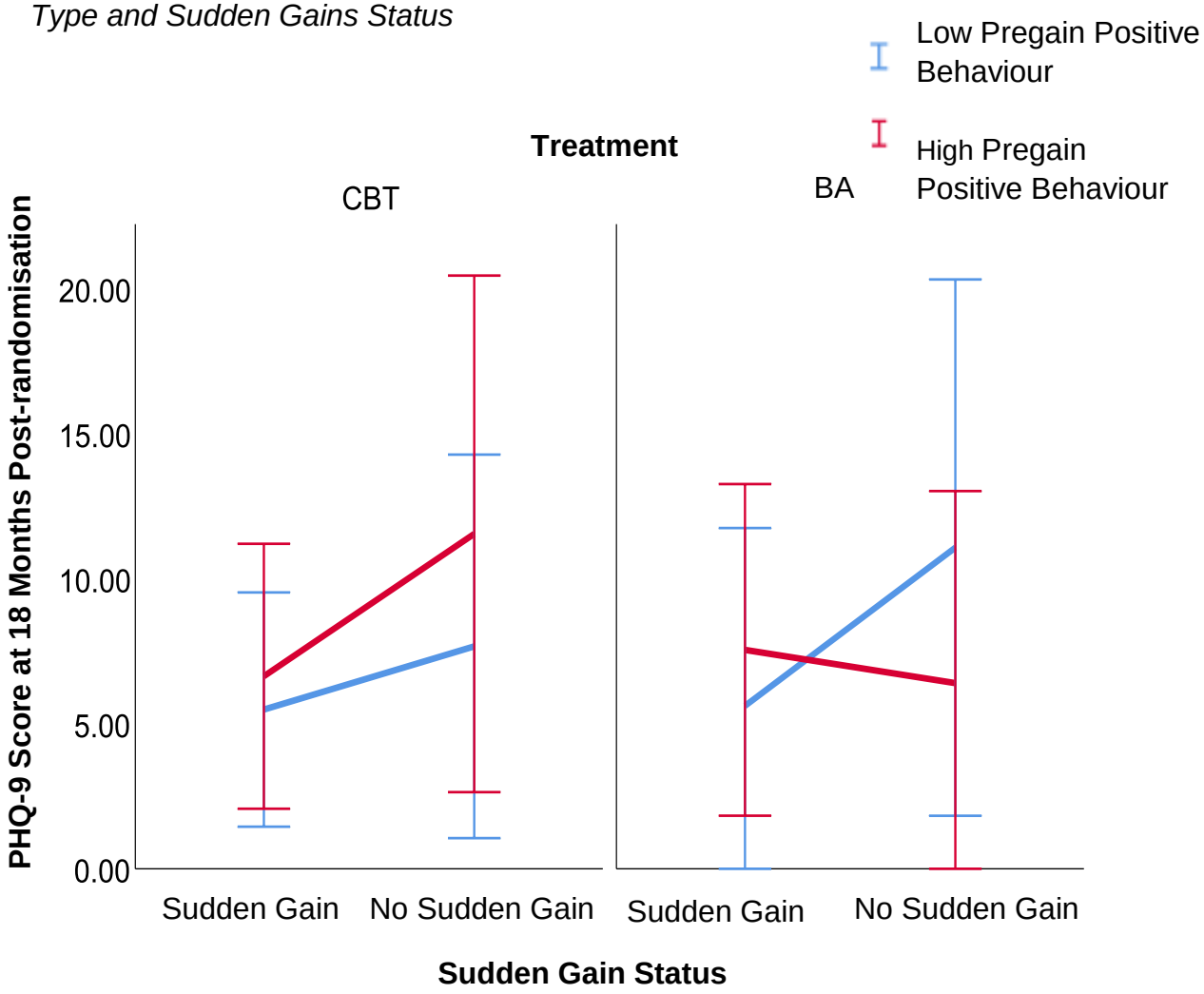
Note. PHQ-9= Patient Health Questionnaire 9; CBT = cognitive behavioural therapy, BA = behavioural activation; Standard deviation error bars displayed.

Explications of the three-way interaction between pregain positive behaviour, sudden gain status and treatment type on 18-month outcome showed there was a significant pregain positive behaviour by treatment interaction for those who did not have a sudden gain (Table 3.7). There was no pregain positive behaviour by treatment interaction for those who experienced a sudden gain. Individuals who did not experience a sudden gain in BA, compared to CBT, and who had higher levels of

positive behaviour in the pregain session, had significantly lower PHQ-9 scores at 18-months outcome (Figure 3.3). There were no other significant three-way interactions for pregain processes.

Figure 3. 3

Pregain Positive Behaviour on 18-Months Post-randomisation Split by Treatment Type and Sudden Gains Status



Note. PHQ-9= Patient Health Questionnaire 9; CBT = cognitive behavioural therapy, BA = behavioural activation; Standard deviation error bars displayed.

3.5.5 Postgain Processes as Predictors of Depression Outcome

Within the postgain session individuals who had greater accommodation and positive behaviour had significantly lower PHQ-9 scores at 12-month outcome (Table 3.8). There were no other main effects of postgain adaptive or maladaptive processes. A significant two-way interaction between postgain avoidance and treatment showed greater levels of avoidance in BA, compared to CBT, was associated with higher PHQ-9 depression scores at 18 months outcome (Table 3.7). This relationship was not moderated by sudden gain status, and there were no other two-way interactions on 12- or 18-month PHQ-9 outcome (Table 3.8). However, there were 2 three-way interactions between postgain process, sudden gains status and treatment for postgain accommodation and overgeneralisation (Table 3.8).

Table 3. 8

Regression Analyses Examining Postgain Client Processes as Predictors of 12 and 18 Month PHQ-9 Outcome

	12 Month PHQ-9						18 Month PHQ-9					
	B(se)	t	p	95% CI	R^2 adj	$R^2\Delta$	B(se)	t	p	95% CI	R^2 adj	$R^2\Delta$
Postgain Accommodation												
Step 1					.06	.07*					.06	.07*
Prepregain BDI Score	.034 (.01)	2.442	.017*	.006, .062			.034(.01)	2.394	.019*	.006, .062		
Step 2					.22	.18**					.09	.07
Sudden Gain	-.526(.21)	-2.520	.014*	-.942, -.110			-.431(.22)	-1.928	.058	-.876, .015		
Treatment	-.042(.20)	-.208	.836	-.442, .358			-.067(.22)	-.310	.758	-.496, .362		
Postgain Accommodation	-.404(.16)	-2.463	.016*	-.732, -.077			-.114(.17)	-.679	.500	-.451, .222		
Step 3					.21	.03					.08	.03
Sudden Gain x Treatment	.248(.42)	.589	.558	-.592, 1.088			.463(.45)	1.021	.311	-.443, 1.370		

Postgain Accommodation x Treatment	-.488(.33)	-1.476	.145	-1.147, .172			-.280(.34)	-.817	.417	-.964, .404		
Postgain Accommodation x Sudden Gain	-.404(.43)	-.942	.349	-1.260, .451			-.464(.47)	-1.019	.312	-1.373, .445		
Step 4					.22	.02					.19	.11**
Postgain Accommodation x Treatment x Sudden Gain	1.202(.89)	1.346	.183	-.580, 2.984			2.868(.89)	3.194	.002**	1.076, 4.661		
Postgain Overgeneralisation												
Step 1					.06	.07*					.06	.07*
Prepregain BDI Score	.034 (.01)	2.442	.017*	.006, .062			.034(.01)	2.394	.019*	.006, .062		
Step 2					.15	.12*					.09	.07
Sudden Gain	-.665(.21)	-3.147	.002**	-1.086, -.244			-.455(.21)	-2.122	.037*	-.883, -.027		
Treatment	-.056(.22)	-.257	.798	-.489, .378			-.043(.22)	-.195	.846	-.482, .396		

Postgain Overgeneralisation	.051(.15)	.337	.737	-.252, .354			.106(.14)	.736	.464	-.181, 3.93		
Step 3					.12	.002					.07	.01
Sudden Gain x Treatment	.054(.45)	.121	.904	-.835, .942			.244(.46)	.531	.597	-.672, 1.160		
Postgain Overgeneralisation x Treatment	.068(.35)	.193	.847	-.632, .767			.309(.34)	.904	.369	-.373, .991		
Postgain Overgeneralisation x Sudden Gain	.097(.34)	.282	.779	-.590, .784			-.119(.34)	-.346	.730	-.807, .569		
Step 4					.11	.01					.12	.06*
Postgain Overgeneralisation x Treatment x Sudden Gain Postgain Avoidance	.516(.70)	.735	.465	-.885, 1.918			1.468(.67)	2.189	.032*	.129, 2.807		
Step 1					.06	.07*					.06	.07*
Prepregain BDI Score	.034 (.01)	2.442	.017*	.006, .062			.034(.01)	2.394	.019*	.006, .062		

Step 2					.19	.16**					.09	.09
Sudden Gain	-.527(.22)	-2.422	.018*	-.961, -.093			-.421(.23)	-1.836	.071	-.878, .036		
Treatment	-.199(.21)	-.933	.354	-.624, .226			-.127(.23)	-.564	.574	-.575, .321		
Postgain Avoidance	.238(.13)	1.912	.060	-.010, .487			.086(.13)	.656	.514	-.175, .347		
Step 3					.18	.03					.15	.09
Sudden Gain x Treatment	-.089(.49)	-.181	.857	-1.067, .889			.707(.50)	1.415	.162	-.290, 1.705		
Postgain Avoidance x Treatment	.151(.27)	.561	.576	-.385, .686			.678(.27)	2.498	.015*	.136, 1.220		
Postgain Avoidance x Sudden Gain	.331(.29)	1.116	.268	-.260, .922			.040(.29)	.134	.894	-.556, .636		
Step 4					.17	.001					.14	.001
Postgain Avoidance x Treatment x Sudden Gain Postgain Positive Behaviour	.181(.64)	.280	.780	-1.108, 1.470			.143(.65)	.219	.827	-1.157, 1.443		
Step 1					.06	.07*					.06	.02*
Prepregain BDI Score	.034 (.01)	2.442	.017*	.006, .062			.034(.01)	2.394	.019*	.006, .062		

Step 2					.23	.19**						
Sudden Gain	-.495(.21)	-2.361	.021*	-.913, -.077			-.427(.22)	-1.904	.061	-.873, .020	.09	.07
Treatment	-.092(.19)	-.463	.645	-.489, .304			-.081(.21)	-.378	.707	-.508, .346		
Postgain Positive Behaviour	-.340(.13)	-2.661	.010*	-.596, -.085			-.099(.14)	-.709	.481	-.376, .179		
Step 3					.22	.03					.07	.02
Sudden Gain x Treatment	.131(.43)	-.306	.761	-.986, .723			.396(.46)	.856	.395	-.527, 1.318		
Postgain Positive Behaviour x Treatment	-.114(.27)	-.418	.677	-.657, .430			-.285(.29)	-.972	.334	-.870, .300		
Postgain Positive Behaviour x Sudden Gain	-.418(.27)	-1.501	.138	-.973, .137			-.085(.29)	-.287	.775	-.677, .506		
Step 4					.21	.001					.09	.04
Postgain Positive Behaviour x Treatment x Sudden Gain	.181(.55)	.326	.746	-.925, 1.286			1.013(.58)	1.754	.084	-.140, 2.166		

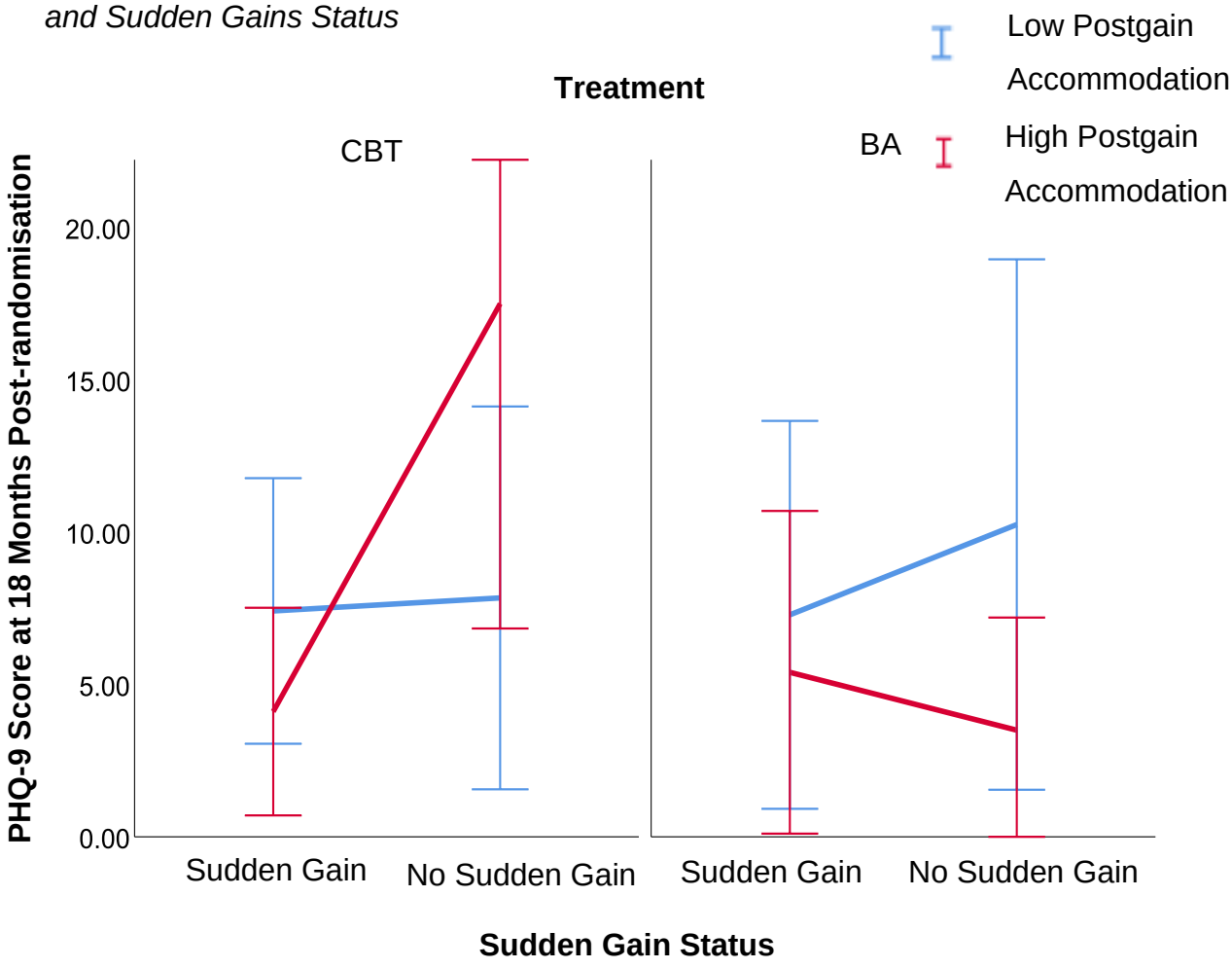
Note. R^2 adj = adjusted R squared; PHQ-9 = Patient Health Questionnaire 9; BDI = Beck Depression Inventory.

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

Explications of the three-way postgain accommodation interaction by sudden gains status indicated that for individuals who did not experience a sudden gain there was a significant treatment by postgain accommodation interaction, but not for individuals who experienced a sudden gain (Table 3.7). Further explications of the two-way interaction for those who did not experience a sudden gain demonstrated that there was a significant positive relationship between postgain accommodation and PHQ-9 outcome at 18-months in individuals who received CBT. This relationship was not significant in those who received BA. Individuals who had higher levels of accommodation in the postgain session in CBT, but did not experience a sudden gain, had higher PHQ-9 scores at 18-month outcome (Figure 3.4).

Figure 3. 4

Postgain Accommodation on 18 months Post-randomisation Split by Treatment Type and Sudden Gains Status



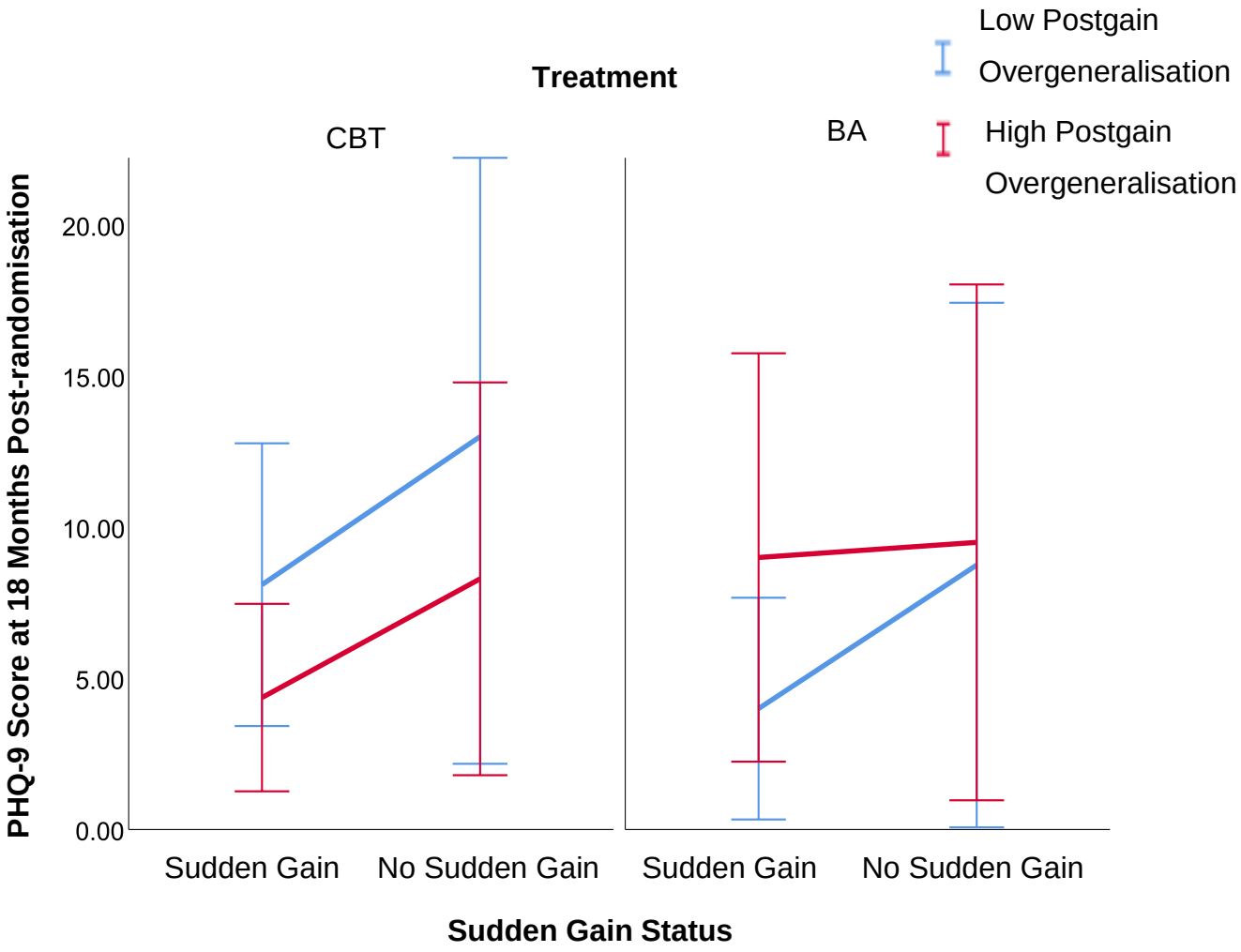
Note. PHQ-9= Patient Health Questionnaire 9; CBT = cognitive behavioural therapy, BA = behavioural activation; Standard deviation error bars displayed.

The explication of the three-way interaction between sudden gains status, postgain overgeneralisation and treatment indicated there was no significant interaction between treatment and overgeneralisation in individuals who did not have a sudden gain, but there was a significant two-way interaction in those who had a sudden gain (Table 3.7). Within CBT participants who had a sudden gain and higher levels of postgain overgeneralisation reported lower PHQ-9 scores at 18-months

(Table 3.7). However for BA participants who had a sudden gain, more postgain overgeneralisation was associated with significantly higher PHQ-9 scores at 18 months post-randomisation (Figure 3.5).

Figure 3. 5

Postgain Overgeneralisation on 18-Months Post-randomisation Split by Treatment Type and Sudden Gains Status



Note. PHQ-9= Patient Health Questionnaire 9; CBT = cognitive behavioural therapy, BA = behavioural activation; Standard deviation error bars displayed.

3.6 Discussion

In a large dataset from a trial comparing CBT and BA for MDD we examined key client processes that preceded and followed sudden gains, and assessed how they related to depression outcomes. To our knowledge this is the first study to directly compare client cognitive and behavioural processes preceding and following sudden gains in CBT and BA.

Contrary to our predictions neither accommodation, overgeneralisation, positive behaviour, nor avoidance in the pregain session were associated with experiencing a sudden gain. Our findings are in contrast to Tang and DeRubeis' (1999) hypothesis that cognitive processes, such as more accommodation and lowered overgeneralisation, drive sudden gains in treatment. Further, treatment condition did not moderate these effects. Thus, we did not find any support for the idea that cognitive processes might be more strongly related to the onset of a sudden gain in treatments that directly target these processes in their change procedures, as in CBT, versus BA which explicitly does not include cognitive change procedures. To our knowledge this is the first study to examine cognitive process in the pregain session as direct predictors of sudden gain status. The current literature examines *change* in cognitive processes prior to a sudden gain and some find change in cognitive processes precedes a sudden gain in CBT (e.g., Abel et al., 2016; Tang & DeRubeis, 1999) whereas other research fails to find a prospective relationship between change in cognitive processes and sudden gains (Bohn et al., 2013; Kelly et al., 2005; Lemmens et al., 2021). There maybe two possible reasons for the discrepancies between the previous literature and our study. By not examining change in cognitive process and instead examining the absolute level of process prior to a sudden gain, it is possible we have examined trait levels of these

processes. Alternatively, the type of cognitive process examined in this study may explain why we did not find an association between cognitive process and sudden gain. In Tang and DeRubeis' (1999) original sudden gains study they used the PCCS which examines seven categories of cognitive change, whereas in the current study the CHANGE examines broader cognitive processes. It may be that granularity in defining these processes matters. Beyond cognitive processes, neither positive behaviour nor avoidance predicted having a sudden gain, nor did we find treatment moderated these associations. This is despite therapy adherence checks suggesting that more cognitive corrective information was delivered by the therapists in CBT, whereas more behavioural corrective information was provided in BA.

The lack of association between client processes and sudden gains is not uncommon within the literature (Aderka & Shalom, 2021) and research also fails to find robust demographic and clinical characteristic predictors of sudden gains (Aderka et al., 2021; Zilcha-Mano et al., 2019). While it is possible that processes such as therapist effects (Deisenhofer et al., 2021) or interactions between client and/or therapist variables may be involved in the generation of a sudden gain, recent research shows symptom fluctuations predict sudden gains (Shalom et al., 2018; Shalom et al., 2020). A recently revised theory suggests sudden gains may result from natural fluctuations of depression symptoms (both in and outside of treatment), but within the context of active treatment sudden gains can be harnessed and lead to better treatment outcomes (Aderka & Shalom, 2021). Taking into account the findings of the current study and previous research (Aderka et al., 2021; Wucherpfennig, Rubel, Hofmann, et al., 2017; Zilcha-Mano et al., 2019), this suggests that rather than exploring the factors that bring about a sudden gain within treatment we should focus on what happens in clients following a sudden gain, as

this is where therapists may be able to employ strategies to maximise the upward spiral and enhance the clinical benefits of a gain.

In line with this we found partial support for our hypotheses about the relationship between experiencing a sudden gain and postgain processes. Experiencing a sudden gain was associated with lower levels of postgain avoidance, compared to the matched session of those who did not experience a sudden gain. It is possible that reductions in maladaptive processes, like avoidance, following a sudden gain may help to facilitate other processes and ultimately the upward spiral of further cognitive processes and therapeutic alliance discussed in sudden gains theories (Aderka & Shalom, 2021; Tang & DeRubeis, 1999). We also found greater postgain avoidance in BA, but not CBT, was associated with higher depression scores at 18 months outcome, but without the sudden gain moderation this is hard to interpret and may be a test of general therapeutic processes and outcome differences between CBT and BA. Levels of the other adaptive and maladaptive processes were also not found to differ in the postgain session. While this might suggest that change in accommodation, overgeneralisation, or positive behaviour does not occur following a sudden gain in CBT or BA the sessions examined in the current study were only a ‘snapshot’ of therapy. Other research examining processes of change following a sudden gain have found increases in both postgain therapeutic alliance and coping skills, and coping skills continued to increase in the sessions following the sudden gain (Wucherpfennig, Rubel, Hofmann, et al., 2017). Thus it is possible that continued cognitive and/or behavioural change may occur within two or three sessions following the sudden gain. Future research replicating and extending this may further highlight what occurs following a sudden gain.

With regards to how processes were associated with treatment outcomes, pregain accommodation and positive behaviour, and postgain accommodation and overgeneralisation were related to treatment outcomes. Unexpectedly, all but one of the results were for individuals who *did not* have a sudden gain, rather than for individuals who did have a sudden gain. Interestingly, all the three-way interactions were associated with longer term outcome at 18- rather than 12- months. Although further replication is needed, this suggest that other process research should also examine long-term consequences of within therapy client processes. The findings for those who did not have a sudden gain are difficult to interpret and perhaps gives more of an insight into non-systematically selected treatment sessions, which in the current study were not chosen at random but were yoked to the timing of the sudden gains in the comparison group. However, they do represent a window into the relationship between therapy process and treatment outcomes for a point in time that happens to vary across participants. We found greater levels of pregain accommodation and pregain positive behaviour in individuals who do not have a sudden gain in BA was associated with lower depression scores at 18 months outcome. In the postgain session greater accommodation in those who did not have a sudden gain in CBT was associated with higher PHQ-9 scores at 18 months. These findings are potentially an interesting insight into how therapies might differ in the relationship between client processes, therapy content, and outcome. Considering the association between greater pregain positive behaviour and accommodation, and better longer term outcomes in those without a sudden gain who received BA, it is possible that behavioural techniques allow patients to capitalise on these positive processes. Examining baseline and weekly levels of process may also give us an understanding of whether therapy helps cultivate these

processes or individuals enter therapy with high levels of these positive processes. If it is the latter this may suggest, consistent with a capitalisation model of treatment (Rude & Rehm, 1991), that BA utilises individuals' pre-existing strengths. Tailoring treatment to harness patients strengths in CBT have been found to lead to more favourable treatment outcomes in a depressed sample, compared to reducing deficits and compensating (Cheavens et al., 2012). Further theoretically driven research is needed to determine whether these processes during therapy are associated with treatment outcomes, and whether treatment can be personalised to an individuals' strengths to improve treatment outcomes. Given that we found no difference in this relationship for individuals who had a sudden gain, it is possible that this personalisation may be particularly important for individuals who do not show sudden early improvements.

The only process that influenced depression outcomes for individuals who experienced a sudden gain was levels of postgain overgeneralisation. Greater levels of postgain overgeneralisation in individuals who experienced a sudden gain in BA were associated with higher depression scores, whereas in CBT higher postgain overgeneralisation was associated with lower depression scores at 18-months outcome. While this remains tentative until replicated in other samples, it may suggest that there are client processes following a sudden gain, such as overgeneralisation, that therapists should be alert to and which would indicate the optimal therapeutic strategies to deliver postgain. It is possible that engaging in cognitive techniques in the face of overgeneralisation may enhance the benefits of a sudden gain. This result may also partially explain treatment differences at follow up in another study using this sample. Individuals in CBT who had a sudden gain were found to have significantly lower depression scores at 18-month follow-up than those

who had a sudden gain in BA (O'Mahen et al., 2021). Together with that study, these results suggest it may be important to increase strategies that focus supporting clients to challenge negative overgeneralised thinking styles following a sudden gain.

3.6.1 Strengths and Limitations

Within the context of a large RCT we were able to directly compare client processes using a consistent definition of sudden gains within CBT and BA. The CHANGE coding system captures a range of processes allowing us to explore previously unstudied variables in relation to sudden gains and treatment outcome. However, only content that is verbalised and discussed within the therapy sessions can be coded and other processes may be missed. Future work using a range of measures, including self-report measures, may highlight factors that are not observable during therapy sessions. We also note that specific therapeutic procedures were not coded and therefore we cannot assess whether therapy procedures or events outside of therapy influenced client processes. Most of the findings associated with treatment outcomes involve processes observed in those who do not have a sudden gain. While this might give us insight into what is happening for these individuals, the results must be interpreted with caution as they are capturing client processes occurring in a randomly matched session to individuals who experienced a sudden gain. Additionally it is of note that no statistical method was used to match individuals who did and did not experience a sudden gain. Statistical methods such as ‘propensity score matching’ (PSM) can be used to balance two samples on a range of baseline characteristics (Rosenbaum & Rubin, 1983) and to ensure an optimal nearest-neighbour matching. Despite not using a PSM method in the current study a comparison of baseline characteristics between

those who did and did not have a sudden gain showed there were no significant differences between the groups.

With 100 participants, our sample size exceeds those used in the majority of studies examining processes in relation to symptom change. However, sensitivity analyses suggest that still only a small-to-medium effect could be detected and therefore we may have been unable to detect small differences between therapies, or those with and without sudden gains. From our study we are not able to accept the null hypothesis with confidence. Thus, where we did not find associations between sudden gains status, treatment and particular client processes, these remain worthy of future investigation.

Additionally, although we selected a subset of CHANGE variables for analysis and made a-priori predictions, our analyses include multiple comparisons and therefore the chances of type I error were elevated. We did not correct for multiple testing because this is the first study to examine a variety of client processes at different points of a sudden gain across BA and CBT simultaneously; as such we intend this research to contribute towards theory-building and the generation of predictions for future testing. While this is also in line with previous process sudden gains research (Abel et al., 2016; Bohn et al., 2013; Tang & DeRubeis, 1999) there is the potential for elevated false positive results.

3.6.2 Conclusions and Implications

The results of this study did not identify any cognitive or behavioural client processes that preceded a sudden gain in either CBT or BA; instead, findings suggest it may be valuable to focus upon the immediate aftermath of a sudden gain. Should our findings be replicated, they have important theoretical and clinical

implications. They do not accord with the cognitive mediation hypothesis of sudden gain occurrence and add to the debate as to whether cognitive processes are necessary for a sudden gain to occur in CBT, and a non-cognitive therapy, BA. The findings do however, lend partial support to the upward spiral hypothesis. Within clinical settings it may be beneficial to heighten therapists' awareness of overgeneralised negative thinking following a sudden gain. For individuals who do not experience a sudden gain, it may be important to capitalise on client's strengths, particularly with respect to building upon the client's new perspectives (accommodation) using behavioural techniques. The exploratory nature of this study and its observational design, however, means that these suggestions remain tentative until confirmed by future research. Nevertheless, this study furthers the literature examining client processes over this robust pattern of depression change.

Chapter three appendices

Appendix 1

Examples of the 'Change and Growth Experiences Scale' (CHANGE; Hayes, Feldman, & Goldfried, 2007) Coding System Variables

Process Variable	Example of Process Variable
Accommodation	Example of level 3 accommodation: "I used to think that I wasn't doing enough, but over the last couple of months my perceptions of others has changed and now I have stopped worrying if I am doing enough, because I know I am doing enough. I feel better about my relationships and I used to think other people's actions were my fault, but I am letting that go. "
Positive behaviour	Example of level 3 positive behaviour: "I did it! I finally had that conversation with my ex-husband that I was avoiding for the last couple of months. Even though it was awful and horrible, it was a big, positive step for me."
Overgeneralisation	Example of level 3 overgeneralisation: "When I procrastinate and waste time I am being lazy and wasting my life away. If I can't even control my day to day life, how can I be capable of doing bigger things in life like taking my life further and moving forward?"

Avoidance	Example of level 3 avoidance : “I always have to put on a happy face at work and I have to mask my feelings when I feel upset, stressed or tense… this happens every day and I tend to go into my office and sit away from people to avoid having to put on a happy face.”

Appendix 2

Patient Demographics and Clinical Characteristics Comparing Participants who Experienced Sudden Gains and were Coded, and the Whole COBRA Sample*

	Sudden gains (*n* = 50)			Whole COBRA sample (*n* =183)						
Variable	*n* (%)	*M*	*SD*	*n* (%)	*M*	*SD*	χ^2	*t*	*df*	*p*
Treatment							.011		1	1.000
CBT	25 (50%)			93 (50.8%)						
BA	25 (50%)			90 (49.2%)						
Age (years)	50	44.86	14.99	183	44.04	14.15		-.359	231	.720
Site							2.712		2	.255
Devon	22 (44%)			63 (34.4%)						
Durham	18 (36%)			63 (34.4%)						
Leeds	10 (20%)			57 (31.2%)						
Antidepressant use							.682		1	.453
Yes	36 (72%)			142 (77.6%)						
No	14 (28%)			41 (22.4%)						

Baseline PHQ-9	50	17.320	4.377	183	17.481	4.923		.209	231	.834
Number of previous MDD episodes	41	4.342	5.620	155	3.839	4.312		-.621	194	.535
Sex							.214		1	.738
Female	34 (68%)			118 (64.5%)						
Male	16 (32%)			65 (35.5%)						
Relationship status							1.441		4	.821
Not in a relationship	10 (20%)			50 (27.3%)						
In a relationship	40(80%)			133 (72.7%)						
Ethnicity							4.873		6	.571
Caucasian	49 (98%)			177 (96.7%)						
Other	1 (2%)			6 (3.3%)						
Education							3.183		7	.890
No qualifications	3 (6%)			22 (12%)						
Secondary School	30 (60%)			107 (58.5%)						
Degree	17 (34%)			54 (29.5%)						

Note. CBT= cognitive behavioural therapy; BA= behavioural activation; PHQ-9 = Patient Health Questionnaire 9; MMD = Major Depressive Disorder. *Individuals who had sudden gains and were not coded were removed before examining differences in demographics and baseline clinical characteristics

Appendix 3

Patient Demographics and Clinical Characteristics Comparing Participants Who Experienced Sudden Gains and Were Coded, Compared to Those Who Experienced Sudden Gains and Were Not Coded

	Coded Sudden Gains (*n* = 50)			Non-coded Sudden Gains (*n* = 67)						
Variable	*N* (%)	*M*	*SD*	*N* (%)	*M*	*SD*	χ^2	*t*	*df*	*p*
Treatment							.520		1	.574
CBT	25 (50%)			38 (56.7%)						
BA	25 (50%)			29 (43.3%)						
Age (years)	50	44.86	14.994	67	46.239	14.307		-.505	115	.614
Antidepressant use							2.161		1	.174
Yes	36 (72%)			56 (83.5%)						
No	14 (28%)			11 (16.5)						
Baseline PHQ-9	50	17.320	4.377	67	17.597	4.809		-.320	115	.749
Number of previous MDD episodes	41	4.342	5.62	54	2.778	2.944		1.754	93	.083
Number of sessions	50	14.52	5.407	67	15.54	5.541		-.993	115	.323

Sex			.186	1	.698
Female	34 (68%)	43 (64.2%)			
Male	16 (32%)	24 (35.8%)			
Relationship status			3.331	4	.516
Not in a relationship	10 (20%)	16 (23.9%)			
In a relationship	40 (80%)	51 (76.1%)			
Ethnicity			5.075	5	.458
Caucasian	49 (98%)	65 (97%)			
Other	1 (2%)	2 (3%)			
Education			3.076	7	.920
No qualifications	3 (6%)	6 (9%)			
Secondary School	30 (60%)	35 (52.2%)			
Degree	17 (34%)	26 (38.8%)			

Note. CBT= cognitive behavioural therapy; BA= behavioural activation; PHQ-9 = Patient Health Questionnaire 9; MMD = Major Depressive Disorder.

Appendix 4

Comparison of Demographic and Clinical Characteristics

	Sudden Gain			No Sudden Gain						
Variable	*n* (%)	*M*	*SD*	*n* (%)	*M*	*SD*	χ^2	*t*	*df*	*p*
Treatment										
CBT	25 (25%)			25 (25%)			.000		1	1.000
BA	25 (25%)			25 (25%)						
Age (years)		44.86	14.99		44.50	13.05		-0.128	98	.898
Antidepressant use							.208		1	.820
Yes	36			38						
No	14			12						
Baseline PHQ-9		17.32	4.377		17.38	4.681		.066	98	.947
Number of previous	41	4.34	5.62	43	6.12	6.573		1.327	82	.188
MDD episodes										
Number of sessions		14.52	5.407		16.92	5.543		2.192	98	.031*
Sex							.794		1	.504

Female	34	38			
Male	16	12			
Relationship status			3.481	3	.332
Not in a relationship	21	24			
In a relationship	29	26			
Ethnicity			3.941	3	.211
White	49	50			
Other	1	0			
Education			2.388	6	.904
No qualifications	3	7			
Secondary School	30	29			
Degree	17	14			

Note. CBT = cognitive behavioural therapy; BA = behavioural activation; PHQ-9 = Patient Health Questionnaire; MDD = Major Depressive Disorder. * p <.05, ** p <.010, *** p <.001

Chapter Four: An investigation of processes associated with depression spikes and treatment outcomes in cognitive behavioural therapy (CBT) and behavioural activation (BA).

Study 3

Asha Ladwa [a], Heather O'Mahen [a], Adele Hayes [b], Leigh Andrews [b], Elizabeth Alpert [b], David Richards [c], Kim Wright [a]

[a] Mood Disorders Centre, University of Exeter, Washington Singer Building, Exeter, EX4 4QG, UK

[b] University of Delaware, 226 Wolf Hall Newark, DE 19716 302-831-0484

[c] Medical School, University of Exeter, St Luke's Campus, Exeter, EX1 2LU

4.1 Preface

This study builds upon study one (chapter two) to examine therapeutically important client and therapist variables occurring prior to and during a depression spike. This study aimed to elucidate what depression spikes represent in therapies which do not have intended therapeutic procedures to deliberately bring them about. The findings from study one demonstrated that depression spikes in a large naturalistic, clinic-based dataset are associated with favourable treatment outcomes in four therapies (low- and high-intensity cognitive behavioural therapy, counselling and group treatment) for depression. This is in line with the original depression spikes research which examined them in exposure based cognitive therapy (EBCT), a therapy which deliberately instigates a depression spike to allow processing of depression content to occur, for depression (Hayes, Feldman, Beevers, et al., 2007). It is still unclear why depression spikes occur in therapies outside EBCT and whether they are associated with depression treatment outcome, and in the limited literature to date outside EBCT treatments their association with treatment outcomes have been mixed (e.g. Abel, 2014; O'Mahen et al., 2021) .

This study utilised data from the Cost and Outcome of Behavioural Activation and Cognitive Behavioural Therapy for depression (COBRA) trial, a non-inferiority, randomised controlled trial (RCT) conducted at three sites in the United Kingdom (UK). The original trial examined the cost and clinical effectiveness of BA compared to CBT and found that BA was as clinically effective as CBT, but was more cost effective (Richards et al., 2016). In a recent study using the COBRA trial data, O'Mahen et al. (2021) examined the rates, timings and influence of depression spikes on depression outcome at 6-, 12- and 18-months follow up. They found 86 (29%) of individuals experienced a depression spike and there were no differences

between rates or timing of depression spikes between CBT and BA. Baseline behavioural activation, dysfunctional cognitions, relationship status, education status, or gender were not found to be associated with depression spikes in either treatment. Contrary to expectations, depression spikes were associated with higher dimensional depression scores (PHQ-9) at 6-, 12- and 18-month follow up, compared to individuals who did not experience a depression spike. At 6- and 12-month follow up there was no treatment moderation, but at 18-months individuals who experienced a depression spike in CBT had non-significantly higher PHQ-9 depression scores, compared to their counterparts in BA. When examining the association between depression spikes and categorical SCID outcomes, individuals who experienced a depression spike, compared to those who did not, were significantly more likely to meet SCID MDD criteria. Similarly to the dimensional results, those who experienced a depression spike in CBT were 23% more likely to meet SCID MDD criteria at 18-month follow up, than their counterparts in BA.

The current study aimed to examine whether important therapeutic client processes and therapist strategies are associated with both the onset of a depression spike and later treatment outcomes in CBT and BA. The main body of this chapter consists of a paper that is currently being prepared for publication and the intention is to submit to the Journal of Affective Disorders.

4.2 Abstract

Background: Research has shown that depression spikes (Hayes, Feldman, Beevers, et al., 2007), a transient increase of depression symptoms that subsequently decrease, are associated with long-term (18 month) treatment outcomes in cognitive behavioural therapy (CBT) and behavioural activation (BA) (O'Mahen et al., 2021). However, depression spikes in these treatments are not well understood, in terms of what processes lead to a spike and are associated with treatment outcomes. This study examined whether cognitive and behavioural, client processes and therapist strategies and life events were associated with a depression spike and treatment outcomes (12-month) and follow up (18-month) in CBT and BA.

Method: Data from a non-inferiority randomised controlled trial examining the effectiveness of CBT and BA for adult depression were used. Individuals who experienced a depression spike (*n* = 44; 17 in BA and 27 in CBT) were included in this study. Participants who did not experience a depression spike (*n* = 44) were matched to those who experienced a depression spike by baseline depression score, treatment modality and session number. Sessions before (pre-spike session) and during (spike session) the depression spike were coded for client and therapist variables using the CHANGE coding system (Hayes, Feldman, & Goldfried, 2007).

Results: No hypothesised pre-spike variables were associated with a depression spike, nor were there any interactions with treatment. There were no relationships at the peak of the depression spike, between hypothesised client and therapist variables, treatment type, and depression spike status at 12- or 18-month depression outcome.

Conclusions: This is the first study to examine processes of change in depression spike sessions in CBT and BA. Our study did not find evidence that cognitive and behavioural client processes and therapist strategies were associated with a depression spike, nor were these processes during a depression spike associated with treatment outcomes. It is still unclear what depression spikes represent in CBT and BA, and whether specific in-therapy processes during a depression spike influences depression treatment outcomes. Further replication is needed in larger sample sizes using alternative ways of measuring process and life stressors to see whether they contribute to depression spikes in treatment and at outcome.

Keywords: depression, depression spikes, cognitive behavioural therapy (CBT), behavioural activation (BA), process of change

4.3 Introduction

As the leading cause of disability worldwide (WHO, 2017) depression is a major public health concern. Psychological treatments for depression can effectively reduce symptoms, yet only half of individuals who receive treatment for depression recover (Cuijpers et al., 2014; Hollon & Ponniah, 2010) and relapse rates are high (between 36-43%; Steinert et al., 2014). Therefore, it is important to understand how psychological therapies lead to improvement in depression symptoms in an effort to further improve their effectiveness.

One way in which research identifies key times in therapy to explore processes of change is to look at times when there are sudden depression symptom changes in therapy. In line with complex systems research across scientific disciplines there is growing evidence that change in depression symptoms in psychotherapy is often non-linear (Andrews et al., 2020; Hayes & Andrews, 2020; Hayes, Laurenceau, et al., 2007; Saunders et al., 2019) and these non-linear shifts may signal an imminent transition between states, where new information is embedded and processing occurs (Hayes & Andrews, 2020; Olthof et al., 2020). During these transitional periods, therapy change procedures may be used to facilitate these shifts and introduce more adaptive strategies to cope. Research has identified patterns of discontinuous depression symptom change in psychotherapy that are associated with favourable treatment outcomes (Hayes, Feldman, Beevers, et al., 2007; Shalom & Aderka, 2020) and some treatment process research has focused on these times to examine mechanisms of change within treatments (Abel et al., 2016; Lemmens et al., 2021; Schilling et al., 2020; Wucherpfennig, Rubel, Hofmann, et al., 2017; Yasinski et al., 2019). Perhaps the most widely examined symptom discontinuity studied in the psychotherapy literature is a sudden gain,

which is a rapid improvement of depression symptoms between a single session interval, that is large in magnitude and relative to depression scores prior to the reduction in symptoms (Tang & DeRubeis, 1999). Sudden gains have been repeatedly found to be associated with favourable end of treatment outcomes in a range of psychological therapies across disorders (Deisenhofer et al., 2021; Shalom & Aderka, 2020). Another less researched pattern of change, which is considered to be the conceptual opposite of a sudden gain, is a depression spike (Hayes, Feldman, Beevers, et al., 2007). Characterised by a rapid increase of depression symptoms that subsequently decreases by the same amount or more in the same phase of therapy (Hayes, Feldman, Beevers, et al., 2007), depression spikes were initially observed in an exposure-based cognitive therapy (EBCT) for depression (Hayes, Feldman, Beevers, et al., 2007). EBCT was developed for depression to directly target avoidance and rumination, and to address cognitive, behavioural, emotion regulation and interpersonal issues to enhance resilience and promote good mental health (Hayes & Harris, 2000). The treatment encompasses exposure techniques to encourage the processing of disturbed cognitions and emotions, as well as behavioural activation techniques to reduce avoidance and mindfulness skills to help clients disengage from patterns of avoidance and rumination. During the exposure phase (sessions 9-18) of EBCT the goal is to activate the depression network by exposing the individual to previously avoided, distressing content to induce destabilisation of the depressive network and allow processing to occur. In line with this hypothesis, Hayes et al. (2007) observed a cubic pattern of change in EBCT where an initial rapid improvement in depression symptoms was followed by a transient depression spike. In the middle of treatment during the exposure phase, 62% of individuals experienced a depression spike which was associated with

reduced depression at the end of treatment. In line with the theory that new information and processing occurs within these periods of destabilisation (Hayes & Andrews, 2020) at the peak of a depression spike greater levels of emotional processing occurred, and this processing mediated the association between depression spikes and treatment outcomes (Hayes, Feldman, Beevers, et al., 2007). Although little research directly examining depression spikes in other studies of EBCT has been conducted, the cubic pattern of depression change (which includes a depression spike) has been found in other studies of EBCT. This cubic pattern has been found to be associated with an increase in emotional processing and positive treatment outcomes (Grosse Holtforth et al., 2012; Holtforth et al., 2014) suggesting a transient depression spike is beneficial in EBCT.

Depression spikes have also been found to occur in non-EBCT treatments in which there are no intended therapeutic processes to instigate a depression spike. In these studies the rates of depression spikes and association with treatment outcomes differ to Hayes et al.'s (2007) depression spikes. In non-EBCT treatments depression spikes have been shown generally to occur less frequently (10-50%; Keller et al., 2014; O'Mahen et al., 2021; O'Mahen et al., 2017; O'Mahen et al., 2019; Abel et al., 2014; Ladwa et al. in prep, study one of this thesis), than in Hayes et al.'s (2007) seminal study. Examinations of depression spikes outside EBCT treatments have found they usually occur around the middle of treatment (session 9/18, Abel, 2014; session 5/12, O'Mahen et al., 2017; session 3 or 4/10, O'Mahen et al., 2019) with the exception of Ladwa et al. (in prep; study one, chapter two of this thesis) who found depression spikes were more likely to occur at the beginning of low- and high-intensity depression therapy in everyday clinical practice settings. With regards to treatment outcomes depression spikes in EBCT that occur during periods in the

treatment that are associated with intensive processing, are theoretically and empirically linked to better treatment outcomes (Hayes, Feldman, Beevers, et al., 2007). However, depression spikes in EBCT that occur outside this period of intensive processing, and depression spikes that occur in non-EBCT have varied associations with treatment outcomes. In EBCT, depression spikes that occur outside the period of intensive processing were not associated with outcomes (Hayes, Feldman, Beevers, et al., 2007). Depression spikes in individuals receiving prolonged exposure or pharmacotherapy for Post-Traumatic Stress Disorder (PTSD) (Keller et al., 2014), and in non-exposure based CBT adjunct to pharmacotherapy for individuals with treatment resistant depression (Abel, 2014) were not related to treatment outcome. In a smaller study there was difficulty examining the association between depression spikes and treatment outcome in BA because of the low number of spikes identified (8/41, 19.5%; O'Mahen et al., 2017), which is likely partly due study sample sizes. However, in a larger study of group BA only 10% (7/77) of individuals experienced a depression spike and they were unrelated to treatment outcome (O'Mahen et al., 2019). In contrast, in a large primary care sample looking across low- and high-intensity CBT, group treatment and counselling therapy for depression, depression spikes were associated with positive treatment outcomes (Ladwa et al., in prep; study one, chapter two in this thesis). On the other hand, in a recent study comparing depression spikes between CBT and BA in a large trial, O'Mahen et al. (2021) found depression spikes were associated with higher depression scores at 6-, 12- and 18-months post-randomisation, regardless of treatment type. Further, there was a non-significant trend towards depression spikes in CBT, compared to BA, being associated with higher depression scores at the 18-month follow-up point (O'Mahen et al., 2021). Together, these mixed results suggest

that depression spikes may represent different processes across different treatments. What a depression spike signifies in therapies outside EBCT, like CBT and BA, is unclear. Examining therapeutically important client and therapist factors that may drive a depression spike in these therapies and exploring how these processes during a depression spike relate to treatment outcomes may help to elucidate what depression spikes may represent in non-exposure based treatments for depression. Therefore the current study firstly aimed to examine theoretically relevant processes that might elucidate why depression spikes occur in CBT and BA, and secondly investigated whether these processes during the spike session, which is theorised to be a rich opportunity for corrective processing (Hayes, Feldman, Beevers, et al., 2007), are associated with 12-and 18-month treatment outcomes in CBT and BA.

Firstly, regarding the processes related to depression spikes, the studies to date examining processes factors related to depression spikes outside of EBCT have focused on how baseline factors were associated with depression spikes. In one study in a trial setting, neither self-reported baseline behavioural activation, dysfunctional cognitions, relationship status, nor education status were found to be related to experiencing a depression spike in either CBT or BA for depression in a trial setting (O'Mahen et al., 2021). However, in another study, a PTSD sample of individuals with greater baseline negative trauma-related support were more likely to experience a depression spike (Keller et al., 2014). No research has examined proximal, within therapy factors and their association with the onset of depression spikes in non-EBCT therapies. In the current study we investigate three possible explanations for the presence of depression spikes in non-EBCT therapies; firstly that treatment related client processes or therapist strategies contribute to a

depression spike, secondly that a depression spike represents therapeutic relationship difficulty, or thirdly that a transient increase in depression symptoms is the consequence of external life stressors.

With regards to the first possibility that treatment related processes contribute to depression spike, it is important to consider key processes in CBT and BA that could result in an exacerbation of depression symptoms. Within CBT the goal of therapy is to identify and modify maladaptive cognitions to alleviate depression symptoms and this is achieved through cognitive restructuring (Beck et al., 1979). Cognitive restructuring techniques include identifying and challenging negative thoughts and underlying beliefs (Beck et al., 1979) with the ultimate aim of giving the client access to more realistic beliefs and interpretations of situations. When using these techniques clients are invited to confront and examine negative beliefs about the self and world to develop cognitive flexibility and promote cognitive emotional processing. To aid this there may be cognitive corrective information from the therapist. Because of the need to focus upon and challenge negative material it is possible that increases in cognitive flexibility and cognitive emotional processing may be associated with a transient worsening in depression symptoms. For example, within EBCT greater cognitive emotional processing was found during a depression spike (Hayes, Feldman, Beevers, et al., 2007) but this has not been examined in non-EBCT treatments. In the current study we therefore focus on the client processes of cognitive flexibility and cognitive emotional processing, and the therapist strategy of providing cognitive corrective information as specific aspects of CBT that might be associated with experiencing a depression spike. For BA where the focus is to re-engage clients in positive behaviours to reduce avoidance (Manos et al., 2010), breaking this pattern of avoidance and minimising maladaptive

behaviours to disrupt the cycle of depression may increase exposure to difficult situations and emotions, resulting in a temporary increase in depression symptoms even though the individual is beginning to engage in more positive behaviours. Within therapy clinicians may facilitate engaging in approach behaviour and reducing avoidance through behavioural corrective information. In the current study we therefore also focus on increases in client positive behaviour and therapist behavioural corrective information, and reductions in client avoidance as processes that might be associated with a depression spike. We note, however, that both CBT and BA include behavioural strategies and therefore in our study we expect to see this relationship in both treatments.

In addition to modifications of maladaptive thoughts and behaviours in psychotherapy, the collaborative relationship between a therapist and client is an important part of therapy (Ardito & Rabellino, 2011; Horvath & Luborsky, 1993). Therapeutic alliance in psychotherapy encompasses agreement of therapy goals, collaboration on treatment tasks, and an emotional bond between the therapist and client (Bordin, 1979). Positive alliance is robustly associated with good treatment outcomes (Baier et al., 2020; Flückiger et al., 2018), as well as predictors of change in depression symptoms in subsequent therapy sessions (Falkenström et al., 2013; Webb et al., 2011). In contrast, difficulties in the client-therapist relationship can have the opposite effect and can be associated with worsening in depression. For instance, a recent study examining client and therapist alliance ratings in cognitive behavioural analysis system of psychotherapy (CBASP) and supportive psychotherapy found client, but not therapist, ratings of alliance difficulty were associated with poorer depression outcomes in individuals with chronic depression (Humer et al., 2021). The literature in this area mostly focuses on how to repair

ruptures to mitigate the negative association a breakdown in the therapeutic relationship has on treatment outcomes (Eubanks et al., 2018; Larsson et al., 2018). It is less clear whether there may be more immediate impacts of negativity in the therapeutic relationship on depression symptoms. Therefore, our second hypothesis was that difficulty in the therapeutic relationship would be associated with the worsening of depression symptoms observed in a depression spike. Therapeutic relationship is often considered to be a common factor of psychotherapies (Frank, 1961; Grencavage & Norcross, 1990) and therefore in the current study we did not expect differences in therapeutic difficulty between CBT and BA and the association with a depression spike.

Our third hypothesis was that depression spikes may be the consequence of a transient external stressor. The link between stressful life events and the onset of depression has been repeatedly observed (Hammen, 2005; Paykel, 2003; Tennant, 2002), but it is also the case that individuals with depression are more likely to experience life stressors compared to individuals without depression (Mazure, 1998). Therefore, in the current study we examined whether a depression spike might represent the impact of negative life events or stressors. As these may not be linked to therapy, we did not expect treatment type (CBT or BA) would moderate the association between stressful life events and experiencing a depression spike.

The second aim of our study was to examine processes at the peak of the spike, which is theorised to be a rich opportunity for corrective processing (Hayes, Feldman, Beevers, et al., 2007), and their association with treatment outcomes. It is currently unclear, theoretically, whether depression spikes in CBT and BA should be linked to better or worse treatment outcomes, and the current literature examining depression spikes in non-EBCT treatments reflects this (e.g., Abel, 2014; Ladwa et

al., in prep; O'Mahen et al., 2021). Results from a recent study suggest that there may be differential long-term effects of depression spikes in CBT and BA, with CBT depression spikes being associated with non-significantly worse depression scores than their counterparts in BA. Examining the association of spike processes with immediate and long-term treatment outcomes may allow us elucidate the factors involved.

Given that in CBT it is hypothesised that fundamental changes in negative belief structures promote long term wellness in depression (Beck et al., 1979), if an individual is able to engage in significant cognitive emotional processing despite exacerbations in depression symptoms, this may be beneficial. Following the logic from Hayes et al. (2007), at the peak of the spike, intensive cognitive-emotional processing would be expected to contribute to longer-term changes in the form of better treatment outcomes. Alongside this, changes to rigidity in cognitive thinking that characterise depression (Joormann, 2010), such as cognitive flexibility, during a depression spike may also influence treatment outcomes because it may allow an individual to consider different perspectives and engage in problem solving. No research has examined this process within the context of a depression spike, however Yasinski et al. (2019) used depression spike sessions as guides to examine processes changes in CBT and found increases in cognitive flexibility predicted favourable depression outcomes at 12-months post-treatment. In addition to greater cognitive emotional processing and cognitive flexibility in the client, at the peak of the spike greater cognitive corrective information supplied by the therapist, such as skilfully challenging negative thinking despite being in a heightened mood state, may also be associated with positive long terms depression outcome. We expected these cognitive factors to have more of an impact on treatment outcomes in individuals in

CBT, which employs strategies to target cognitive processes, compared to BA where there is a behavioural focus.

With regard to behavioural processes, we would expect greater positive behaviour and reduced avoidance in the client, and behavioural corrective information from the therapist at the peak of the spike to be associated with better treatment outcomes. This is in line with the rationale of BA (Martell et al., 2001) where reductions in avoidance allow an individual to work through their difficulty and engage in activities (positive behaviours) that lead to positive reinforcement and improved mood. This is also applicable to CBT where there is a behavioural focus in the early stages of treatment to reduce depression symptoms before moving to work on cognitive restructuring strategies (Beck et al., 1979). Because both therapies include a focus on behavioural change we would expect to see this effect in both CBT and BA.

4.3.1 The Current Study and Hypotheses

In this study we sought to examine whether client process and therapist strategies of interest (Table 4.1) were related to depression spikes and treatment outcome at 12- and 18- months post-randomisation using data from a large, non-inferiority, randomised controlled trial (RCT) of CBT and BA for adults with Major Depressive Disorder (MDD) (Richards et al., 2016). We were interested in the in-session client processes and therapist strategies that may predict depression spikes and better or worse long term depression outcome. Furthermore, we sought to explore whether the relationships between processes, depression spikes and depression outcome differ according to therapy type (CBT/BA).

Table 4. 1

Process Variables of Interest in the Current Study

	Cognitive	Behavioural	Other
Therapist	Corrective cognitive information	Corrective behavioural information	Therapeutic difficulty
Client	Cognitive-emotional processing Cognitive flexibility	Positive Behaviour Avoidance	Negative life event

The first aim of this study was to examine whether the client processes and therapist strategies of interest were prospectively associated with experiencing a depression spike and whether this would vary between CBT and BA. We tested three hypotheses that might explain the presence of a depression spike in CBT or BA

Firstly, we examined whether there were specific processes in the session before the escalation in depression symptoms (henceforth referred to as the pre-spike session) that were associated with a depression spike (hypothesis one). We expected that increases in cognitive client processes and therapist strategies would be associated with having a depression spike, and this would be more likely in CBT than BA. We also hypothesised depression spikes would be associated with increased positive behaviour, reduced avoidance, and increased therapist behavioural corrective information in the pre-spike session, however we did not hypothesise that this relationship would be moderated by therapy type.

Secondly, we tested whether a depression spike could be the result of difficulty in the therapeutic relationship (hypothesis two). We hypothesised that an increase in therapeutic difficulty within the client-therapist relationship in the pre-spike session would be associated with the presence of a depression spike. We explored whether there would be treatment differences.

Lastly, we examined whether negative life events in the session at the peak of the spike (henceforth referred to as the spike session), reflecting life stressors in the week preceding the spike were associated with a depression spike (hypothesis three). We expected a negative life event in the week before the spike in depression scores would be associated with experiencing a depression spike. We explored whether there would be any treatment differences.

The second aim of this study was to examine whether treatment related processes at the peak of the depression spike (the spike session), which is theorised to be a rich opportunity for corrective processing (Hayes, Feldman, Beevers, et al., 2007) are associated with depression treatment outcomes at 12- and 18-months post-randomisation. For hypothesis four we expected that higher levels of client cognitive emotional processing and cognitive flexibility, and therapist cognitive corrective information in individuals who experienced a depression spike, compared to those who do not, would be associated with lower depression scores at 18-months post-randomisation. We expected these processes/strategies to be associated with beneficial outcomes more so in CBT compared to BA. We hypothesised greater therapist behavioural corrective information, client positive behaviour, reduced avoidance in individuals who experience a depression spike, compared to match yolked controls who do not experience a spike, would be associated with better depression scores at 18-months post-randomisation. Similarly to hypothesis one,

because both CBT and BA encompass behavioural processes/strategies we did not make a directional hypothesis about relationship with treatment type and instead explored whether treatment type moderated these associations.

4.4 Methods

4.4.1 Data source: The COBRA Trial

This analysis was an extended part of a process analysis for the 'Cost and Outcome of Behavioural Activation versus Cognitive Behavioural Therapy for Depression' (COBRA) trial, a non-inferiority, randomised controlled trial (RCT) which examined the cost and clinical effectiveness of BA compared to CBT for adults with major depressive disorder (MDD) (ethical approval reference NRES/07/H1208/60) (Richards et al, 2016). Adults over the age of 18 who met the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV; APA, 2000) criteria for MDD were recruited from primary care and psychological services at three sites in the United Kingdom (UK). A total of 440 participants consented to the trial and were randomly allocated to receive BA (*n* = 221) or CBT (*n* = 219), stratified by baseline depression severity, antidepressant use and recruitment site. Individuals were excluded if they were receiving psychological therapy, were alcohol or drug dependent, acutely suicidal, cognitively impaired, and had bipolar disorder or psychotic symptoms. The full trial protocol (Rhodes et al., 2014) and main findings (Richards et al., 2016) are reported elsewhere.

4.4.2 Therapy and Therapists

Two therapies for depression, BA and CBT, were examined in this trial. BA aims to disrupt the cycle of depression by encouraging re-engagement with previously avoided behaviours despite negative mood. Within the trial BA was

delivered according to the National Institute for Health and Care Excellence (NICE) guideline which recommends 16-20 sessions over 3-4 months (NICE, 2009), using a revised treatment manual (Ekers et al., 2011). This revised manual followed standard BA treatment set out by Martell et al. (2001) with optional modules to help with rumination, communication, problem-solving strategies, approaches to managing anxiety, and to find equivalent behaviours within a BA framework. On the other hand, CBT targets both dysfunctional thoughts that contribute to maladaptive behaviours and subsequently negative mood. Strategies are employed to modify these thoughts as well as test out adaptive behaviours to reduce depression. In the trial, a CBT manual based on Beck et al.'s (1979) approach was followed.

Participants received a maximum of 20 sessions of either BA (*M* = 17.56, *SD* = 4.94) or CBT (*M* = 18.06, *SD* = 5.28) over 16 weeks, with the option of four booster sessions. Therapy sessions were delivered face-to-face and lasted approximately 60 minutes. Junior mental health workers (MHWs) delivered BA, whereas CBT was delivered by senior mental health workers with a postgraduate diploma (Two years or more of study) in CBT. Therapist competency of treatment was assessed during the main trial by the rating of random treatment audio recordings by independent experts of both treatments. Both MHWs and therapists met acceptable competency standards (Richards et al., 2016).

4.4.3 Measures

4.4.3.1 Baseline and Outcome Depression. The Patient Health Questionnaire (PHQ-9; Kroenke, Spitzer, & Williams, 2001) is a nine item measure of depression severity over the previous two weeks and is scored on a four-point Likert scale, where scores can range from 0-27. The PHQ-9 is sensitive to detecting change over time (Löwe et al., 2004), and is a valid and reliable measure of

depression severity (Kroenke et al., 2001). In the original trial the PHQ-9 was used to measure depression severity at baseline, 6-, 12- and 18-month outcome.

4.4.3.2 Weekly Depression Scores. The Beck Depression Inventory (BDI; Beck & Steer, 1987) is a 21 item self-report of depressive symptoms over the previous week and is score on a four point (0-3) Likert scale, where higher scores indicate severe depression. The BDI has good reliability and validity (Beck et al., 1988). In the original trial, the BDI was used to measure weekly depression symptoms. For the current study weekly BDI scores were used to identify depression spikes (O'Mahen et al., 2021).

4.4.3.3 Change and Growth Experiences Scale (CHANGE; Hayes, Feldman, & Goldfried, 2007). The CHANGE is an observational coding system designed to examine a range of client processes of change and therapist strategies in psychotherapy. The processes are rated on a four point Likert scale (0, not present; 1, low; 2, medium; 3, high) and variables are not mutually exclusive and can co-occur. During coding content from both within the session and experiences from the week prior to the therapy session are considered. The CHANGE has good reliability across a range of treatments and disorders (Abel et al., 2016; Cummings et al., 2012; Yasinski et al., 2019).

For the current study a range of cognitive, behavioural and non-specific processes were coded (Table 4.1). Further, an additional code was created for the CHANGE manual, client life events (Appendix 1). This code was created to capture life events that occurred since the last therapy session. The life event was defined as an event that caused a significant change in a person's life or circumstance outside of therapy and had a significant emotional impact on the client. Following the

identification of a life event(s) during coding if there were multiple events all coders consented on the most impactful life event and agreed the event identified caused a significant change in an individual's circumstance outside therapy and had a significant emotional impact. Following this the valence, severity, whether the event was independent or dependent, and whether the event was resolved was rated on the same 0-3 Likert scale used in the original CHANGE manual. Only life events which were rated as negative (irrespective of the severity, dependence or resolution of the event) were used in the current study.

Descriptions of the coded client and therapist variables in the current study can be found in Table 4.2 and examples of each process can be found in the Appendix 2.

Table 4. 2

CHANGE Coding System Variables used in the Current Study

Process Variable	Description	ICC
Therapist cognitive corrective information	Examining and challenging maladaptive perceptions and patterns of thinking that focus on issues related to the self, identity, goals and world view	0.84
Cognitive-Emotional Processing	The extent an individual approaches and explores a problem and try to make meaning of it and challenge it	0.69

Cognitive Flexibility	Ability to see multiple perspectives on a situation or consider points of view different than one's initial point of view. Includes an ability to switch or change perspectives, and consider multiple factors in forming an opinion or any other cognitive response to a situation	0.84
Therapist behavioural corrective information	Identifying maladaptive patterns of behaving, balancing over-or under- control of behaviours, encouraging the client to engage in new experiences.	0.78
Positive behaviour	Adaptive behaviours an individual engages with between therapy sessions.	0.70
Avoidance	Captures events to protect/defend self by pulling away rather than moving towards problems or issues, e.g. social withdrawal, staying in bed	0.72
Therapeutic difficulty	This includes any strain on the client- therapist relationship, including difficulty form an alliance, disagreement on goals, or mismatch of the therapist and client focus.	0.91
Negative Life Event	Captures an event which causes a significant change and negative emotional impact in a person's life or circumstance outside of therapy.	1.00

Note. ICC = Intraclass Correlation

4.4.4 Procedure

4.4.4.1 Defining Depression Spikes. Depression spikes were originally defined by Hayes et al. (2007) as an increase in seven depression points or more, which subsequently decreases by the same amount or more (depression spike recovery) within the same phase of therapy in EBCT. As treatment phases are arbitrary, O'Mahen et al. (2021) examined the average number of sessions it took for a depression spike to recover in the per-protocol COBRA sample across CBT and BA (*M* = 2.52, *SD* = 2.11). A comparison of depression spikes identified using Hayes et al.'s (2007) original depression spike criterion and the modified criterion of depression spikes recovering within a three-session period resulted in no differences in the number of depression spikes identified (O'Mahen et al., personal communications). Depression spikes that reversed and were captured as another spike were included in the depression spike identification. If individuals experienced more than one depression spike the spike closest to the middle of treatment was selected.

It is important to note that depression spikes are different from 'sudden losses' which are defined as increases in depression scores which do not return (see Lutz et al., 2013).

4.4.4.2 Session Selection. The original trial sample was initially restricted to individuals who attended the per-protocol number of eight therapy sessions (Richards et al., 2016; NICE, 2009) to ensure that participants had received an adequate dose of treatment, and who had completed the BDI within at least six sessions. This resulted in a sample of 300/400 (75%). A total of 77/300 (26%) experienced a depression spike (33/77, 43% BA; 44/77, 57% CBT) (O'Mahen et al., 2021). For the current study individuals who consented to their audio therapy tapes

being used for further research, met the criteria for depression spikes, and who had therapy recordings for the exact pre-spike and spike sessions were eligible for this secondary analysis. This resulted in the analysis of 44 individuals with a depression spike (17/44, 39% BA; 27/44, 61% CBT).

As there was no control group within the original trial a comparison yoked group was created from 44 individuals who did not experience a depression spike at any point in treatment. Individuals in the yoked group were matched to participants who experienced a depression spike by treatment type (CBT/BA), baseline PHQ-9 band score (0-4 minimal depression; 5-9 mild; 10-14 moderate, 15-19 moderately severe; 20-27 severe depression), and depression spike session number. If the recording of the exact session number was not available, the session as close as possible to that of the depression spikes participant was chosen (+/-1). Participants were also matched according to whether or not they had experienced a sudden gain (Tang & DeRubeis, 1999) at any point during the therapy period. If no sudden gain was experienced the depression spike participant was matched to a control participant who had not experienced either a sudden gain or depression spike. This resulted in 88 individuals (*n* = 44 depression spike participants) being included in the current study.

A sensitivity calculation in G*Power (Faul et al., 2007) indicated that a sample size of 88 participants, with 95% power, an alpha of 0.05, and 28 predictors (all main effects and interactions) would allow us to detect a small-medium effect (Cohen's *d* = 0.3) (Cohen, 1988).

4.4.4.3 Coding and Coders. De-identified audio therapy tapes were coded by three coders who attended a three day training in the CHANGE coding system and

had previous experience of using the system. Coders were blind to treatment type (CBT/BA), depression spike status, therapy session number, and treatment outcome. Out of 176 tapes 52 (30%) were double coded to prevent rater drift and assess inter-rater reliability. Weekly meetings were held to discuss discrepancies in coding of two or more points for sessions which were double coded. Consensus codes replaced discrepant codes and then all ratings were averaged between the two coders. The inter-rater agreement between coders (intraclass correlations, ICC) for each process ranged from (0.60-1.00, see Table 4.2), indicating moderate to excellent agreement (Koo & Li, 2016).

4.4.5 Data Analytical Strategy

Statistical analyses were performed using IBM SPSS Statistics version 25 (IBM Corp, 2017). To examine whether there were any baseline demographic and clinical characteristic differences between individuals who did and did not experience a depression spike, *t*-tests and chi-square analyses were conducted[5].

A logistic regression analysis was conducted to examine whether cognitive and behavioural client processes and therapist strategies, therapeutic difficulty in the pre-spike session, and negative life events in the spike session, were associated with depression spike status. In the first step, pre-spike BDI score was entered to account for depression severity just before the depression spike. In the second step treatment type (CBT/BA) and the process variables were entered, and in the final

[5] For additional demographic and baseline clinical characteristic comparisons between the current sample compared with the full trial sample (Appendix 3) and those who experienced a depression spike but were not coded within the current study (Appendix 4) see chapter appendices.

step two-way interactions between treatment type (CBT/BA) and process variables were entered. The dependent variable was depression spike status (0, 1). All process variables and the two-way interactions were entered into a single regression model as these variables do not occur in isolation in treatment and this is the most conservative model[6]. All model assumptions were met.

To examine how spike treatment related processes/strategies[7] were associated with 12- and 18-month PHQ-9 depression treatment outcome, two hierarchical linear regression models were conducted. In the first step, pre-spike BDI score was entered to account for depression severity prior to the depression spike. The main effects of treatment type (CBT/BA), depression spike status (0, 1), and the process variables were entered in step two. In the third step the two-way interactions were entered, and the three-way interactions between treatment type, depression spike status, and process variable was entered in fourth step. The dependent variable was 12- or 18-month PHQ-9 score. All assumptions were met, except the assumption of homoscedasticity. Visual examination of plots suggested there was heteroscedasticity within the data (a funnel shape of data points). A Box-Cox transformation was applied to the outcome variables (PHQ-9 at 12- and 18-months post-randomisation). Following this visual examination of a scatter plot with this transformed dependent variable showed a random scatter of data points, suggesting the transformation had corrected the heteroscedasticity and the assumption of homoscedasticity was met.

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[6] Separate regression models were also run for each process variable and can be found in appendix 5 and 6. The results did not differ to the family model presented in the result section of the study

[7] The association between therapeutic difficulty and negative life events in the spike session and their relation to 12- and 18-month treatment outcomes are in appendix 7

4.5 Results

4.5.1 Participants

Individuals with and without depression spikes did not differ in age, antidepressant use, baseline PHQ-9 score, sex, relationship status, ethnicity or level of education (Table 4.3). However, in this sub-sample individuals who had a depression spike (*M* = 5.66, *SD* = 6.75) reported significantly more previous MDD episodes than those who did not experience a depression spike (*M* = 2.82, *SD* = 2.32). The means and standard deviations for each process variable split by treatment and depression spike status can be found in Table 4.4 and the correlations between prespike and spike variables are in Table 4.5.

Table 4. 3

Comparison of Demographic and Clinical Characteristics in the Current Study

	No Depression Spike (*n* = 44)			Depression Spike (*n* = 44)						
Variable	*n* (%)	*M*	*SD*	*n* (%)	*M*	*SD*	χ^2	*t*	*df*	*p*
Treatment							0.00		1	1.000
CBT	27 (30.7%)			27 (30.7%)						
BA	17 (19.3%)			17 (19.3%)						
Age (years)		47.52	14.81		45.45	14.05		0.67	86	.503
Antidepressant use							0.31		1	.783
Yes	35 (39.8%)			37 (42%)						
No	9 (10.2%)			7 (8%)						
Baseline PHQ-9		17.25	4.39		18.41	5.06		-1.15	86	.254
Number of previous MDD episodes		2.82	2.32		5.66	6.75		-2.25	37.82	.030*
Number of treatment sessions		17.09	5.15		18.64	5.04		-1.42	86	.159

Sex			0.06	1	1.000
Female	31 (35.2%)	32 (36.4%)			
Male	13 (14.8%)	12 (13.6%)			
Relationship status			0.44	1	.660
Not in a relationship	18 (20.5%)	15 (17%)			
In a relationship	26 (29.5%)	29 (33%)			
Ethnicity			0.21	1	1.000
White	42 (47.7%)	41 (46.6%)			
Other	2 (2.3%)	3(3.4%)			
Education			2.59	2	.293
No qualifications	3 (3.4%)	8 (9.1%)			
Secondary School	26 (29.5%)	23(26.1%)			
Degree	15 (17%)	13 (14.8%)			

Note. CBT = Cognitive Behavioural Therapy; BA = Behavioural Activation; PHQ-9 = Patient Health Questionnaire; MDD = Major Depressive Disorder.

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

Table 4. 4

Means (Standard Deviation) for Each Process Variable Split by Depression Spike Status and Treatment Type

	Cognitive Behavioural Therapy				Behavioural Activation			
	No Depression Spike		Depression Spike		No Depression Spike		Depression Spike	
	Pre-Spike	Spike	Pre-Spike	Spike	Pre-Spike	Spike	Pre-Spike	Spike
Therapist Cognitive Corrective Information	0.98 (0.78)	1.15 (0.71)	1.17(0.92)	0.91 (0.77)	0.47(0.48)	0.68 (0.66)	0.38 (0.78)	0.35 (0.55)
Cognitive Emotional Processing	0.79 (0.82)	0.43 (0.66)	0.48(0.67)	0.59 (0.69)	0.74(0.77)	0.85 (0.86)	0.56 (0.86)	0.44 (0.53)
Cognitive Flexibility	0.56 (0.64)	0.43 (0.55)	0.42(0.69)	0.41 (0.54)	0.44(0.50)	0.44 (0.50)	0.44 (0.58)	0.32 (0.43)
Therapist Behavioural Corrective Information	0.54 (0.54)	0.76 (0.58)	1.02(0.83)	1.07 (0.69)	1.65(0.52)	1.62 (0.55)	1.21 (0.83)	1.35 (0.49)

Positive Behaviour	1.17 (0.80)	1.06 (0.58)	1.09 (0.80)	1.04 (0.77)	1.50(0.66)	1.41 (0.85)	1.41 (0.78)	0.88 (0.67)
Avoidance	1.00 (0.76)	1.19(0.89)	1.39 (0.95)	1.78 (0.86)	1.47(0.74)	1.59 (0.73)	1.62 (1.04)	1.29 (0.83)
Therapeutic Difficulty	0.24 (0.51)	0.32 (0.46)	0.11(0.29)	0.24 (0.58)	0.41(0.48)	0.24 (0.40)	0.56 (0.83)	0.29 (0.44)

Table 4. 5

Correlations between Prespike and Spike Client Process and Therapist Variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.	14.
1. Prespike therapist cognitive corrective information	1	.576***	.381***	.360**	.384***	.143	.064	.065	-.018	-.013	-.264*	-.109	-.143	-.137
2. Spike therapist cognitive corrective information		1	.454***	.210*	.434***	.193	-.163	-.028	.082	.049	-.254*	-.100	-.173	-.192
3. Prespike cognitive emotional processing			1	.323**	.570***	.341**	.105	-.016	.279**	.208	-.188	-.152	-.291**	-.275**
4. Spike cognitive emotional processing				1	.380***	.398***	.322**	.068	.316**	.404***	-.061	-.283**	-.185	-.275***

5. Prespike cognitive flexibility	1	.416***	.102	.137	.294**	.090	-.256*	-.312**	-.224*	-.228*
6. Spike cognitive flexibility		1	-.036	-.037	.195	.202	-.018	-.106	-.166	-.186
7. Prespike therapist behavioural corrective information			1	.482***	.329**	.295**	.152	.014	-.112	-.156
8. Spike therapist behavioural corrective information				1	.278**	-.012	.156	.184	.157	-.145
9. Prespike positive behaviour					1	.289**	-.219*	-.302**	-.113	-.113

10. Spike positive behaviour	1	.045	-.184	-.021	-.225*
11. Prespike avoidance		1	.404***	.057	.110
12. Spike avoidance			1	.157	.030
13. Prespike Therapeutic difficulty				1	.481***
14. Spike Therapeutic difficulty					1

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

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4.5.2 Processes Associated with a Depression Spike

A logistic regression model was conducted to assess whether processes were associated with depression spike status. The results are in Table 4.6.

Firstly, we assessed whether prespike cognitive or behavioural, client processes and therapist strategies were associated with depression spike status, and if these relationships were moderated by treatment type (hypothesis one). Contrary to expectations, none of the cognitive processes/strategies (therapist cognitive corrective information, client cognitive-emotional processing, and client cognitive flexibility) were associated with depression spike status, nor were these relationships moderated by treatment modality. Similarly, no behavioural processes/strategies (therapist behavioural corrective information, client positive behaviour and client avoidance) in the prespike session were associated with depression spike status. Although positive client behaviour and avoidance were not moderated by treatment type, there was a significant interaction between pre-spike therapist behavioural corrective information and treatment (Figure 4.1). However, this difference was between treatment conditions in individuals who did not have a spike, rather than in those who had a spike. In individuals who did not experience a depression spike higher levels of behavioural corrective information was supplied by the therapist in the pre-spike session in BA compared to CBT.

Contrary to the second hypothesis therapeutic difficulty in the pre-spike session was not associated with depression spike status. This relationship was also not moderated by treatment type.

Further, contrary to our third hypothesis, life events (yes/no) in the spike session were not associated with experiencing a depression spike. Due to low

numbers of negative life events in the spike session (4/13, 30.8% BA; 9/13, 69.2% CBT) it was not possible to examine whether this relationship was moderated by treatment type.

Table 4. 6

Binary Logistic Regression Analyses Examining Processes Associated with Depression Spike Status (0, 1)

	Exp(B)	95% CI
Step 1		
Pre-spike BDI	.998	.954, 1.043
Step 2		
Treatment (CBT/BA)	.797	.236, 2.694
Pre-spike Therapist cognitive corrective information	1.640	.804, 3.348
Pre-spike cognitive emotional processing	.482	.224, 1.038
Pre-spike cognitive flexibility	1.094	.433, 2.761
Pre-spike Therapist behavioural corrective information	1.028	.516, 2.048
Pre-spike Positive behaviour	.992	.495, 1.987
Pre-spike Avoidance	1.705	.926, 3.139
Pre-spike therapeutic difficulty	.672	.264, 1.714
Spike Negative Life event (0/1)	1.666	.425, 6.531
Step 3		
Pre-spike Therapist cognitive corrective information x Treatment	2.088	.350, 12.470

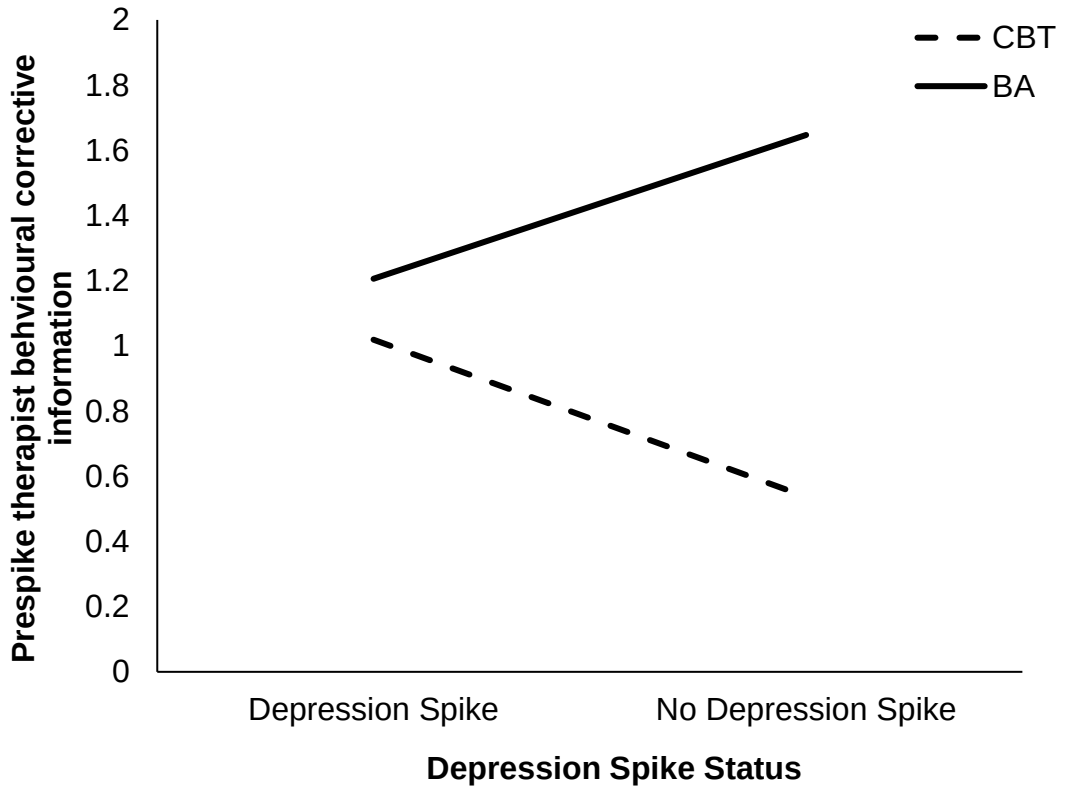
Pre-spike Cognitive-Emotional Processing x Treatment	.687	.124, 3.804
Pre-spike Cognitive flexibility x Treatment	.403	.041, 4.006
Pre-spike Therapist behavioural corrective information x Treatment	9.703**	1.837, 5.257
Pre-spike Positive Behaviour x Treatment	.811	.173, 3.794
Pre-spike Avoidance x Treatment	1.282	.354, 4.637
Pre-spike Therapeutic Difficulty x treatment	.617	.152, 2.495

Note. BDI = Beck Depression Inventory; CBT = cognitive behavioural therapy; BA = behavioural activation.

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

Figure 4. 1

Treatment by Prespike Therapist Behavioural Corrective Information Interaction on Depression Spike Status



4.5.3 Spike Processes Associated with 12-months Post-Randomisation Outcome

The results of the linear regression are presented in Table 4.7. The results showed there was a main effect of depression spike status; individuals with a depression spike, compared to those who did not have a spike, had significantly higher PHQ-9 scores at 12-months outcome. Additionally there was also a main effect of therapist behavioural corrective information; individuals who received greater behavioural corrective information from the therapist experienced lower

PHQ-9 scores at 12-months outcome. This association was not moderated by treatment type or depression spike status. There were no other significant main effects.

Contrary to expectations, there were no significant two-way interactions between spike processes and depression spike status or treatment type, nor were there any significant three-way interactions between processes, treatment type and depression spike status on 12-month treatment outcomes.

Table 4. 7

Spike Processes Linear Regression Model on 12 and 18 Month Post-randomisation PHQ-9 Outcome

	12 month PHQ-9						18 month PHQ-9					
	B(*se*)	*t*	*p*	95% CI	R^2adj	$R^2\Delta$	*B*(*se*)	*t*	*p*	95% CI	R^2adj	$R^2\Delta$
Step 1					.087	.099**					.093	.104**
Constant	-0.66(0.25)	-2.68	.009**	-1.16, -0.17			-0.68(0.24)	-2.79	.007**	-1.16, -0.19		
Prespike BDI Score	.034(.01)	2.96	.004**	.011, .058			0.04(0.01)	3.08	.003**	0.01, 0.05		
Step 2					.155	.151					.062	.060
Depression Spike	.540(.21)	2.517	.014*	.112, .967			0.26(0.23)	1.16	.249	-0.18, 0.71		
Treatment	-.430(.26)	-1.64	.106	-.953, .093			-0.08(0.27)	-0.27	.790	-0.61, 0.47		
Spike Therapist cognitive corrective information	.257(.17)	1.55	.124	-.073, .587			0.08(0.17)	0.44	.663	-0.27, 0.42		
Spike Cognitive emotional processing	-.112(.18)	-.624	.534	-.470, .246			0.29(0.19)	1.51	.136	-0.09, 0.67		
Spike Cognitive flexibility	.119(.22)	.553	.582	-.311, .549			-0.15(0.22)	-0.62	.516	-0.60, 0.30		

Spike Therapist behavioural corrective information	-.429(.18)	-2.413	.018*	-.784, -.075		-0.22(0.18)	-1.20	.235	-0.58, 0.15	
Spike Positive behaviour	-.012(.17)	-.073	.942	-.343, .319		-0.13(0.17)	-0.74	.460	-0.46, 0.21	
Spike Avoidance	.161(.14)	1.188	.239	-.109, .431		0.10(0.14)	0.72	.476	-0.18, 0.38	
Step 3					.046 .056					.071 .153
Depression Spike x Treatment	-.460(.65)	-.704	.484	-1.77, .847		-.243(.64)	-.38	.705	-1.52, 1.03	
Spike Therapist cognitive corrective information x Treatment	.233(.45)	.516	.608	-.671, 1.137		0.34(0.45)	0.76	.451	-.553, 1.229	
Spike Cognitive emotional processing x Treatment	-.153(.46)	-.335	.739	-1.06, .758		0.27(0.45)	.602	.549	-0.63, 1.163	
Spike Cognitive flexibility x Treatment	.195(.53)	.372	.711	-.855, 1.246		-0.31(0.52)	-0.59	.555	-1.34, 0.73	

Spike Therapist behavioural corrective information x Treatment	-.168(.52)	.323	.748	-1.209, .873	0.29(0.47)	.61	.542	-.653, 1.230
Spike Positive behaviour x Treatment	.121(.39)	.308	.759	-.663, .905	-0.17(0.38)	-0.46	.649	-.928, .582
Spike Avoidance x Treatment	-.214(.32)	-.666	.508	-.859, .430	-0.51(0.31)	-1.65	.104	-1.135, 0.108
Spike Therapist cognitive corrective information x Depression Spike	.581(.39)	1.477	.145	-.206, 1.368	0.69(0.39)	1.79	.079	-.083, 1.466
Spike Cognitive emotional processing x Depression Spike	.045(.47)	.097	.923	-.885, .975	0.52(0.46)	1.13	.264	-.399, 1.428
Spike Cognitive flexibility x Depression Spike	.081(.49)	.166	.869	-.893, 1.055	-0.24(0.48)	-0.05	.960	-.977, .929

Spike Therapist behavioural corrective information x Depression Spike	-.069(.41)	-.171	.865	-.881, .742			0.01(0.39)	-.022	.983	-0.78, 0.77	
Spike Positive behaviour x Depression Spike	-.039(.38)	-.104	.918	-.792, .714			-0.01(0.36)	-0.03	.977	-0.73, 0.70	
Spike Avoidance x Depression Spike	.191(.30)	.631	.531	-.415, .797			0.18(0.29)	0.59	.558	-0.42, 0.77	
Step 4					-.018	.029					.024 .036
Spike Therapist cognitive corrective information x Treatment x Depression Spike	.479(1.09)	.441	.661	-1.700, 2.658			-.566 (1.03)	-.549	.585	-2.631, 1.499	
Spike Cognitive emotional processing x Treatment x Depression Spike	-.090(1.12)	-.080	.936	-2.343, 2.163			-1.25 (1.09)	-1.14	.259	-3.431, .941	

Spike Cognitive flexibility x Treatment x Depression Spike	1.354(1.14)	1.190	.239	-.928, 3.636	1.43(1.09)	1.30	.198	-.771, 3.63
Spike Therapist behavioural corrective information x Treatment x Depression Spike	-.718(1.14)	-.629	.532	-3.008, 1.572	-.451 (.99)	-.454	.652	-2.442, 1.540
Spike Positive behaviour x Treatment x Depression Spike	-.123(.87)	-.142	.887	-1.861, 1.614	.038(.79)	.048	.962	-1.553, 1.629
Spike Avoidance x Treatment x Depression Spike	.099(.71)	.139	.890	-1.323, 1.520	.19(.68)	.287	.776	-1.160, 1.547

Note. BDI = Beck Depression Inventory.

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

4.5.3 Spike Processes Associated with 18-Months Post-Randomisation Outcome

The results of the linear regression are presented in Table 4.7. Contrary to our hypotheses, no main effects of cognitive or behavioural, client processes or therapist strategies were associated with 18-month outcome. Similarly, there were no two-way interactions, nor were there any significant three-way interactions between cognitive or behavioural client process/therapist strategy, depression spike status, and treatment type on 18-month post-randomisation PHQ-9 score.

4.6 Discussion

This study is the first to explore potential client processes and therapist strategies that may be associated with a depression spike and treatment outcomes in CBT and BA. Contrary to expectations, did not find any of our hypothesised processes were associated with experiencing a depression spike. Similarly none of our hypothesised processes were associated with 12- or 18-month treatment outcomes. We found those who experienced a depression spike had worse depression scores at 12-months outcome, but this was not moderated by depression spike status or treatment type. Further, we found individuals who received greater behavioural corrective information from the therapist experienced lower PHQ-9 scores at 12-months treatment outcome, but similarly this was not moderated by depression spike status or treatment type. Nevertheless, this study contributes to the limited process research on depression spikes in treatments that do not use deliberate therapeutic strategies to instigate a temporary exacerbation in depression symptoms.

Previous examination of client processes during depression spike sessions have been conducted in EBCT treatment (Hayes, Feldman, Beevers, et al., 2007), but none have examined processes associated with depression spikes in CBT and BA. In the current study we tested three possible hypotheses of why depression spikes may be seen in non-exposure based treatments, like CBT and BA. Contrary to predictions we did not find support for any of our hypotheses. With regards to the first hypothesis, this is the first study to empirically examine whether theoretically important client processes and therapist strategies in CBT and BA are related to a depression spike. Although neither client behavioural (positive behaviour and avoidance) or cognitive (cognitive emotional processing and cognitive flexibility) processes, and therapist cognitive corrective information was not associated with a depression spike, we did observe treatment differences in therapist behavioural corrective information in the session prior to the depression spike. However, the pattern of results was not as we had predicted. In contrast, in the prespike session, there were higher levels of behavioural corrective information provided in BA, but not CBT, in individuals who *did not* have a spike. Although speculative, it is possible that therapist procedures such as behavioural corrective information functions on mood in a different fashion than procedures such as encouraging in-depth cognitive-emotional processing. Perhaps individuals provided with more behavioural corrective information implement concrete behavioural changes that are then associated with a lesser risk of having a sudden upward shift in depressive mood. If EBCT focusses on intensive cognitive processing in an attempt to destabilise unhealthy depressive networks, then it may be possible that strategies like behavioural corrective information stabilise healthy networks. To test this approach, however, it would be important to examine prospective negative and healthy network organisation.

Further, it is of note that these individuals are yoked to those who experienced a depression spike and therefore the random assignment pattern was broken so further investigations of this would be needed in non-yoked individuals.

Our second hypothesis sought to investigate whether depression spikes in CBT and BA may represent outside-therapy negative life events or stressors, but no association was found. It was not possible to examine whether treatment moderated this relationship because too few negative life events were captured through coding. It is possible the CHANGE measure was not sensitive enough to pick up all negative life events. Although the method used in the current study allows coders to have some context around life events and not rely on subjective checklists of pre-determined event types (Harkness & Monroe, 2016) further refinement, validation, and replication of the life events measure in the CHANGE coding system is needed. It has been repeatedly shown that stressful life events are a risk factor for depression (Kessler, 1997) and exacerbation in symptoms (Hassanzadeh et al., 2017; Sokratous et al., 2013) and this perhaps suggests regular assessment of life events during therapy is needed for both research and clinical practice. For research it would better allow us to understand whether stressful events were contributing to discontinuous depression changes. Whereas for clinical practice the lack of life events identified within the current study may also suggest that therapists are not identifying or discussing negative life events or stressors which may be contributing to depression and how an individual may engage in therapy. To aid identification of life events in both research and clinical practice there are a number of validated tools. Semi-structured interviews of life events can be time consuming and impractical (Harkness & Monroe, 2016) but research has begun to validate computerised measure of life events (for example the ‘Computerized Life Events

Assessment Record', CLEAR; Bifulco et al., 2019) which may allow the tracking of the evolvement of a life event and symptom scores across a number of therapy sessions. Although the current study could not ascertain whether depression spikes within therapy were influenced by external life stressors or event, future research is needed to understand this.

We also considered that the rapid exacerbation of depression symptoms seen in a depression spike may have been due to difficulty in the therapeutic relationship, however no association was found in this sample. Similarly to the life events measures this was not a standardised, validated measure of alliance. Further, if active discussion of problems in the therapeutic relationship did not occur then this would not be captured during coding. Additional client and therapist self-report measures of alliance may help to understand whether problems in the therapeutic relationship contributed to the worsening of symptoms seen in a depression spike. Relating our findings to the wider literature, there is an absence of studies that directly examine whether difficulty in the client-therapist relationship is correlated with exacerbations in depression symptoms in subsequent therapy sessions. Instead, the alliance literature tends to focus on the beneficial value of therapeutic alliance on *reductions* of depression symptoms in subsequent therapy sessions (Falkenström et al., 2013) and the repair of therapeutic ruptures (Eubanks et al., 2018). Further examination of problems in the therapeutic relationship can help understand not only if this may contribute to a depression spike, but also if they are associated with subsequent unfavourable depression symptoms within therapy.

From the findings of the current study it remains unclear what instigates a depression spike in non-EBCT therapies like CBT and BA. Although we found no prespike client processes or therapist strategies were associated with a depression

spike in the current sample, replication is needed. In Hayes et al.'s study greater cognitive emotional processing and reductions in avoidance occurred during a depression spike in EBCT (Hayes, Feldman, Beevers, et al., 2007). One explanation for the discrepancy between our findings and those of Hayes et al.'s is that depression spikes which occur outside of EBCT are fundamentally different as they are not intentionally brought about by the therapy. Unlike EBCT where there is a clear theoretical rationale to examine depression spikes in the middle of treatment, theoretically in CBT and BA we would not expect the middle of treatment is an especially important time to focus on. Further, because of the low number of therapy tapes available from those who experienced a depression spike in the current study, we did not restrict our investigations of depression spikes to those occurring in the middle of treatment in CBT or BA. Consequently, our lack of findings could be because we looked across the treatment period and future research may wish to focus on examining processes surrounding depression spikes in the middle of treatment. Alternatively, it may be the case that other processes, interactions between client processes and/or therapist strategies, or iatrogenic effects (Linden, 2013; Parry et al., 2016) of therapy may explain the occurrence of depression spikes in CBT and BA. Conversely, rather than change occurring within therapy it is possible that outside-therapy processes of change are occurring which may not have been captured in the current study. Other methods can be used to assess this, such as ecological momentary assessment (EMA) which can capture thoughts, emotions and depression symptoms in real time through smartphone devices or watches at repeated assessments (McDevitt-Murphy et al., 2018; Shiffman et al., 2008). EMA methods may also make it possible to capture reactivity to depression changes outside of therapy (Wenze & Miller, 2010).

One unexpected finding was that individuals who had a depression spike had a greater number of previous episodes of MDD. This may suggest that those who have a more chronic course of depression are more likely to experience a depression spike in treatment. Drawing from the sudden gains literature in a recent revision of the theory, Aderka and Shalom (2021) suggest discontinuous symptom fluctuations, such as sudden gains, may result from natural depression symptom fluctuations. Rather than being brought about by treatment they suggest that when discontinuous changes occur in the context of treatment they can be harnessed. The authors have also found that symptom variability predicts sudden gains in a range of treatments across disorders (Shalom et al., 2018; Shalom et al., 2020) and symptom fluctuations have been found to be an early warning signal that discontinuous change may occur (Olthof et al., 2020). Although speculative, this may also apply to depression spikes and suggests symptom fluctuation may be an intrinsic characteristic of this group of patients. While beyond the scope of the current study, future research may wish to examine whether depression symptom fluctuations, both pre-treatment and within treatment, are associated with depression spikes.

With regard to the association of processes at the peak of the spike and treatment outcomes, contrary to expectations, we did not find the hypothesised cognitive or behavioural, client processes or therapist strategies were associated with 12- or 18-month depression outcome, in either treatment. In this study we focused on examining process in the therapy session at the peak of the spike as this is theorised to be a rich opportunity for processing to occur during a depression spike in EBCT and emotional processing during this session has been found to be a predictor of treatment outcome (Hayes, Feldman, Beevers, et al., 2007). However it is still unclear whether depression spikes outside of EBCT are comparable to Hayes

et al.'s depression spikes in this respect, as theoretically neither CBT nor BA use therapeutic strategies to exacerbate depression symptoms to allow processing to occur. This is further illustrated within the depression spike literature where there is variability in the association between depression spikes and treatment outcomes in non-EBCT treatments with some finding beneficial (Ladwa et al., in prep; study one) and others finding unfavourable associations with treatment outcomes (O'Mahen et al., 2021). It is possible that critical processes are changing during the spike session but these differences were indiscernible between individuals who experience a depression spike and those who do not. Instead perhaps examining whether there are differences in processes between those who have a depression spike, compared to individuals who have a worsening in symptoms which does not recover (a sudden loss; Lutz et al., 2013) may elucidate important processes during this session that are associated with treatment outcomes. We also note that within a depression spike there are three points of inflexion and it is possible that any of these could be the target of investigation of adaptive and maladaptive processes of change.

This is the first study to examine processes of change in depression spike sessions in CBT and BA, and using the CHANGE coding system we were able to examine a range of client processes and therapist strategies. Nevertheless, there are a number of limitations to note. Firstly, only content that is verbalised during therapy sessions is able to be coded using the CHANGE coding system. Using both client and therapist report of processes both during and following therapy sessions may allow us to identify further important processes of change. This is particularly relevant for life events and therapeutic difficulty in the current study. Although the client and therapist variables assessed here were not associated with depression spike status or treatment outcomes at follow up, we are not able to accept the null

hypothesis with confidence as with the limited sample size it is possible that there were elevated type II errors. Additionally, similarly to other process research in therapy sessions surrounding discontinuous change (Abel et al., 2016; Alpert et al., 2021) adjustments for multiple testing were not made and thus the conclusions drawn from these results are only preliminary. Further replication is needed in larger samples.

4.6.1 Conclusion

The results of this study did not find any of the variables assessed were associated with depression spikes in CBT and BA. There is some suggestion that individuals with previous episodes of major depression may be more likely to experience a depression spike in therapy, but further examination of this is needed in larger samples. It is still unclear what depression spikes represent in therapies where there are no intended therapeutic strategies used to bring about a depression spike. Further no processes at the peak of a depression spike were found to be associated treatment outcomes. This study contributes to the limited process research in the context of depression spikes and further investigation is needed.

Chapter four Appendices

Appendix 1

Life Events Code

Definition: This category captures a life event mentioned by the client during the therapy session. The life event must have occurred since the last therapy session (or within the last week, if coding the first therapy session). A life event is defined as an event or experience that causes a significant change in a person's life or circumstance outside of therapy and that has a significant emotional impact on the client.

Note that there may be some events that cause significant change in an individual's life but have little emotional impact (e.g. a student goes back to university after the summer holidays) and there is no stress or emotional impact. On the other hand, there may be an emotional impact, but the event does not cause a significant change in the person's circumstance (e.g. it is the anniversary of a client's mother's death). In this situation there is no current change in the individual's life, but there may be a significant emotional impact. Neither of these cases would be coded as life events, as the situation must cause both a significant change in the person's life and have an emotional impact.

If an event concerns the clients own behaviour (e.g. starting an argument), then to be coded as a life event the impact must be non-trivial and not a regular occurrence. For example, a client gets into a serious argument with her boyfriend's daughter on Facebook. The argument escalates and both the client and daughter engage in personal insults, resulting in neither of them speaking to each other and a strain on the client's relationship with her boyfriend. This event resulted in a

significant change and had an emotional impact. If this instead were part of an everyday transaction or hassle (e.g. the client comments on her boyfriend's daughter's Facebook profile picture and the daughter complains about this to her father, which makes the client feel annoyed), this would not be coded as a life event. Large scale collectively experienced events (e.g. 9/11 terrorist attacks, or Brexit) can also be coded as a life event if it causes a significant change in circumstance and emotional impact to the individual.

The life event is categorised as positive or negative and as 'dependent' or 'independent'. Dependent life events are those that the client has influenced or contributed to in some way (e.g. getting a new job, eating unhealthily and being diagnosed with diabetes, or breaking up with a long-term partner). If the client's actions and/or psychopathology has influenced the outcome of an event, this should be rated as dependent even if the client did not choose the outcome. For example, a depressed client's partner breaks up with her because she has been moody and non-communicative. This upsets the client, who does not believe her behaviour warranted a breakup. Here the event would be coded as dependent because the client's psychopathology has influenced the event, irrespective of the client's choice. Independent life events are those that the client did not influence or contribute to (e.g. death of a family member, engagement of a family member, a weather-related incident).

Please take note of each different life event mentioned in the session, but only code the life event that seems to have or potentially have the most impact on the client. If the life event is mentioned over several sessions, please only code the first time the event is mentioned. It is possible for separate life events to occur that are related to the same matter. For example, in one session the client discloses that his

mother has found a lump on her breast. During the next therapy session the client says his mother has been diagnosed with cancer. At the next session the client tells the therapist that his mother has suddenly died. During each session these would be coded as separate life events.

1. Life event rating:

    1. Has the client mentioned a life event that has occurred within the last week?

0 : No	No life event has been mentioned during the therapy session
1: Yes	A life event(s) has been mentioned during the therapy session that occurred in the past week.
If yes, please list life events:	
Independent/dependent rating: Please indicate how many independent and dependent life events were mentioned in the session Independent …… Dependent ……..	

For the rest of the life event coding, please choose the event that had the most impact on the client.

2. Independent/dependent rating

Rate the extent to which the life event was independent (e.g. a thunderstorm caused a tree to fall and block the road, making the client miss her daughter's wedding dress fitting) or dependent (e.g. lost a large amount of money gambling) of the client. Please rate the event itself and not the client's reaction to the event.

Independent life event	
0	Not at all
1	A little
2	Moderately
3	Very

Dependent life event	
0	Not at all
1	A little
2	Moderately
3	Very

3. Valence rating

Separately rate how positive and negative the life event was. Contextual information can also be used to rate the valence of the event, but the rating should be based on how a typical person under identical circumstances would experience the event.

Please note that the life event can be both positive and negative (e.g. the client spoke very positively about the wedding, but there were also family disputes that were disturbing).

How **POSITIVE** was the life event?	
0	Not at all
1	A little
2	Moderately
3	Very

How **NEGATIVE** was the life event?	
0	Not at all
1	A little
2	Moderately
3	Very

4. Severity rating:

Rate the severity of the life event from your perspective as an objective rater and then based on the client's perception of the life event. Please indicate whether the life event was mild, (e.g. at a traffic light someone bumped into the client's car, leaving a dent. There were no injuries, but there was a lengthy insurance process and the client was without a car for a week whilst undergoing repairs), moderate (e.g. the client was put up for review at work and there is a possibility of being fired, although they have not been fired yet), or severe (e.g. an unexpected death of a parent).

Note: Severity is independent of valence. For example, an event can be severe and positive, such as winning a large amount of money from the lottery. Some events may be severe in intensity and evoke both positive and negative feelings. For example, a client has been promoted to her dream job at a high level (positive valence, positive severity), but there is also a high level of anxiety (negative valence).

Severity rating as an **objective rater**, taking into account the client's context:	
0	Not applicable
1	Mild
2	Moderate
3	Severe

Severity rating based on the **client's perception** of the life event:	
0	Not applicable
1	Mild
2	Moderate
3	Severe

5. Event resolution:

Please rate the extent to which the life event has been resolved (e.g. in the previous session a client stated that his online bank account had been hacked and a large amount of money had been stolen, but during the current session he says the police investigated and the money was returned by the insurance company. This event would be rated as resolved). Resolution can include developing a plan of action or coping, making meaning of the event, or acceptance of the problem.

Please indicate whether there is no resolution (e.g. a client's partner has unexpectedly left and there has been no contact), a little resolution (e.g. a client lent a large amount of money to a friend and has not been repaid. The client is struggling to keep up with their rent, but they have been lent some money by a family member), moderate (e.g. a client's partner has filed for divorce and while the client is upset, they realise that the marriage was making them both unhappy and it will be better for the children if they are apart), or resolved (e.g. a client missed an appointment with their probation officer, which could result in them going to prison. At the court hearing, the charges were overturned, and the client is free from any court proceedings).

To what extent has the event been resolved?	
0	Not resolved
1	A little resolved or moving toward resolution
2	Moderately resolved
3	Resolved

Appendix 2

Examples of CHANGE Coding System Variables

Process Variable	Example of Process Variable
Therapist variables	
Therapeutic Difficulty	A client reports being pinned to the wall in relationships and the urge to avoid. They report similar feelings in therapy and begin to miss appointments. The therapist highlights this and this becomes the focus of the session
Therapist Cognitive Corrective Information	A client believes they have to be in control in all aspects of their life and they are a failure. The therapist explores the messages he received from his parents related to themes of control and perfectionistic standards
Therapist Behavioural Corrective Information	To combat a clients' tendency to avoid, the therapist asks her to face three situations per week that she previously would avoid
Client variables	
Cognitive Emotional Processing	"Bad things still come my way but I somehow don't let it devastate me as I did before. I am starting to see bad things are not personal, it's a part of life."

Cognitive Flexibility	“I can now see why [my friend] is acting the way she is towards me. It’s not just me, which is what my go to thinking was before, but it’s how she was raised and her life situation at the moment.’
Positive Behaviour	“I did it! I finally had that conversation with my ex-husband that I was avoiding for the last couple of months. Even though it was awful and horrible, it was a big, positive step for me.”
Avoidance	“I always have to put on a happy face at work and I have to mask my feelings when I feel upset, stressed or tense… this happens everyday and I tend to go into my office and sit away from people to avoid having to put on a happy face.”

Appendix 3

Patient Demographics and Clinical Characteristics Comparing Participants who Experienced Depression Spikes and were Coded, and the Whole COBRA Sample*

	Depression Spikes (*n* = 44)			Whole COBRA sample (*n* = 223)						
Variable	*n* (%)	*M*	*SD*	*n* (%)	*M*	*SD*	χ^2	*t*	*df*	*p*
Treatment							1.976		1	.188
CBT	27 (61.4%)			111 (49.8%)						
BA	17 (38.6%			112 (50.2%						
Age (years)		45.45	14.05		44.70	14.65		-.311	265	.756
Site							11.564		2	.003**
Devon	27 (61.4%)			76 (34.1%)						
Durham	9 (20.5%)			81 (36.3%)						
Leeds	8 (18.2%)			66 (29.6%						
Antidepressant use							1.302		1	.324
Yes	37 (84.1%)			170 (76.2%)						
No	7 (15.9%)			53 (23.8%)						

Baseline PHQ-9		18.40	5.05		17.08	4.65		-1.707	265	.089
Number of previous MDD episodes		8.31	11.27		4.42	7.12		-1.977	38.94	.055
Sex							1.326		1	.300
Female	32 (72.7%)			142 (63.7%)						
Male	12 (27.3%)			81 (36.3%)						
Relationship status							1.462		1	.247
Not in a relationship	15 (34.1%)			98 (43.9%)						
In a relationship	29 (65/9%)			125 (56.1%)						
Ethnicity							.000		1	1.000
Caucasian	41 (93.2%)			208 (93.3%)						
Other	3 (6.8%)			15 (6.7%)						
Education							3.840		2	.159
No qualifications	8 (18.2%)			19 (8.5%)						
Secondary School	23 (52.3%)			135 (60.5%)						
Degree	13 (29.5%)			69 (30.9%)						

Note. CBT= cognitive behavioural therapy; BA= behavioural activation; PHQ-9 = Patient Health Questionnaire 9; MMD = Major Depressive Disorder.

Appendix 4

Patient Demographics and Clinical Characteristics Comparing Participants who Experienced Depression Spikes and were Coded, Compared to those who Experienced Depression Spikes and were Not Coded

	Coded Depression Spikes (*n* = 44)			Non-coded Depression Spikes (*n* = 33)						
Variable	*n* (%)	*M*	*SD*	*n* (%)	*M*	*SD*	χ^2	*t*	*df*	*p*
Treatment							1.925		1	.247
CBT	27 (61.4%)			15 (45.5%)						
BA	17 (38.6%)			18 (54.5%)						
Age (years)		45.45	14.05		41.15	12.82		-1.380	75	.172
Antidepressant use							2.271		1	.169
Yes	37 (84.1%)			23 (69.7%)						
No	7 (15.9%)			10 (30.3%)						
Baseline PHQ-9		18.41	5.06		17.33	5.12		-.919	75	.361
Number of previous MDD episodes		8.31	11.27		847	12.76		.051	63	.959
Number of treatment sessions		18.64	5.04		17.15	5.45		-1.235	75	.221

Sex			.726	1	.460
Female	32 (72.7%)	21 (63.6%)			
Male	12 (27.3%)	12 (36.4%)			
Relationship status			1.624	1	.244
Not in a relationship	15 (34.1%)	16 (48.5%)			
In a relationship	29 (65.9%)	17 (51.5%)			
Ethnicity			.136	1	1.00
Caucasian	41 (93.2%)	30 (90.9%)			
Other	3 (6.8%)	3 (9.1%)			
Education			5.553	2	.056
No qualifications	8 (18.2%)	1 (3%)			
Secondary School	23 (52.3%)	16(48.5%)			
Degree	13 (29.5%)	16 (48.5%)			

Note. CBT= cognitive behavioural therapy; BA= behavioural activation; PHQ-9 = Patient Health Questionnaire 9; MMD = Major Depressive Disorder.

Appendix 5

Separate Binary Logistic Regression Analyses Examining Processes Associated with Depression Spike Status (0, 1)

	Exp(B)	95% CI
Prespike Therapist cognitive corrective information		
Prespike BDI	0.99	0.95, 1.04
Prespike Therapist cognitive corrective information	1.15	0.65, 1.99
Treatment	1.09	0.43, 2.79
Prespike Therapist cognitive corrective information x Treatment	0.58	0.16, 2.19
Prespike Cognitive-Emotional		
Prespike BDI	0.99	0.95, 1.04
Prespike Cognitive-Emotional Processing	0.63	0.36, 1.11
Treatment	0.99	0.41, 2.38
Prespike Cognitive-Emotional Processing x Treatment	1.38	0.44, 4.36
Prespike cognitive flexibility		
Prespike BDI	0.99	0.95, 1.04
Prespike cognitive flexibility	0.79	0.39, 1.61
Treatment	0.98	0.41, 2.33
Pre-spike Cognitive flexibility x Treatment	1.37	0.29, 6.32
Prespike Therapist behavioural corrective information		

Pre-spike BDI	0.99	0.95, 1.04
Pre-spike Therapist behavioural corrective information	1.27	0.71, 2.27
Treatment	0.86	0.33, 2.19
Prespike Therapist behavioural corrective information x Treatment	0.14**	0.03, 0.56
Pre-spike Positive behaviour		
Prespike BDI	0.99	0.95, 1.04
Prespike Positive behaviour	0.86	0.49, 1.52
Treatment	1.04	0.43, 2.51
Pre-spike Positive Behaviour x Treatment	0.93	0.29, 2.06
Pre-spike Avoidance		
Prespike BDI	0.99	0.95, 1.04
Prespike Avoidance	1.61	0.93, 2.77
Treatment	0.82	0.33, 2.03
Pre-spike Avoidance x Treatment	0.69	0.25, 1.91
Prespike Therapeutic Difficulty		
Prespike BDI	0.99	0.95, 1.04
Prespike Therapeutic Difficulty	0.92	0.40, 2.05
Treatment	1.02	0.41, 2.50
Pre-spike Therapeutic Difficulty x treatment	3.27	0.54, 19.62

Note. CI = confidence interval; CBT = cognitive behavioural therapy; BA = behavioural activation. *p <.05, ** p <.01, *** p <.001

Appendix 6

Spike Process Separate Linear Regressions on 12 and 18 month Post-randomisation PHQ-9 score

	12 month PHQ-9 Scores				18 month PHQ-9 Scores			
Model	*B*(se)	*t*	*p*	95% CI	*B*(se)	*t*	*p*	95% CI
Therapist cognitive corrective information								
Constant	-.660(.25)	-2.675	.009**	-1.15, -.169	-0.68(0.24)	-2.79	.007**	-1.16, -0.19
Prespike BDI Score	.034(.01)	2.959	.004**	.011, .058	0.04(0.01)	3.08	.003**	0.01, 0.05
Depression Spike	.539(.21)	2.531	.013*	.115, .963	0.28(0.22)	1.313	.193	-0.14, 0.71
Treatment	-.146(.24)	-.612	.543	-.619, .328	0.04(0.24)	0.158	.875	-0.44, 0.52
Spike Therapist cognitive corrective information	.191(.16)	1.180	.242	-.131, .514	0.07(0.16)	0.405	.686	-0.26, 0.39
Depression Spike x Treatment	-.491(.51)	-.967	.337	-1.504, .521	0.44(0.28)	1.54	.126	-0.12, 1.02
Spike Therapist cognitive corrective information x Treatment	.295(.38)	.775	.441	-4.62, 1.052	0.64(0.34)	1.858	.067	-0.04, 1.33
Spike Therapist cognitive corrective information x Depression Spike	.406(.34)	1.181	.241	-.279, 1.090	0.54(0.30)	1.78	.079	-0.06, 1.14

Spike Therapist cognitive corrective information x Treatment x Depression Spike	.333(.86)	.386	.701	-1.39, 2.05	-0.45(0.67)	-0.67	.501	-1.81, 0.89
Cognitive emotional processing								
Constant	-.660(.25)	-2.675	.009**	-1.15, -.169	-0.68(0.24)	-2.79	.007**	-1.16, -0.19
Prespike BDI Score	.034(.01)	2.959	.004**	.011, .058	0.04(0.01)	3.08	.003**	0.01, 0.05
Depression Spike	.474(.21)	2.269	.026*	.058, .891	0.27(0.20)	1.320	.191	-0.14, 0.69
Treatment	-.036(.22)	-.164	.870	-.468, .397	0.09(0.21)	0.444	.658	-0.33, 0.52
Spike Cognitive emotional processing	-.094(.15)	-.625	.534	-.392, .205	0.16(0.15)	1.045	.299	-0.14, 0.45
Depression Spike x Treatment	-.226(.47)	-.483	.630	-1.158, .706	0.48(0.29)	1.668	.099	-0.09, 1.06
Spike Cognitive emotional processing x Treatment	.054(.34)	.155	.877	-.633, .740	0.27(0.33)	0.81	.416	-0.39, 0.93
Spike Cognitive emotional processing x Depression Spike	-.021(.34)	-.063	.950	-.697, .654	0.40(0.32)	1.22	.224	-0.25, 1.05
Spike Cognitive emotional processing x Treatment x Depression Spike	.150(.77)	.196	.845	-1.37, 1.676	-0.74(0.61)	-1.218	.227	-1.94, 0.47
Cognitive flexibility								

Constant	-.660(.25)	-2.675	.009**	-1.15, -.169	-0.68(0.24)	-2.79	.007**	-1.16, -0.19
Prespike BDI Score	.034(.01)	2.959	.004**	.011, .058	0.04(0.01)	3.08	.003**	0.01, 0.05
Depression Spike	.488(.21)	2.334	.022	.072, .905	0.26(0.21)	1.238	.219	-0.15, 0.68
Treatment	-.029(.22)	-.134	.894	-.461, .403	0.08(0.21)	0.37	.708	-0.35, 0.51
Spike Cognitive flexibility	.105(.20)	.519	.605	-.297, .507	-0.03(0.21)	-0.14	.882	-0.43, 0.37
Depression Spike x Treatment	-.297(.44)	-.671	.504	-1.181, .586	0.46(0.29)	1.55	.124	-0.12, 1.05
Spike Cognitive flexibility x Treatment	.245(.45)	.542	.590	-.655, 1.145	0.02(0.45)	0.05	.956	-0.87, 0.93
Spike Cognitive flexibility x Depression Spike	.221(.41)	.536	.594	-.602, 1.044	0.18(0.41)	0.46	.647	-0.64, 1.02
Spike Cognitive flexibility x Treatment x Depression Spike	.765(.90)	.849	.399	-1.03, 2.56	-0.45(0.77)	-0.58	.564	-1.98, 1.08
Therapist behavioural corrective information								
Constant	-.660(.25)	-2.675	.009**	-1.15, -.169	-0.68(0.24)	-2.79	.007**	-1.16, -0.19
Prespike BDI Score	.034(.01)	2.959	.004**	.011, .058	0.04(0.01)	3.08	.003**	0.01, 0.05
Depression Spike	.509(.20)	2.490	.015*	.102, .916	0.27(0.21)	1.323	.190	-0.14, 0.69
Treatment	-.204(.23)	-.886	.378	-.661, .254	-0.004(0.24)	-0.19	.985	-0.47, 0.46

Spike Therapist behavioural corrective information	-.337(.17)	-2.001	.050	-.673, .002	-0.15(0.17)	-0.91	.367	-0.29, 0.18
Depression Spike x Treatment	-.075(.49)	-.155	.878	-1.048, .897	0.62(0.30)	2.03	.045*	0.14, 1.22
Spike Therapist behavioural corrective information x Treatment	-.261(.43)	-.608	.545	-1.115, .594	0.06(0.39)	0.17	.865	-0.72, 0.85
Spike Therapist behavioural corrective information x Depression Spike	.094(.36)	.259	.797	-.628, .816	0.29(0.33)	0.87	.386	-0.37, 0.96
Spike Therapist behavioural corrective information x Treatment x Depression Spike	-.622(.86)	-.724	.471	-2.33, 1.09	-0.34(0.37)	-0.90	.369	-1.09, 0.41
Positive behaviour								
Constant	-.660(.25)	-2.675	.009**	-1.15, -.169	-0.68(0.24)	-2.79	.007**	-1.16, -0.19
Prespike BDI Score	.034(.01)	2.959	.004**	.011, .058	0.04(0.01)	3.08	.003**	0.01, 0.05
Depression Spike	.473(.21)	2.249	.027*	.054, .893	0.25(0.21)	1.200	.234	-0.16, 0.68
Treatment	-.037(.22)	-.170	.866	-.475, .400	0.07(0.22)	0.34	.733	-0.35, 0.51
Spike Positive behaviour	-.049(.15)	-.327	.745	-.346, .248	-0.04(0.14)	-0.24	.813	-0.32, 0.25
Depression Spike x Treatment	-.290(.45)	-.640	.524	-1.195, .614	0.51(0.29)	1.70	.093	-0.08, 1.10

Spike Positive behaviour x Treatment	.033(.33)	.101	.920	-.615, .681	0.16(0.31)	0.52	.602	-0.45, 0.78
Spike Positive behaviour x Depression Spike	-.066(.32)	-.206	.838	-.705, .573	0.12(0.31)	0.38	.698	-0.49, 0.73
Spike Positive behaviour x Treatment x Depression Spike	-.283(.66)	-.425	.672	-1.61, 1.044	-0.24(0.41)	-0.58	.562	-1.06, 0.58
Avoidance								
Constant	-.660(.25)	-2.675	.009**	-1.15, -.169	-0.68(0.24)	-2.79	.007**	-1.16, -0.19
Prespike BDI Score	.034(.01)	2.959	.004**	.011, .058	0.04(0.01)	3.08	.003**	0.01, 0.05
Depression Spike	.45(.21)	2.121	.037*	.028, .873	0.25(0.21)	1.18	.238	-0.17, 0.68
Treatment	-.025 (.22)	-.114	.910	-.455, .406	0.08(0.21)	0.37	.712	-0.35, 0.51
Spike Avoidance	.099(.13)	.733	.442	-.155, .352	0.03(0.13)	0.22	.826	-0.23, 0.28
Depression Spike x Treatment	-.42(.45)	-.922	.359	-1.313, .482	0.52(0.29)	1.80	.076	-0.05, 1.10
Spike Avoidance x Treatment	-.123(.27)	-.449	.654	-.667, .422	-0.54(0.26)	-2.04	.044*[8]	-1.07, -0.01
Spike Avoidance x Depression Spike	.209(.25)	.829	.410	-.293, .710	0.04(0.24)	0.15	.877	-0.45, 0.53

[8] Explications of this two-way spike avoidance by treatment interaction revealed that there were no main effects of avoidance within either treatment (BA, *b*(*se*) = 0.31(0.16), *t* = 1.89, *p* = .068, 95% CI -0.02, 0.65; CBT, *b*(*se*) = -0.03(0.18), *t* = -0.16, *p* = .867, 95% CI -0.39, 0.33).

Spike Avoidance x Treatment x Depression Spike	.004(.55)	.007	.995	-1.085, 1.093	-0.30(0.29)	-1.03	.303	-0.88, 0.28

Note. BDI = beck depression inventory; BA = behavioural activation; CBT = cognitive behavioural therapy.

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

Appendix 7

Spike Negative Life Events and Therapeutic Difficulty Analyses on Treatment Outcomes

An ANOVA showed negative life events measured at the spike session were not associated with either 12- ($F(1, 75) = .739$, $p = .569$, $\eta_p^2 = .038$) nor 18-month ($F(1, 77) = .163$, $p = .957$, $\eta_p^2 = .008$) treatment outcome. Due to the low numbers of negative life events we were not powered to examine whether depression spike status (0/1) or treatment type (CBT/BA) moderated these relationships.

Separate linear hierarchical regression models were conducted to examine whether therapeutic relationship difficulty during the spike session were associated with treatment outcomes and 12- and 18-months treatment outcome. Similar to the other process regression models outline in the main body of the study, in step one pre-spike BDI depression score was entered to account for depression severity prior to the depression spike. The main effects of treatment type (CBT/BA), depression spike status (0, 1), and spike therapeutic relationship difficulty were entered in step two. In the third step the two-way interactions were entered, and the three-way interactions between treatment type, depression spike status and spike therapeutic relationship difficulty was entered in fourth step. The dependent variable was 12- or 18-month PHQ-9 score with a Box-Cox transformation applied. The results are in the table below.

Similar to the regression models in the main body of the paper, there was a main effect of depression spike status on 12-month treatment outcome which indicated individuals who had a depression spike, compared to those who did not, has higher PHQ-9 scores at 12 month outcome. There were no other significant

main effects. There were no significant two-way interactions, nor was the three-way interaction between depression spike status, treatment type and spike therapeutic relationship difficulty significant on 12 month treatment outcome.

There were no significant main effects, two-way, or three way interactions from the hierarchical linear regression model for spike therapeutic relationship difficulty on 18-month outcome.

Linear Regression Models Examining the Association between Therapeutic Relationship Difficulty and Treatment Outcomes

	12 month PHQ-9						18 month PHQ-9					
	B(*se*)	*t*	*p*	95% CI	R^2adj	$R^2\Delta$	*B*(*se*)	*t*	*p*	95% CI	R^2adj	$R^2\Delta$
Step 1					.087	.099**					.093	.104**
Constant	-.66(.25)	-2.675	.009**	-1.15, -.02			-.69(.24)	-2.780	.007**	-1.16, -.19		
Prespike BDI Score	.034(.01)	2.959	.004**	.011, .058			.035(.01)	3.081	.033**	.01, .05		
Step 2					.143	.086					.079	.019
Depression Spike	.476(.21)	2.316	.023*	.067, .886			.042(.75)	.057	.955	-1.4,1.53		
Treatment	-.039(.21)	-.182	.856	-.464, .387			.161(.33)	.493	.623	-.49, .81		
Spike Therapeutic relationship difficulty	.348(.21)	1.638	.106	-.075, .770			.054(.22)	.345	.807	-.38,0.49		
Step 3					.147	.036					.072	.027
Depression Spike x Treatment	-.179(.43)	-.413	.681	-1.04, .68			.166(.44)	.378	.706	-.70,1.04		

Spike Therapeutic relationship difficulty x Treatment	-.719(.49)	-1.451	.151	-1.71, .26			-.70(.51)	-1.386	.170	-1.71,.30		
Spike Therapeutic relationship difficulty x Depression Spike	.454(.44)	1.044	.151	-1.70, .26			-.29(.45)	-.631	.530	-1.18,.61		
Step 4					.140	.004					.080	.019
Spike Therapeutic relationship difficulty x Treatment x Depression Spike	.636(1.02)	.626	.533	-1.39, 2.66			1.36 (1.04)	1.310	.194	-.71, 3.45		

Chapter Five: When putting it off until tomorrow hurts- the association of cognitive and behavioural avoidance with depression during stressful life events

Study 4

Asha Ladwa [a], Kim Wright [a], and Heather O'Mahen [a]

[a] Mood Disorders Centre, University of Exeter, Washington Singer Building, Exeter, EX4 4QG, UK

5.1 Preface

Studies two (chapter three) and three (chapter four) in this thesis examined important depressionogenic processes that may be associated with depression symptom discontinuities. The majority of this literature examines processes of change in relation to symptom discontinuities in psychotherapy settings, however it is also the case that depression symptom fluctuations occur outside of treatment (Shalom et al., 2018; Shalom et al., 2020). Experiencing fluctuations outside of treatment has been found to be associated with within treatment sudden gains (Aderka & Shalom, 2021). In a recent paper, Aderka and Shalom (2021) highlighted that depression symptoms may vary around a constant mean outside of treatment, but the context of therapy increases the chance to create a rapid reduction of symptoms, leading to a sudden gain. Little research has explored important key depressionogenic maintenance processes during depression symptom fluctuations outside of treatment.

Avoidance is one key depressionogenic process that is a risk (Grant et al., 2013) and maintenance (Trew, 2011) factor for depression, and therefore is an important therapeutic target in both CBT and BA. In study two (chapter three) we found individuals who experienced a sudden gain had lower levels of avoidance in the postgain session (when their depression symptoms were lowered). Much of the literature examining avoidance and depression looks at how avoidance may influence depression, but the results of study two are consistent with the behavioural theory of depression which hypothesises that depressive symptoms also influence levels of avoidance behaviour. Furthermore, avoidance is a multifaceted construct and can occur in both the cognitive and behavioural domain. The current study was split into two parts. The first part developed an exam specific avoidance

questionnaire and the second part focused on examining the reciprocal prospective impact of cognitive and behavioural avoidance and depressive mood across a stressful period outside of treatment in which we may expect to see natural variability in depression symptoms; final year university examinations.

5.2 Abstract

Background: The association between avoidance and depression has been widely observed, but it is unclear how behavioural and cognitive avoidance differentially influence depression during times of depression symptom variability. Further, most research examines the relationship between avoidance and depression but less so the impact that depression may have on avoidance. The current study firstly developed an exam specific avoidance questionnaire and then investigated the reciprocal associations between cognitive and behavioural avoidance and depression symptoms during a naturally occurring life stressor which would be expected to promote variation in mood and avoidance; final year university examination period.

Method: Non-clinical undergraduate students (*N* = 81) completed measurements of exam specific cognitive and behavioural avoidance and depression symptoms over three points during the final year examination period; before exams during revision (T1; preparation period), after exams but before receiving results (T2; anticipatory period), and after receiving results (T3; recovery period).

Results: There were fluctuations in depression symptoms and cognitive and behavioural avoidance over the exam period. Cross-lagged analyses showed cognitive avoidance predicted greater prospective depression symptoms over the exam period from T1 to T2 and from T2 to T3. Behavioural avoidance at T2 prospectively predicted greater depression at T3 only. Furthermore, at each time point greater depressed mood prospectively predicted greater behavioural and cognitive avoidance.

Conclusion: Periods of transient stress, like examination periods, can be utilised to examine reciprocal relationships between depressionogenic processes, like cognitive and behavioural avoidance, depressive symptoms outside of therapy. This study highlights the importance of looking at how maladaptive processes evolve over time.

Keywords: cognitive avoidance, behavioural avoidance, depressive symptoms

5.3 Introduction

Depression is a debilitating mental health problem characterised primarily by intense sadness and loss of interest. It is estimated to affect over 300 million people worldwide (WHO, 2017) and is the leading cause of disability worldwide (Friedrich, 2017). The course of depression is often recurrent and of the individuals that do recover more than 50% will relapse within two years (Cuijpers et al., 2021; Cuijpers et al., 2008; Vittengl et al., 2007). Therefore it is important to understand the processes that contribute to the development and maintenance of depressive symptoms.

One way in which depression may develop and be maintained is through ineffective emotion regulation. Emotion regulation strategies refer to processes through which individuals respond to and modify their emotions (Aldo et al., 2010; Joormann & Stanton, 2016) in relation to environmental demands (Gross & Muñoz, 1995). One key maladaptive emotion regulation strategy that is both a risk (Dobson et al., 2014; Grant et al., 2013) and a maintenance factor (Ottenbreit & Dobson, 2004; Trew, 2011) for depression is avoidance. Avoidance is a multifaceted construct (Ottenbreit & Dobson, 2008) that can be referred to as a response style (e.g. worry or rumination; Nolen-Hoeksema, 2000) or a coping style (Ottenbreit & Dobson, 2004) and can be conceptualised as either behavioural or cognitive in nature. Behavioural avoidance refers to responses aimed at escaping or refraining behaving in direct response to a stressor or engaging in activities that relieve tension of negative feelings, whereas cognitive avoidance includes efforts aimed at avoiding thinking about the problem or supressing thoughts (Cronkite & Moos, 1995; Ottenbreit & Dobson, 2004). Avoidant coping styles aim to minimise specific stressors (Moos & Schaefer, 1993; Ottenbreit & Dobson, 2004). In the short term this

can be beneficial and provide temporarily relief of stress, but in the long term avoidance coping can lead to exacerbation of problems which may lead to depression (Dobson & Dozois, 2008; Jacobson et al., 2001; Martell et al., 2001; Trew, 2011). Avoidance is also highlighted in theories of depression and an important target in psychotherapy. For instance, the behavioural model of depression suggests that avoidance contributes to depression by limiting an individual's exposure to sources of positive reinforcement (Martell et al., 2001). As an individual escapes aversive stimuli, avoidant behaviours are negatively reinforced and with greater avoidance and less positive reinforcement this results in a vicious cycle of low mood and further avoidance (Martell et al., 2001). Therefore behavioural treatments for depression (e.g. Behavioural Activation, BA) aim to increase activation and reduce avoidance to positively reinforcing valued activities (Martell et al., 2001). Conversely, cognitive avoidance can also function to escape negative affect, in the form of rumination (Martell et al., 2001), and may encourage negative processing biases and limit exposure to positively rewarding experiences (Trew, 2011). Although avoidance is more explicitly discussed in behavioural therapies, in cognitive therapies avoidance is indirectly targeted through activity scheduling, problem solving, and thought challenging. Further, avoidance may also be directly addressed within conditional assumptions work where compensatory behaviours are revised.

There is considerable research showing links between avoidance and depression. Cross sectional research has shown that both cognitive and behavioural avoidance and depression symptoms are correlated in non-clinical student samples (Carvalho & Hopko, 2011; Cribb et al., 2006; Kroska et al., 2017; Moulds et al., 2007; Ottenbreit & Dobson, 2004; Penland et al., 2000). Longitudinal research has also demonstrated associations between avoidance and depression. For example, in a

study examining the long term consequences of maladaptive avoidant coping in a community adult sample, Holahan et al. (2005) found baseline avoidance coping style, which included cognitive avoidance, was indirectly associated with depressive symptoms 10 years later via life stressors. Some research also suggests that there are differences between cognitive and behavioural avoidant styles and depression. For instance in a clinical sample, behavioural avoidance was indirectly associated with depression through reduced positive reinforcement (Brockmeyer et al., 2015). In adult men and women, Wagener et al. (2016) found behavioural avoidance positively predicted concurrent depression symptoms. Another study found greater cognitive avoidant coping in women only in a student community sample was associated with increased prospective depression symptoms, but behavioural avoidance coping was unrelated to depression symptom changes over a three week period (Blalock & Joiner, 2000). In an adolescent sample over a seven day period, cognitive but not behavioural avoidance, predicted increases in subsequent sadness (Dickson et al., 2012). The research shows that avoidance prospectively predicts depression and also suggests that behavioural and cognitive avoidance may differentially impact on depression symptoms. The majority of research focuses on either cognitive or behavioural avoidance and the impact on depression symptoms, and further research is needed to help us understand how and when each subtype of avoidance may influence depression.

It is also the case that, typically, longitudinal research focuses on examining the prospective relationships between avoidance on subsequent depression symptoms, suggesting that avoidance temporally precedes depression symptoms (Ottenbreit & Dobson, 2008). However, depression may also influence avoidant coping. A study by Grant et al. (2013) examined prospective, bi-directional

relationships between cognitive and behavioural avoidance and depression symptoms in an undergraduate sample across two time points with an eight week gap. They found greater behavioural avoidance at time one predicted greater depression symptoms at time two, *and* greater depression symptoms at time one predicted greater behavioural avoidance at time two. Anxiety symptoms, but not depression, predicted subsequent cognitive avoidance symptoms, and cognitive avoidance predicted anxiety but not depression symptoms. This lends support to behavioural theories of depression which suggest that increased avoidance reduces positive reinforcement and leads to depression symptoms, but also that greater depression symptoms may lead to subsequent avoidance (Martell et al., 2001; Trew, 2011). This study highlights the importance of examining the reciprocal relationships between avoidance and depression over time to help elucidate how one may influence the other.

One way in which to examine the relationships between avoidance and depression (and vice versa) is to look over periods of depression symptom variability. Within the psychotherapy literature, there has been a focus on examining how important depressionogenic processes may influence discontinuous patterns of depression symptom change to further understand how treatments work. Various patterns of discontinuous changes have been identified including rapid improvements (Tang & DeRubeis, 1999), rapid deterioration (Lutz et al., 2013) and temporary worsening (Hayes, Feldman, Beevers, et al., 2007) in depression symptoms, and process research has examined adaptive and maladaptive (e.g. Abel et al., 2016; Hayes et al., 2005; Hayes, Feldman, Beevers, et al., 2007; Lutz et al., 2013; Yasinski et al., 2019) processes around these patterns of depression change. The majority of this research has examined processes over discontinuous

depression change in therapy, but depression symptom variability also occurs outside of therapy (Aderka & Shalom, 2021; Shalom et al., 2018). This was recently highlighted by Aderka and Shalom (2021) who note that even in the absence of treatment individuals experience natural depression symptom fluctuations. The authors suggest that in the context of therapy these depression symptom fluctuations may begin to reduce to create sudden reductions of symptoms and this in turn influences processes which lead to better treatment outcomes (Aderka & Shalom, 2021). Understanding how key depressionogenic processes, like cognitive and behavioural avoidance, influence depression changes outside treatment can help us to understand how maladaptive processes may influence vulnerability to, and development of depression. Although the avoidance and depression relationship has been examined outside of therapy (e.g. Moulds et al., 2007; Ottenbreit & Dobson, 2004, 2008; Wagener et al., 2016) there has been little research examining how depression symptoms and avoidant coping might unfold longitudinally over the course of typical stressful events, including across the period of time in which the stressful event resolves and there may be less pressure to engage in avoidant coping.

Therefore, the current study prospectively examined the reciprocal relationships between cognitive and behavioural avoidance and depression across a potentially stressful event; undergraduate examinations. University examination periods, for some, can be salient goal striving events which may be likely to produce stress and impact on self-regulatory coping strategies (Carver et al., 2008), and therefore be a periods of naturally occurring heightened stress during that we may expect to see variation in depression symptoms, as well as avoidance. Several studies have used undergraduate examinations as stressful events to examine how

processes change with stress and depression (Trueba et al., 2013; Vanderhasselt et al., 2016; Vanderhasselt et al., 2014) but they do so by examining levels at a baseline period, during the event and/or post event. The current study differs from those by looking at specific times within the stressful event to see if cognitive and behavioural avoidance differentially influences depression, or vice versa, at different times of the event. This is comparable to the psychotherapy discontinuities literature where processes are examined before, during and after change in depression symptoms (e.g. Abel et al., 2016; Lemmens et al., 2021; Wucherpfennig, Rubel, Hofmann, et al., 2017). In the current study we looked to see whether there would be differences in avoidance coping during different demands of the situation; before examinations during revision (the 'preparation period'; T1), after exams but before receiving results ('anticipatory period'; T2) and after receiving results ('recovery period', T3).

Firstly, we expected there would be variability in depression, cognitive and behavioural avoidance over the exam period and we explored this.

Next, we assessed the longitudinal associations between avoidance and depression symptoms over the examination period. In line with previous literature (e.g. Blalock & Joiner, 2000; Holahan et al., 2005; Wagner et al., 2012) we hypothesised that there would be positive prospective associations between cognitive or behavioural avoidance and depression at each time point. We explored differences between behavioural and cognitive avoidance's impact on mood at specific times over the stressful period. Further, in line with the behavioural theory of depression we also expected that greater depression at each point would be associated with prospective cognitive and behavioural avoidance.

Lastly, we expected that behavioural avoidance, more so than cognitive avoidance, during the examination preparation period would have a real-world impact on performance due to insufficient preparation. Therefore we hypothesised that behavioural avoidance during the preparation period (T1) would be associated with reduced likelihood of meeting, versus not meeting, expectations of examination outcomes at T3. We also explored the association between cognitive avoidance at T1 and meeting, versus not meeting, expectations of examination outcome at T3. Additionally, in keeping with the behavioural theory of depression which proposes that avoidance behaviour negatively impacts upon depression in part due to its negative effects on the individual's context, we hypothesised that not meeting, compared to meeting, examination grade expectations at T3 would be associated with increased depression levels at T3.

This study contains two parts. The first part of the study focused on developing an avoidance questionnaire that specifically relates to the examination period. The second part focused on the reciprocal relationship between avoidance and depression over the examination period.

5.4 Methods

5.4.1 Participants

Participants were recruited through posters and handouts (Appendix 1) distributed at the University of Exeter and on Facebook, as well as online through the recruitment platform, Prolific. Individuals were eligible for the study if they were over 18 years of age, a current undergraduate student in their final year of study at a university in the United Kingdom (UK), and fluent in English.

An a-priori power analysis in GPower (Faul et al., 2007) indicated for three repeated measurements with a medium effect size (f^2 = 0.15) and 80% power a sample size of 78 was required. Ethical approval for the study was obtained from the Psychology Ethics Committee at the University of Exeter (eCLESPsy000927).

5.5 Part One – Exam Avoidance Questionnaire Development

The exam avoidance questionnaire EAQ (Appendix 2) was developed to assess cognitive and behavioural avoidance specific to examinations at each of the three stages of interest (preparation, anticipatory and recovery period), and was based on the Cognitive and Behavioural Avoidance Scale (CBAS; Ottenbreit & Dobson, 2004). The CBAS is a 35 item self-report measure and assesses cognitive social (CS) and non-social (CN), and behavioural social (BS) and non-social (BN) avoidance. Items for the EAQ were loosely based on the CBAS items but were modified to be related to examinations. For example, an item from the BN subscale on the CBAS, 'Rather than getting out and doing things, I just sit at home and watch TV' was modified to 'I find myself watching TV or surfing the internet rather than revising for the exam(s)' for the EAQ. Similarly to the CBAS, the EAQ items were scored on a five-point Likert scale (1, not at all true for me; 5, extremely true for me). However, the EAQ differs from the CBAS and also includes positive, approach cognitive and behavioural based items, e.g. 'I think about the positives rather than worry about revision and the exam(s)'. Further, at each of the three time points the items of the EAQ slightly differ to reflect that stage in the exam period. For instance, one behavioural item at T1 (preparation stage) is 'I avoid revision', whereas at T2 (anticipatory period) this item is 'I avoid any reminders of the exam(s)'. At each time point there were cognitive and behavioural items, counterbalanced with five avoidance and five reversed scored approach items, resulting in 20 items. Higher

scores indicated greater avoidance. Examples of other items included, ‘I try not to think about revising for the exam(s)’ (cognitive avoidance), ‘I imagine being successful in my revision and exam(s)’ (cognitive approach), ‘I put off revising until it’s too late’ (behavioural avoidance), and ‘I sit down and revise’ (behavioural approach). Participants of the study were asked to complete the EAQ at three time points (the preparation, anticipatory and recovery period) across the examination period.

5.5.1 Analysis

An exploratory factor analysis (EFA) was conducted on the 20 items of the EAQ. To assess whether EFA was appropriate for the data the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy and Bartlett’s Test of Sphericity were examined. KMO values range from 0-1, where values close to 1 indicate the patterns of correlations are compact and therefore should produce distinct factors. Multicollinearity amongst variables was examined using the Determinant and correlations. Only factor loadings of greater than .3 were considered. The EFA was conducted with an oblique rotation (direct oblimin) which allowed extracted factors to be correlated (Browne, 2001). When deciding how many factors to extract, Kaiser’s criterion of extracting eigenvalues over 1 (Kaiser, 1960) can lead to over- or under-extraction of factors (Zwick & Velicer, 1986) and determining the point of inflexion on scree plots (Cattell, 1966) can be subjective and can also lead to under- or over-extraction. Therefore, alongside visual examination of the scree plot, Parallel analysis (Horn, 1965) was conducted to determine the number of factors to extract. Parallel analysis tests the probability that a factor is due to chance by comparing observed eigenvalues to reference eigenvalues generated from random data (data without the factor structure) that is simulated from the data (Braeken & van Assen,

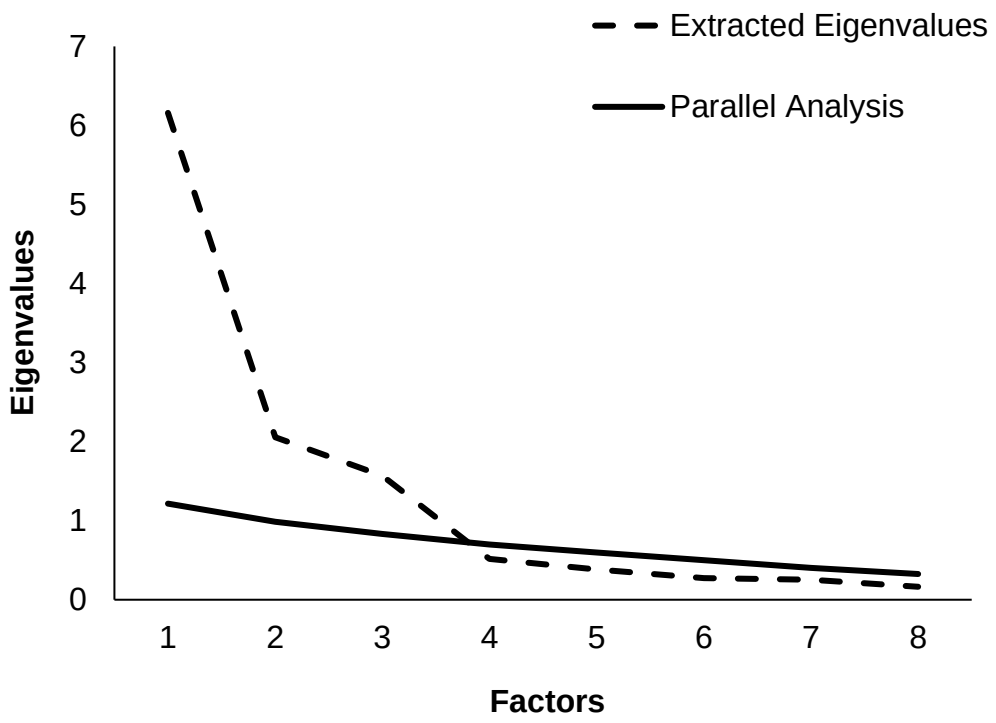
2017). Following this, the Cronbach's alpha was examined for each factor to check whether deleting any items would yield higher reliability. The factor analysis used the time one EAQ, but because the questions vary slightly at each time point an EFA was run for the questionnaire at time two and three to check the number of extracted factors matched, by visually examining the scree plot. The extracted factors were then used for subsequent analyses.

5.5.2 Results

An EFA with an oblique (direct oblimin) rotation showed the KMO was .841 which indicates there was a good sample size making the data suitable for EFA. Bartlett's Test of Sphericity was significant (χ^2 = 1074.567, p = <.001) indicating the correlations between the items were significantly significant from 0, but the Determinant (1.635E-5) and the correlations between items, which were all below 0.90, showed there was no significant issues with multicollinearity between the items. The scree plot indicated a four-factor solution should be extracted. A parallel analysis was conducted, with 100 permutations of the data and average eigenvalues with 95% confidence intervals were produced. Figure 5.1 shows a comparison of the extracted eigenvalues and parallel analysis eigenvalues. Because the eigenvalues within the EFA extraction were all below the eigenvalues generated by the parallel analysis, this suggests a four-factor solution should be retained. To check the stability of the factors we selected a random 50% of the data and then performed the EFA again, which again suggested that four factors should be extracted and these factors were stable. Additionally, because the items in this questionnaire vary slightly across the time points of the study the EFA was run with data from anticipatory (T2) and recovery (T3) period to check the factor solution. Visual examination of the scree plots suggested a four factor solution should be retained for the study.

Figure 5. 1

A Visual Comparison of the Eigenvalues in the EFA Extraction Compared to the Eigenvalues Obtained in the Parallel Analysis for the Exam Avoidance Questionnaire in the Preparation Period (Time 1)



Note. EFA = Exploratory Factor Analysis

The final factor solution consisted of four factors from 16 items. The factor loadings can be found in Table 5.1. The first factor with four items related to behavioural approach, e.g. ‘I sit down and revise’ (item 12). The second factor had three items and related to worry, e.g. I get caught up in my worries about revision (item 2). The third factor contained five items related to avoidance and encompassed both cognitive (item 1; ‘I try not to think about revising for the exam(s)’) and behavioural (item 8; ‘I avoid revision’) avoidance. The final factor had four items

which related to being cognitively proactive (item 4; ‘I think constructively about revising for the exam(s)’).

Table 5. 1

Exploratory Factor Analysis for a Four Factor Solution with Oblique Rotation for Time 1 data

Item	Factor 1: Behavioural approach	Factor 2: Worry	Factor 3: Avoidance	Factor 4: Cognitive approach
I can get back into the flow of revision after I have taken a break	.372			
I am sticking more or less to my plan for revising	.454			
I imagine being successful in my revision and exam(s)				-.458
I break down revision tasks and do them one by one				-.492
I think about the positives rather than worry about revision and the exam(s)		-.554		
I put off revising until it’s too late			.575	
I think constructively about revising for the exam(s)				-.582
I try not to think about revising for the exam(s)			.606	
I find myself watching TV or surfing the internet rather than revising for the exam(s)			.645	
I will find other jobs to do rather than revising for the upcoming exam(s)			.676	

I sit down and revise	.687			
I am making appropriate progress on my revision	.693			
I avoid revision			.696	
I plan out my exam revision in my head				-.737
I worry about all the things that might go wrong in the exam(s)		.859		
I get caught up in my worries about revision		.900		

To form conceptually coherent subscales each with a sufficient number of items, factors one and three were combined to create a behavioural avoidance subscale, and factors two and four were combined to form a cognitive avoidance subscale. In the new cognitive and behavioural factors, the behavioural and cognitive approach items were reverse coded. The Cronbach's alpha for the behavioural scale at time 1 was .884 and the alpha for the cognitive scale at time 1 was .784. At time 2 both the behavioural (α = .718) and cognitive (α = .810) avoidance subscales had acceptable internal consistency. This was also the case at T3 behavioural avoidance (α = .741) and cognitive avoidance (α = .717).

The validity of the EAQ was assessed by examining the inter-correlations between the extracted cognitive and behavioural avoidance factors on the EAQ and the cognitive and behavioural non-social avoidance subscales on the CBAS (Ottenbreit & Dobson, 2004). In the current study the CBAS had good to excellent reliability at time 1 (BN α = .820; CN α = .889), time 2 (BN α = .708; CN α = .892) and time 3 (BN α = .914; CN α = .914). Table 5.2 shows the Pearson's correlations

between the CBAS and the EAQ cognitive and behavioural avoidance subscales over time. There are significant but moderate positive correlations between the CBAS and EAQ cognitive subscales and the behavioural subscales, suggesting they are measuring different things. Examining the items of the subscales suggests the CBAS cognitive and behavioural avoidance scales enquire more about broader, trait like avoidance, such as ‘When uncertain about my future, I fail to sit down and think about what I really want’ (CN avoidance) and ‘I quit activities that challenge me too much’ (BN avoidance). Whereas, the EAQ focuses on state cognitive and behavioural avoidance specific to the examination period, for example during the revision period, ‘I get caught up in my worries about revision’ (cognitive avoidance), and ‘I will find other jobs to do rather than revising for the upcoming exam(s)’ (behavioural avoidance). The EAQ cognitive and behavioural avoidance scales were used in part two of this study.

Table 5. 2

Correlations between CBAS and EAQ over the Exam Period

	Time 1 (Preparation Period)				Time 2 (Anticipatory Period)				Time 3 (Recovery Period)			
	BN	CN	EAQ- C	EAQ- B	BN	CN	EAQ- C	EAQ- B	BN	CN	EAQ- C	EAQ- B
T1 CBAS BN	1	.717***	.573***	.476***	.773***	.677***	.455***	.439***	.780***	.666***	.479***	.509***
T1 CBAS CN		1	.602***	.621***	.571***	.798***	.309**	.474***	.620***	.738***	.330**	.393***
T1 EAQ- C			1	.622***	.415***	.542***	.566***	.551***	.516***	.525	.442***	.386***
T1 EAQ- B				1	.445***	.638***	.274*	.354**	.434***	.538***	.207	.277*
T2 CBAS BN					1	.725***	.430***	.423***	.769***	.684***	.372**	.484***
T2 CBAS CN						1	.395***	.597***	.660***	.833***	.393***	.514***
T2 EAQ- C							1	.673***	.499	.459***	.458***	.439***
T2 EAQ- B								1	.540***	.616***	.452***	.726***
T3 CBAS BN									1	.784***	.478***	.620***
T3 CBAS CN										1	.447***	.613***

T3 EAQ- C	1	.633***
T3 EAQ- B		1

Note. CBAS = Cognitive and Behavioural Avoidance Scale; EAQ = Exam Avoidance Questionnaire; BN = Behavioural Nonsocial subscale of the CBAS; CN = Cognitive Nonsocial subscale of the CBAS; EAQ- C = Exam Avoidance Questionnaire Cognitive Avoidance; EAQ- B = Exam Avoidance Questionnaire Behavioural Avoidance.

*p<.05, **p <.010, ***p <.001

5.6 Part Two- Examination of the Reciprocal Relationship between Avoidance and Depression across Time

The second part of the study examined the reciprocal relationship between cognitive and behavioural avoidance measured on the EAQ and depression across the examination period. The participant sample was the same that was used in the development of the EAQ.

5.6.1 Measures

5.6.1.1 Depression Anxiety Stress Scale 21 (DASS; Lovibond & Lovibond, 1995). The DASS-21 is a 21-item is a reliable and valid self-report scale assessing depression, anxiety and stress symptoms over the past week. The current study used only the depression subscale. The subscale contains seven items scored on a four-point (0, did not apply to me; 3, applied to me very much or most of the time) Likert scale. In the current study, the DASS-21 depression subscale had high levels of internal consistency at each time point (T1 α = .910; T2 α = .892; T3 α = .920).

5.6.1.2 Exam Avoidance Questionnaire (EAQ). Avoidance was measured using the EAQ which has cognitive and behavioural avoidance subscales. Further details of the development of the EAQ are in part one (section 5.5).

5.6.1.3 Examination Expectations. Participants were asked at time 1 (preparation period) what grade they expected to receive during their examinations. At time 3 (recovery period) they were asked what exam grades they received, and a dichotomous ‘expectations’ variable was created by taking the mean of their exam results and seeing whether it met or did not met their expectations (0,1).

5.6.2 Design and Procedure

A within-subjects repeated measures design was used over three time-points. Eligible participants completed the online questionnaire programmed using Qualtrics (an online survey package) in the preparation period (before examinations; T1), the anticipatory (after exams but before receiving results; T2), and the recovery period (after receiving exam results; T3).

All participants gave informed consent to take part in the study and were screened for eligibility by completing a brief online survey in December to indicate whether they had January exams in universities across the UK. All eligible participants who indicated they had January exams were sent the first questionnaire via Prolific in December which closed before the exam week began. The questionnaire contained questions about demographic information, what they expected to achieve in their examinations, the DASS-21 depression subscale questions and the EAQ. Within each questionnaire there were attention checks (e.g. Please respond by clicking 'Rarely') which all participants were reminded about at the beginning of each questionnaire. Individuals who failed the attention checks were then excluded from the study to ensure participants were answering the questions as honestly and to the best of their ability. Participants who completed the first questionnaire received the second questionnaire via Prolific after the winter exam period was completed. Participants who completed both the first and second questionnaires had the final questionnaire released to them on Prolific after they received their exam results. For their time, all participants were remunerated £1 for completing the first questionnaire and £2 upon completion of the second and third questionnaire. As an incentive to complete the questionnaire as soon after the second and third questionnaires were released, participants were given a £0.50

bonus payment if they completed the questionnaire within five days of the questionnaire being released. Participants who withdrew were still remunerated for that questionnaire, but were not remunerated for any subsequent questionnaires, and were automatically redirected to the debrief page (see Appendix 3). The debrief page was displayed at the end of each questionnaire for all participants.

5.6.3 Data Analysis

All statistical analysis were conducted using SPSS and AMOS (version 26) (IBM SPSS, 2017). Descriptive analyses were conducted to characterise the sample

Spearman's correlations were conducted to examine the associations between cognitive and behavioural avoidance on the EAQ and the DASS-21 depression subscale over the three time points. To assess whether DASS-21 depression and the extracted avoidance factors on the EAQ changed over time, separate repeated measures analyses of variance (RANOVA) were conducted. Where the assumption of Sphericity was violated a Huynh-Feldt correction was applied to the degrees of freedom.

Next two, three-wave cross-lagged panel models were conducted to examine the association of the extracted avoidance factors from the EAQ with DASS-21 over the three time points. For variables that are repeatedly measured over time cross-lagged panel models examine the directional effects of variables over time (Cole & Maxwell, 2003). The hypothesised cross-lagged model is show in Figure 5.2. We followed the model testing procedure proposed by Hakanen et al. (2008) for each cross-lagged model. Firstly a stability model containing only the autoregressive effects (no cross-lagged effects) was conducted to assess the stability of the variables over time (M1, Figure 5.2). Next, a normal causation model (M2, Figure

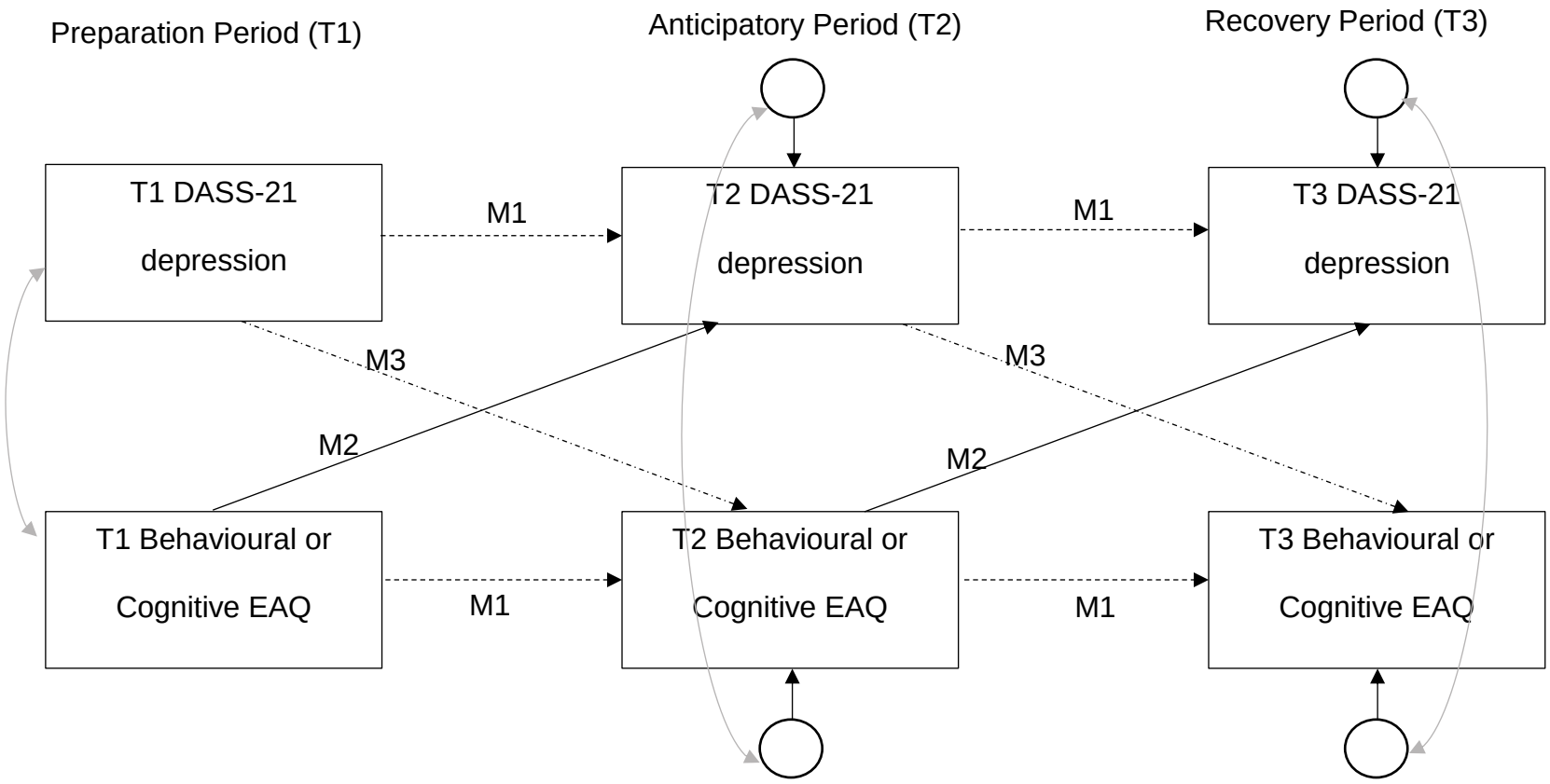
5.2) was run which contained the autoregressive effects from M1, plus the cross lagged effects from the EAQ subscale (either cognitive or behavioural avoidance) to the DASS-21 depression subscale. A reverse causation model (M3, Figure 5.2) was conducted with the autoregressive paths from M1 plus cross-lagged effects from DASS-21 depression subscale to EAQ. Lastly a reciprocal model with all effects (autoregressive and cross-lagged) was conducted. In each model the two exogenous (variables that are not influenced by another variable) variables at T1 were correlated, as well as the error terms at T2 and T3 (Anderson & Williams, 1992). The strength of the relationships are determined through comparisons of standardised regression coefficients which are reported for each path within the model. For each model 10,000 bootstrapped samples were conducted and missing data was handled using full information maximum likelihood estimation. Based on recommended guidelines (Iacobucci, 2010) the model fit was determined by examining the Chi-square test, the root mean square error of approximation (RMSEA), the standardised root mean square residual (SRMR), and the comparative fit index (CFI). The Chi Square test assess overall fit, with a non-significant *p* value indicating there is good model fit. Related to the Chi Square test is the RMSEA which adjusts for sample size. As a rule of thumb RMSEA values of .01, .05 and .08 indicate excellent, good, and mediocre fit respectively (MacCallum et al., 1996). The SRMR is the standardised difference between the observed and predicted correlation where values between 0 and .08 indicate good fit (Hu & Bentler, 1999). The CFI is a comparative fit index and is used to compare the fit between models with values (range 0.00 to 1.0) equal to or greater than .90 indicating good model fit. The Akaike Information Criterion (AIC; Akaike, 1973) was also used to examine

model fit between models, where smaller values indicate better fit (Burnham & Anderson, 2002).

To examine whether cognitive and behavioural EAQ in the preparation period (T1) was related to exam grade expectations (T3), two logistic regression models were conducted. The dependent variable was exam grade expectations (met/non met) and the independent variable was either T1 cognitive or T1 behavioural avoidance. Lastly to assess whether exam grade expectations (T3) were associated with DASS-21 depression in the recovery period (T3) an analysis of variance (ANOVA) was conducted with exam grade expectations (met/not met) as the independent variable and DASS-21 depression subscale at the dependent variable.

Figure 5. 2

The Hypothesised Cross-Lagged Model



Note. Rectangles represent variables measured at the respective time point. Double headed arrows represent correlations. Single-headed arrows represent paths. Horizontal arrows show the autoregressive paths. Diagonal arrows represent cross-lagged paths.

DASS-21 = Depression anxiety and stress scale; EAQ = exam avoidance questionnaire.

M1 represents the stability model with only autoregressive effects

M2 is the normal causation model with the cross-lagged effects from avoidance to depression

M3 is the reversed causation model with the cross-lagged effects from depression to avoidance

M4 is the reciprocal model with all autoregressive and cross-lagged effects

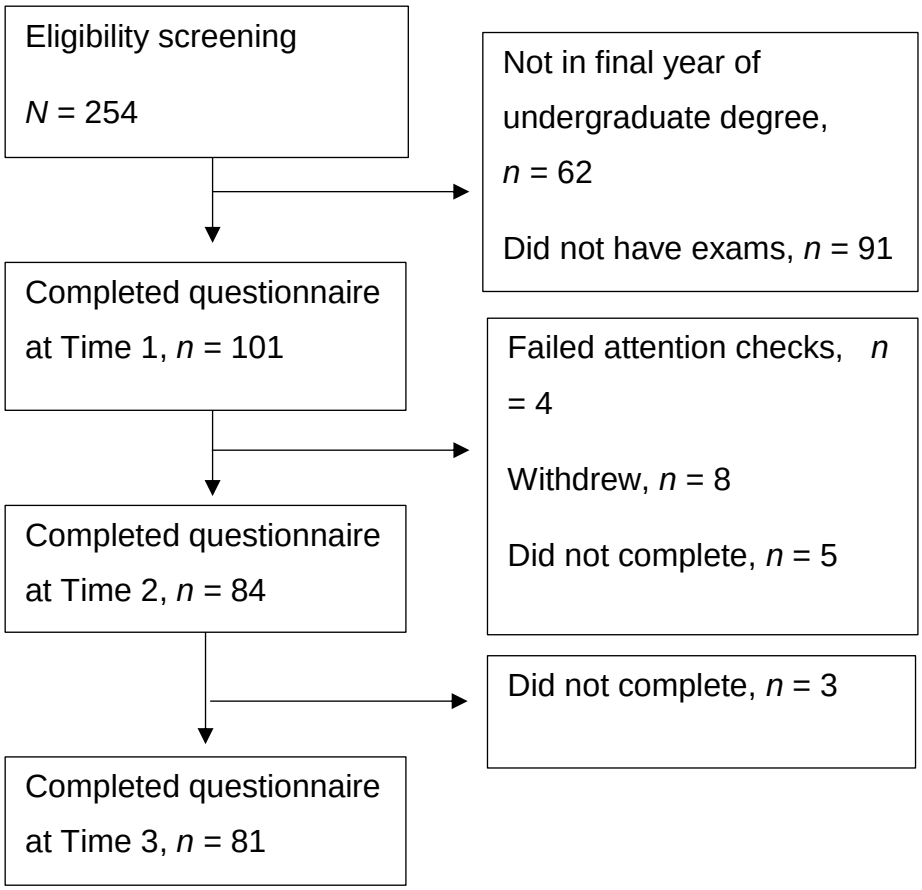
5.7 Results

5.7.1 Participants

A total of 81 participants were recruited for this study. Figure 5.3 shows recruitment; there was an 80.2% retention rate from individuals who completed the first questionnaire to the final questionnaire. The majority of participants in the final sample were female (*n* = 67), aged between 19-51 years old (*M* = 22.34, *SD* = 5.59), with the majority (*n* = 69) being under 25 years of age and reporting a family income of above £30,000 (*n* = 34; 42%).

Figure 5. 3

Flow Chart of Recruitment



Across the time points the DASS-21 depression scores, and EAQ cognitive and behavioural avoidance were relatively stable over the time period (Table 5.4). With regard to examination expectations, 31 (38.3%) did not meet their exam expectation and 50 (61.7%) met their expected examination grades.

Table 5. 3

Means (Standard Deviations) for DASS-21 Depression, Cognitive and Behavioural Avoidance across the Examination Period

	Preparation Period (T1)	Anticipatory Period (T2)	Recovery Period (T3)
DASS- 21 Depression	14.56 (12.06)	14.15 (10.10)	11.90 (11.05)
Cognitive Avoidance	22.30 (5.74)	17.05 (4.48)	17.63 (4.86)
Behavioural Avoidance	26.27 (7.89)	25.07 (6.79)	19.83 (6.17)

5.7.2 Correlations between Variables

The correlations between DASS-21 depression, cognitive and behavioural avoidance across time can be found in Table 5.5. At each time point there were small to moderate significant concurrent positive correlations between depression and cognitive and behavioural avoidance. Depression, cognitive and behavioural avoidance positively and significantly predicted prospective depression, cognitive and behavioural avoidance (respectively) at each time point.

Table 5. 4

Spearman's Correlation of DASS-21 Depression, Cognitive and Behavioural Avoidance over Time (N = 81)

	1.	2.	3.	4.	5.	6.	7.	8.	9.
1. T1 DASS-21 depression	1	.353**	.537***	.577***	.394***	.523***	.586***	.414***	.357**
2. T1 EAQ-B		1	.605***	.344**	.362**	.256*	.248*	.314**	.224*
3. T1 EAQ-C			1	.492***	.502***	.535***	.347**	.327**	.452***
4. T2 DASS-21 depression				1	.620***	.500**	.662***	.628***	.397***
5. T2 EAQ-B					1	.682***	.591***	.679***	.399***
6. T2 EAQ-C						1	.510***	.439***	.488***
7. T3 DASS-21 depression							1	.699***	.480***
8. T3 EAQ-B								1	.632***
9. T3 EAQ-C									1

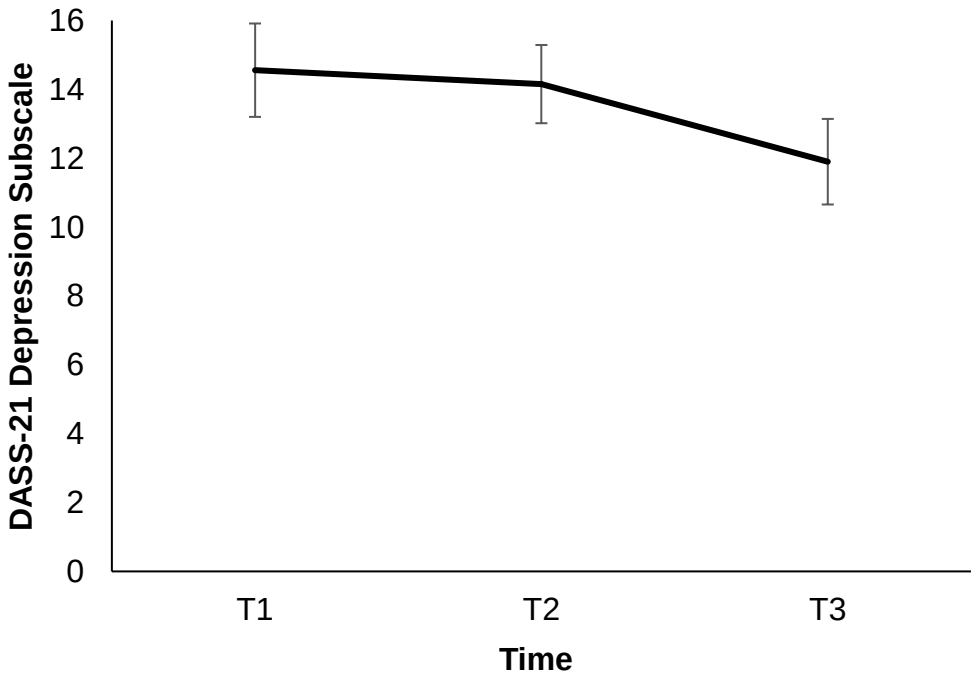
Note. T1 = Time 1, the preparation period; T2 = Time 2, the anticipatory period; T3 = Time 3, the recovery period; DASS-21 = Depression Anxiety Stress Scale; EAQ-B = Exam Avoidance Questionnaire Behavioural subscale; EAQ-C = Exam Avoidance Questionnaire Cognitive subscale. * p <.05, ** p <.010, *** p <.001

5.7.3 How Does Depression and Avoidance Change Over Time?

A RANOVA showed that DASS-21 depression significantly changed over time, $F(2, 160) = 3.701$, $p = .027$, $\eta_p^2 = .045$. There was a significant linear pattern of change $F(1, 80) = 5.780$, $p = .019$, $\eta_p^2 = .069$ (see Figure 5.4). Pairwise comparisons showed there were significant decreases between T1 and T3 (mean difference = 2.658, $p = .019$), and between T2 and T3 (mean difference = 2.253, $p = .014$), but not between T1 and T2 (mean difference = .405, $p = .724$).

Figure 5. 4

Linear Pattern of DASS-21 Depression Change over the Exam Period



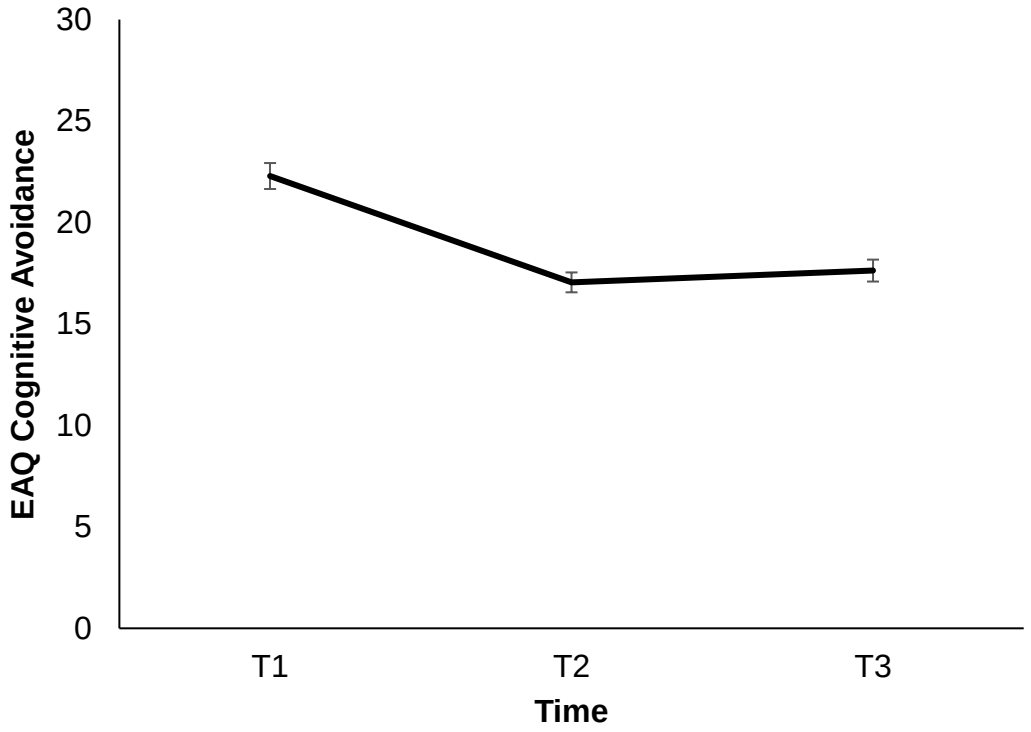
Note. DASS-21= depression and anxiety scale.

Examining change across the exam period the RANOVA showed there was a significant change in cognitive avoidance on the EAQ ($F(2, 160) = 51.334$, $p < .001$, $\eta_p^2 = .391$), which was a significant quadratic change, $F(1, 80) = 45.111$, $p < .001$, $\eta_p^2 = .361$ (Figure 5.5). Pairwise comparisons indicated that there was a significant

difference in levels of cognitive avoidance between T1 and T2 (mean difference = 5.25, p <.001) and between T1 and T3 (mean difference = 4.667, p <.001), but not between T2 and T3 (mean difference, -.580, p = .275), such that cognitive avoidance decreased from T1 to T2 and then levelled out.

Figure 5. 5

Quadratic Pattern of EAQ Cognitive Avoidance over the Exam Period



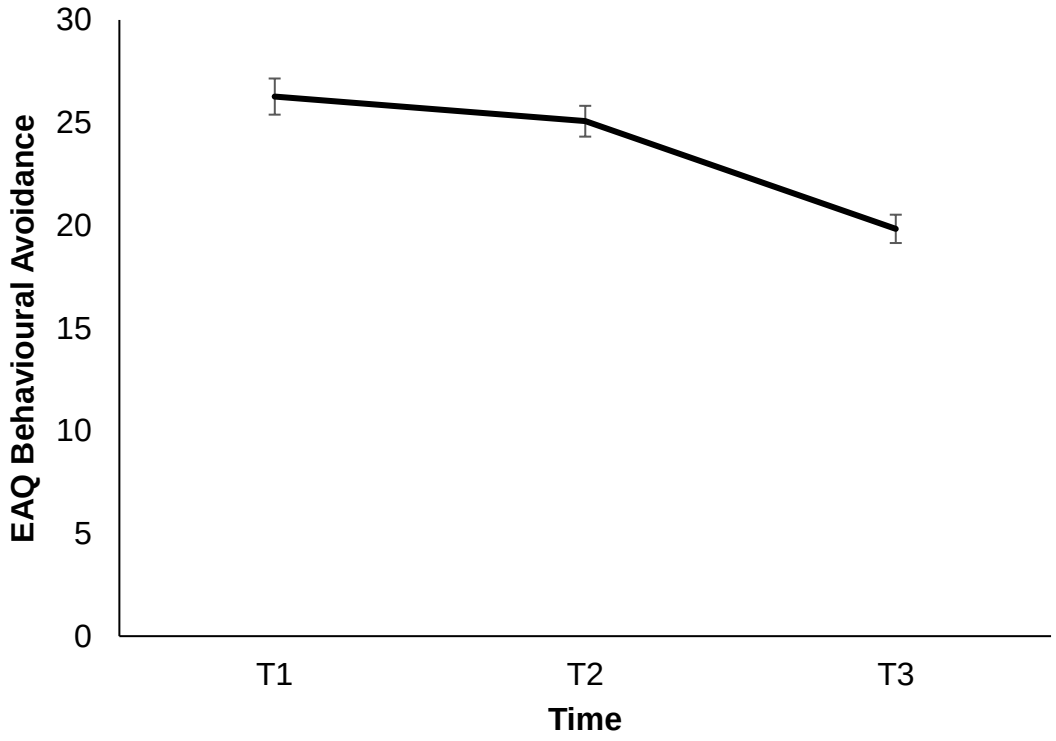
Note. EAQ= exam avoidance questionnaire.

Similarly, the RANOVA indicated that EAQ behavioural avoidance significantly changed over the exam period, $F(1, 160) = 34.162$, $p < .001$, $\eta_p^2 = .299$, and that there was a significant quadratic pattern of change, $F(1,80) = 11.649$, $p = .001$, $\eta_p^2 = .127$ (Figure 5.6). Pairwise comparisons showed there was no significant difference in levels of behavioural avoidance between T1 and T2 (mean difference = 1.198, p = .203). However, there was significant differences in levels of behavioural avoidance

between T1 and T3 (mean difference = 6.44, p <.001), and between T2 and T3 (mean difference = 5.247, p <.001), such that behavioural avoidance remained constant across T1 and T2, then decreased by T3.

Figure 5. 6

Quadratic Pattern of EAQ Behavioural Avoidance over the Exam Period



Note. EAQ= exam avoidance questionnaire.

5.7.4 Is Behavioural and Cognitive Avoidance Prospectively Associated with Depression, and Vice Versa, Over the Exam Period?

5.7.4.1. Behavioural Avoidance and Depression.

A stability and cross-lagged effect model was conducted for behavioural avoidance and DASS-21 depression over the examination period. The fit statistics for the tested four models (M1a-M4a) are reported in Table 5.6. The stability model, which tests the temporal stability of the factors over time with no cross-lagged paths,

indicated the poorest fit of all models and the fully cross-lagged reciprocal model (M4a) indicated the best fit and therefore was the reported model. In all models the RMSEA value was higher than the recommended cut off (.08). Kenny, Kaniskan, and McCoach (2014) note that in models with low degrees of freedom can lead RMSEA to falsely indicate poor fit. For the chosen model (M4a) because the other fit indices (CFI, SRMR, and lowest AIC) were good, suggesting this model was acceptable to use. The exact *p* values for the cross-lagged model is in Appendix 4.

This model (Figure 5.7a) demonstrated that, as expected, depression prospectively predicted depression at each time point, and behavioural avoidance prospectively predicted behavioural avoidance. Regarding the cross-lagged relationships, depression at T1 (preparation period) was associated prospectively with greater behavioural avoidance at T2 (anticipatory period), and behavioural avoidance at T2 was associated with greater negative depression at T3 (recovery period). Additionally greater depression at T2 was associated with greater levels of behavioural avoidance at T3. There were no other significant relationships in the model.

5.7.4.1. Cognitive Avoidance and Depression.

Similar to the behavioural avoidance and depression model, the stability model fit statistics for the cognitive avoidance and depression indicated the poorest fit (M1b) and the reciprocal model (M4b) was the best fitting model (Table 5.6). As with the behavioural avoidance and depression reciprocal model, the RMSEA was higher than the recommended cut off, but the other fit indices were acceptable. The exact *p* values for the cross lagged model is in Appendix 5.

This model (Figure 5.7b) showed depression prospectively predicted greater depression, and cognitive avoidance prospectively predicted cognitive avoidance at subsequent time points. Regarding the cross-lagged relationships, cognitive avoidance at T1 (preparation period) predicted greater depression at T2 (anticipatory period), and in turn, depression at T2 predicted greater cognitive avoidance at T3 (recovery stage). Depression levels at T1 also predicted cognitive avoidance at T2, and cognitive avoidance at T2 predicted depression at T3.

Table 5. 5

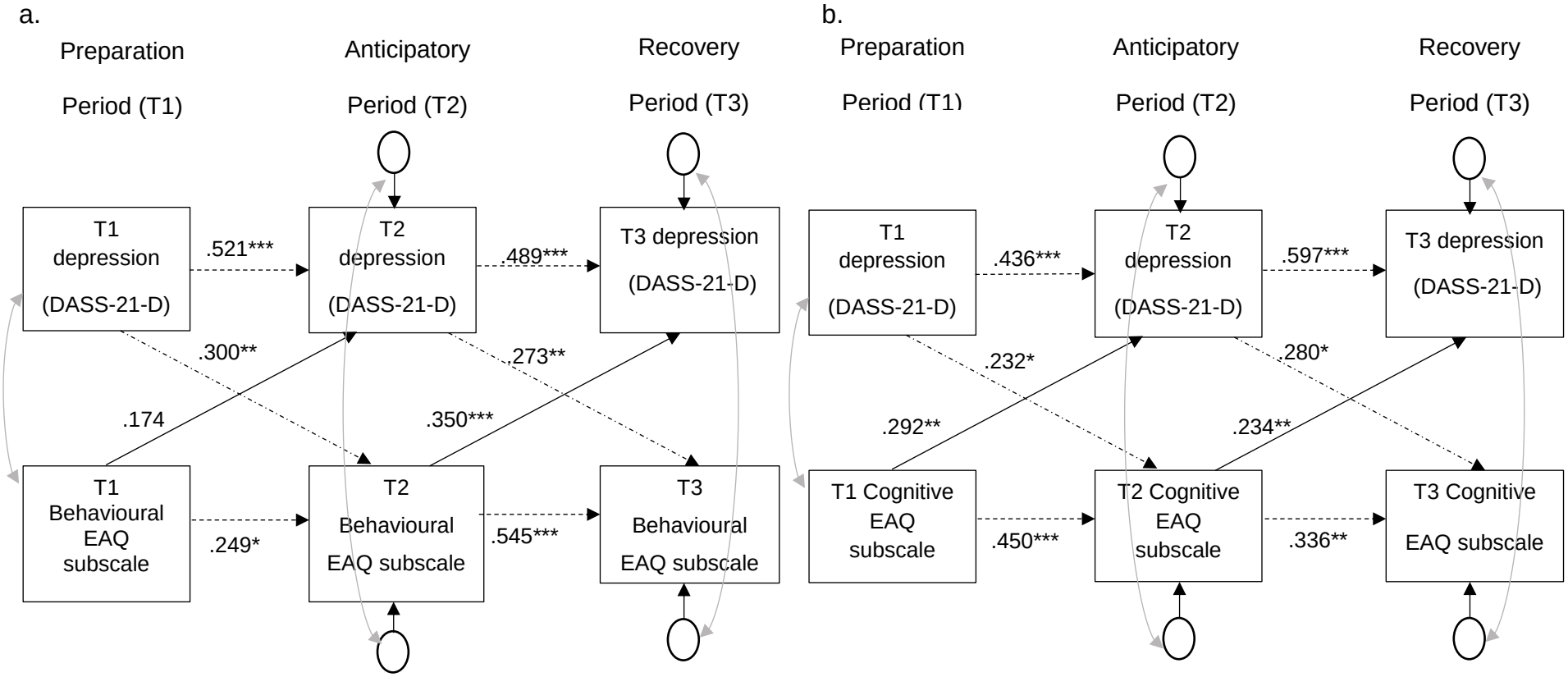
Summary Fit Statistics for the Cross Lagged Models using the DASS-21 Depression Subscale

Model	χ^2	*df*	*p*	RMSEA	SRMR	CFI	AIC
Behavioural EAQ and DASS-21							
$M1a_{stability}$	51.741	8	<.001***	.261	.1843	.822	89.741
$M2a_{normal\ causation}$	28.036	6	<.001***	.214	.1318	.910	70.036
$M3a_{reversed\ causation}$	28.665	6	<.001***	.217	.0815	.908	70.665
$M4a_{reciprocal\ model}$	13.097	4	.011*	.169	.0414	.963	59. 097
Cognitive EAQ and DASS-21							
$M1b_{stability}$	40.111	8	<.001***	.224	.1751	.839	78.111
$M2b_{normal\ causation}$	21.658	6	.001**	.181	.1005	.921	63.658
$M3b_{reversed\ causation}$	25.632	6	<.001***	.202	.1074	.901	67.632
$M4b_{reciprocal\ model}$	10.636	4	.031*	.140	.0407	.967	56.636

Note. RMSEA = root mean square error of approximation; SRMR = standardised root mean square residual; CFI = comparative fit index; AIC = Akaike information criterion; EAQ = exam avoidance questionnaire; DASS-21 = Depression anxiety and stress scale.* *p* <.05, ** *p* <.010, *** *p* <.001

Figure 5. 7

Cross-Lagged Analysis Models between Avoidance and Depression



Note. DASS-21 = Depression Anxiety and Stress Scale; Standardised regression weights reported. *p <.05, ** p <.010, *** p <.001.

a. Cross-lagged model of behavioural avoidance and depression (DASS-21). b. Cross-lagged model of cognitive avoidance and depression (DASS-21).

5.7.5 Is Behavioural and Cognitive Avoidance During the Preparation Period (T1) Associated with Meeting Exam Outcomes in the Recovery Period (T3)?

Contrary to expectations, behavioural avoidance during the preparation period (T1) was not associated with exam outcomes at T3 (recovery period) (OR = .986, 95% CI = .915, 1.062). Similarly, cognitive avoidance at T1 was not associated with meeting exam expectations at T3 (OR = .920, 95% CI = .827, 1.023).

5.7.6 Are Exam Outcomes (T3) Associated with DASS-21 Depression in the Recovery Period (T3)?

Consistent with our hypothesis, the results of the ANOVA showed that individuals who did not meet (*M* = 15.226, *SE* = 1.77) their expected exam results at T3 (recovery period) had significantly greater levels of DASS-21 depression than those who did meet their expected exam results (*M* = 10.24, *SE* = 1.40), $F(1, 79) = 4.891$, $p = .030$, $\eta_p^2 = .058$.

5.8 Discussion

This aim of this study was to examine the reciprocal relationships between cognitive and behavioural avoidance and depression across a stressful life event where there was reasonable probability of there being variability in depressive symptoms outside of treatment; university examination periods. In the first part of the study we developed a questionnaire to specifically measure exam related avoidance. In the second part of this study we found, as expected, some variability in cognitive and behavioural avoidance and depressive symptoms over the examination period. We found there were reciprocal relationships between behavioural and cognitive avoidance and depression across the examination period, with the exception that T1 (preparation period) behavioural avoidance was not associated prospectively with T2

(anticipatory period) depressive symptoms. Contrary to predictions, we did not find that cognitive and behavioural avoidance at T1 were associated with meeting exam expectations at T3 (recovery period), but in the recovery period individuals who did not meet their expected exam results had higher levels of depression.

The development of the EAQ in this study allowed us to examine avoidance specific to the examination period, rather than using a broader measure of avoidance like the CBAS. A particular strength of the EAQ measure is that the items are adapted to each stage of the examination period. The EAQ showed good internal consistency across the time points and there was convergence between the CBAS CN (cognitive non-social) and BN (behavioural non-social) subscales and the EAQ cognitive and behavioural avoidance subscales, suggesting that the EAQ was measuring avoidance. Nevertheless, there were also positive correlations between the CBAS CN subscale and the EAQ behavioural avoidance subscale, and the CBAS BN and the EAQ cognitive avoidance scale suggesting perhaps there may not have been good discriminant validity between the subscales. We assessed this measure in a relatively small sample size and further use of the EAQ in other samples is needed to establish its validity and reliability in assessing cognitive and behavioural avoidance.

In the second part of the study, we found some variability in depressive, cognitive and behavioural avoidance symptoms across the examination period which gives us some insight into how mood and coping unfolds across stressful periods. During the preparation period (T1) and anticipatory period (T2) depression symptoms and behavioural avoidance remained stable and then reduced in the recovery period (T3), whereas cognitive avoidance peaked during the preparation period, after which it remained constant. The depression symptom variation over this

period is partly in line with the wider discontinuities literature which finds fluctuations of depression symptoms. However these studies focus on depression symptom discontinuities within treatment (e.g. Hayes, Laurenceau, et al., 2007; Tang & DeRubeis, 1999; Tang et al., 2005) or outside treatment (e.g. Kelly, Roberts, et al., 2007; Shalom et al., 2018; Shalom et al., 2020) in clinical samples. It is interesting that in this non-clinical sample we found some variability in depressive symptoms and this highlights the utility of using stressful periods as times in which depressive symptoms do fluctuate to examine emotion regulation strategies outside of treatment. Although previous research has examined exam periods at a time of peak stress, little research has also examined how individuals recover from these periods, which is a critical part of the emotion regulation process (Gross, 2014) and a period that the findings from this thesis (chapter three) suggest may also be important to understand in therapy. It is also possible that individuals who do not naturally recover after a stressful period, like those who did not meet their expected examination grades and were exhibiting greater depressive levels than those who did meet their expected grades during the recovery period, may be more vulnerable to poorer mood in the future. In the discontinuities literature, individuals who experience 'sudden losses', which are characterised by worsening in mood during treatment that does not recover, have significantly worse depression outcomes at the end of treatment, than those who did not experience any shifts in mood, or sudden gain which are rapid improvements in mood (Lutz et al., 2013). Little research examines the longer-term effects of depression fluctuations outside of treatment in non-clinical samples. Future research may wish to investigate this to understand whether these individuals who do not recover may be more vulnerable to poorer mood in the long term. Furthermore, our study results suggest that is important to examine the

evolvement of processes over and at the end of times of mood variability. In the wider depression discontinuities literature in therapy settings there is a focus on examining processes that precipitate change (e.g. Abel et al., 2016; Bohn et al., 2013; Kelly, Cyranowski, et al., 2007; Lemmens et al., 2021) but less research examines process changes during and following discontinuous change (Wucherpfennig, Rubel, Hofmann, et al., 2017). Investigating this may help us to understand more about the factors that contribute to immediate and longer term depression symptom changes.

In line with expectations, we found reciprocal relationships between cognitive and behavioural avoidance and depression across all time points, except for behavioural avoidance during the preparation period and the association with depression symptoms in the anticipatory period. Other studies using student samples have found cross sectional (Moulds et al., 2007; Ottenbreit & Dobson, 2004; Wagener et al., 2016) and longitudinal (Grant et al., 2013) associations between behavioural avoidance and depression. Therefore this result is surprising, especially given that during the preparation period the focus was on revising for examinations. This may suggest the EAQ avoidance measure was not measuring behavioural avoidance as we expected and further validation of the EAQ is needed.

We note that we did not look at the interaction between cognitive and behavioural avoidance throughout the time period because of the limited sample size. Further research is needed to understand how cognitive and behavioural avoidance interact with each other and are associated with prospective depression symptoms. Nevertheless, our findings are consistent with theories of depression which highlight the importance of avoidance processes influencing depression but also as a maintenance factor (Martell et al., 2001; Trew, 2011). We recognise that

we have examined avoidance processes and depression symptoms over one example of a life stressor and this may not generalise to other stressors or within therapy contexts. However, the findings of the current study may suggest that in student samples during periods of stress, like examination periods, teaching strategies to help with cognitive and behavioural avoidance, may be helpful.

A limitation of the current study is that we were unable to explore whether gender differences moderated associations between cognitive or behavioural avoidance and depression symptoms because of our limited sample size. Within the depression literature, some studies find women are more likely to ruminate (a type of cognitive avoidance) when experiencing low mood or depression (Butler & Nolen-Hoeksema, 1994; Nolen-Hoeksema et al., 1999; Nolen-Hoeksema et al., 1993) which may make women more vulnerable to depressive symptoms. These gender differences have also been observed in non-clinical university populations (Blalock & Joiner, 2000; Moulds et al., 2007), where women reported more cognitive avoidance and worry compared to men (Robichaud et al., 2003). Some research also finds behavioural avoidance is correlated with depression in males (Moulds et al., 2007). Future research examining this can elucidate individual differences in coping styles and whether they may differentially influence depression variability outside of treatment.

In conclusion, we demonstrated that periods of life stress, like examination periods, are times in which there is variability in depression symptoms outside of therapy in which to explore depressionogenic processes like avoidance. We found cognitive and behavioural avoidance and depression have a reciprocal relationship over this period, and this highlights the importance of looking at processes over time.

Further research is needed to examine the longer terms effects of this depression variability during stress outside treatment in non-clinical samples.

Chapter five Appendices

Appendix 1

Recruitment Poster and Handout

Are you a final year undergraduate student AND have exams in January 2020?

Our study is looking at how we cope with stress over exams

Complete 3 short questionnaires on the online platform Prolific and you will be paid a total of

£5

To take part, sign up for a Prolific account by going to prolific.ac or scan the QR code with your phone camera

and send your ID to al395@exeter.ac.uk

1. Sign up for a Prolific account here: prolific.ac
2. Send your ID to al395@exeter.ac.uk

Study looking at how we cope with stress over exams

Complete 3 short questionnaires and earn

£5

Final year undergrad and have January exams? Sign up

Sign up for a Prolific account at prolific.ac or scan the QR code with your phone camera

Appendix 2

Exam Avoidance Questionnaire

Exam avoidance questionnaire – Preparation period (T1)

Please respond to the items below thinking about how you are currently during the revision period leading up towards your January exam(s)

Cognitive items (5 avoidance, 5 approach counterbalanced)

1. I try not to think about revising for the exam(s)
2. I get caught up in my worries about revision
3. If I don't think about exam(s) revision then I feel better
4. I try to ignore my thoughts about the exam(s) because I think about failing
5. I worry about all the things that might go wrong in the exam(s)
6. I think constructively about revising for the exam(s)
7. I can think about revising without getting caught up and worrying about it
8. I plan out my exam revision in my head
9. I imagine being successful in my revision and exam(s)
10. I think about the positives rather than worry about revision and the exam(s)

Behavioural items (5 avoidance, 5 approach counterbalanced)

1. I avoid revision
2. I find myself watching TV or surfing the internet rather than revising for the exam(s)
3. I will find other jobs to do rather than revising for the upcoming exam(s)
4. I talk to other people about my revision and exam(s) instead of getting on and revising
5. I put off revising until it's too late
6. I sit down and revise
7. I break down revision tasks and do them one by one
8. I can get back into the flow of revision after I have taken a break
9. I am sticking more or less to my plan for revising
10. I am making appropriate progress on my revision

Exam avoidance questionnaire – Anticipatory period (T2)

Cognitive items (5 avoidance, 5 approach counterbalanced)

1. I go over and over again about things I could have done in the exam
2. I get caught up in my worries about the exam(s) results
3. If I don't think about the results then I feel better
4. I try to ignore my thoughts about the exam(s) because I think about failing
5. I worry about my exam results
6. I think positively about the exam results
7. I can think about the results without worrying about it
8. I focus on the good things I did in the exam(s)
9. I think constructively about what I would do if my results were not good
10. I think about the positives rather than worry about the exam(s) and the results

Behavioural items (5 avoidance, 5 approach counterbalanced)

11. I avoid any reminders of the exam(s)
12. I have been drinking or using substances more to avoid thinking about the exam results
13. I find myself being unmotivated to do anything until I get my results
14. I don't talk to other people about my exam(s) or the upcoming results
15. I will put off looking at my exam(s) results when I receive them
16. I keep myself busy in anticipation of the exam(s) results
17. Now that the exams are over I am getting on with things
18. I don't let my upcoming exam results stop me from doing things I enjoy
19. I plan what I will do whichever way my results go
20. I can focus more on other pieces of work in my degree now

Exam avoidance questionnaire – Recovery period (T3)

Cognitive items (5 avoidance, 5 approach counterbalanced)

1. I try not to think about my exam(s) results
2. I keep worrying about what my exam results mean for what I will be able to achieve next time round
3. I find it hard to give myself credit for the fact that I have got through these exams
4. I keep thinking over and over about the exam results
5. Now that I have my results, I worry about all the things that might go wrong in my degree
6. I think constructively about my exam(s) results
7. I can think about my results without getting caught up and worrying about them
8. There is nothing I can do about the results now, so I don't worry about them
9. I imagine being successful in my degree
10. I think about the positives rather than worry about my exam(s) results

Behavioural items (5 avoidance, 5 approach counterbalanced)

11. I have been drinking or using substances more to avoid thinking about the implications of the exams results
12. Even though the exams are over I just can't get on with things
13. I make up excuses and turn down opportunities to socialise and celebrate exam results
14. If friends or family ask me about my results I change the topic
15. I can't stop asking for reassurance from my friends about what my exam results mean for my future
16. I don't let my exam results stop me doing things I enjoy
17. Now that the exams are over I am getting on with things
18. I am happy to attend social gatherings to celebrate exam results
19. If feedback for the exam(s) were available, I would access it
20. I can focus more on other pieces of work in my degree now

Appendix 3

Debrief Form

Thank you for your participation in our research. Your time has been greatly appreciated.

This study is aiming to identify and examine the processes and strategies used at different stages of a stressful life event, your January exams. The online questionnaires asked you about your mood, anxiety, avoidance, hope, rumination, positive and negative affect, and coping strategies. We aim to find out whether these are related to how people manage stressful life events. We hope that by understanding this further we can better help people deal with stressful life events in the future.

We will hold the anonymised data for up to 10 years from the point of publication or last external request to see data. When the report is written we will ensure all participants will remain anonymous. If you wish to withdraw your data from this study, you can do so without penalty.

If you would like to receive a copy of the final report of this study (or a summary of the findings) when it is completed, please feel free to contact us.

Useful contact information:

If you have any further questions, please feel free to contact the research team by emailing, al395@exeter.ac.uk. For any queries you may have regarding ethics please contact Gail Seymour (Research Ethics and Governance Manager) by emailing, G.M.Seymour@exeter.ac.uk

If you feel you have been adversely affected by taking part in this study and would like to speak to an independent support service you are advised to seek help from organisations such as; Samaritans, Nightline and Mind services. Contact information for is listed below.

Samaritans: 24/7 helpline -Telephone 116 123 www.samaritans.org

Saneline: Helpline 4:30pm to 10:30 pm Tel: 08457678000

Mind: www.mind.org

Nightline: https://www.nightline.ac.uk/want-to-talk

Headspace: https://www.headspace.com

Appendix 4

Standardised Regression Coefficients for Behavioural Avoidance and DASS-21 Depression Subscale Cross-Lagged Model

Path	B(Se)	p
T1 EAQ-B → T2 EAQ-B	.249 (.09)	.020*
T1 DASS-21 → T2 DASS-21-D	.521(.08)	<.001***
T1 DASS-21 → T2 EAQ-B	.300 (.06)	.005**
T1 EAQ-B → T2 DASS-21-D	.174 (.12)	.068
T2 EAQ-B → T3 EAQ-B	.545 (.09)	<.001***
T2 DASS-21 → T3 DASS-21-D	.489(.11)	<.001***
T2 DASS-21 → T3 EAQ-B	.273 (.06)	.005**
T2 EAQ-B → T3 DASS-21-D	.350 (.16)	<.001***

Note. EAQ- B = exam avoidance questionnaire- behavioural subscale; DASS-21-D = depression anxiety and stress scale, depression subscale; T1 = time 1, preparation period; T2 = time 2, anticipatory period; T3 = time 3, recovery period.

* p <.05, ** p <.010, *** p <.001

Appendix 5

Standardised Regression Coefficients for Cognitive Avoidance and DASS-21 Depression Subscale Cross-Lagged Model

Path	B(Se)	p
T1 EAQ-C → T2 EAQ-C	.450 (.08)	<.001***
T1 DASS-21 → T2 DASS-21-D	.436 (.08)	<.001***
T1 DASS-21 → T2 EAQ-C	.232 (.04)	.024*
T1 EAQ-C → T2 DASS-21-D	.292 (.17)	.003**
T2 EAQ-C → T3 EAQ-C	.336 (.12)	.002**
T2 DASS-21 → T3 DASS-21-D	.597 (.09)	<.001***
T2 DASS-21 → T3 EAQ-C	.280 (.05)	.012*
T2 EAQ-C → T3 DASS-21-D	.234 (.22)	.007**

Note. EAQ- C = exam avoidance questionnaire- cognitive subscale; DASS-21-D = depression anxiety and stress scale, depression subscale; T1 = time 1, preparation period; T2 = time 2, anticipatory period; T3 = time 3, recovery period.

* p <.05, ** p <.010, *** p <.001

Chapter 6

6.1 General Discussion

This thesis investigated patterns of discontinuous change and whether key depressionogenic processes occur over these patterns of change and are associated with treatment outcomes. The main findings of each study are briefly outlined in Table 6.1. To minimise repetition of points outlined in each study discussion this chapter will focus primarily on the wider thesis questions and discuss the methodological, theoretical, and clinical implications of the current research, and directions for future research in this field.

Table 6. 1

Overview of Thesis Findings

	Study findings
Study 1 (chapter 2): Sudden gains and depression spikes in a large IAPT dataset	• 19% (*n* = 1836) experienced sudden gains and 24% (*n* = 2265) experienced depression spikes. Rates of both patterns of discontinuous change were highest in HiCBT. Sudden gains and depression spikes were mostly likely to occur early in treatments • In line with expectations, both sudden gains and depression spikes were associated with improved depression outcomes, regardless of treatment modality. They were also associated with improved anxiety and functioning outcomes at treatment end, regardless of treatment modality • Individuals with higher baseline clinical severity benefitted most from experiencing a sudden gain or depression spike
Study 2 (chapter 3): Processes surrounding sudden gains and treatment outcomes in an RCT sample comparing CBT and BA	• Contrary to expectations no client processes, neither cognitive (accommodation and overgeneralisation) nor behavioural (avoidance and positive behaviour), predicted sudden gains in either treatment • Partially in support of hypotheses, individuals who had a sudden gain, compared to no gain, had lower levels of avoidance in the postgain session regardless of treatment modality • In the postgain session individuals who experienced a sudden gain in BA, compared to CBT, and had greater levels of overgeneralisation, reported higher PHQ-9 scores at 18-month treatment outcome. The opposite was found in CBT; greater postgain overgeneralisation was associated with lower PHQ-9 scores at 18-month outcome

	• Other three-way interactions between sudden gain status, treatment and process were in individuals who did not experience a sudden gain
Study 3 (chapter 4): Processes surrounding depression spikes and associations with treatment outcomes in an RCT sample comparing CBT and BA	• Contrary to expectations no hypothesised client (cognitive emotional processing, cognitive flexibility, positive behaviours, avoidance, or negative life events) or therapist (therapeutic difficulty, therapist cognitive and behavioural corrective information) processes were associated with a depression spike in either CBT or BA However, in individuals who did not experience a depression spike, higher levels of behavioural corrective information was supplied by the therapist in the matched prespike session in BA compared to CBT • No hypothesised main effects, two-way, or three-way interactions between process, treatment type and depression spike status were associated with 12- or 18-month treatment outcomes
Study 4 (chapter 5): Association of cognitive and behavioural avoidance with depression during stressful life events	• Consistent with expectations there was a linear pattern of reducing depression change. Further, there were quadratic patterns of cognitive (U shape) and behavioural (inverted U shape) avoidance over the examination period • Partially consistent with hypotheses, greater behavioural avoidance at T2 (post-examinations) prospectively predicted greater depression levels at T3 (post-results) • In line with expectations, greater cognitive avoidance in T1 (pre-examination) and T2 prospectively predicted greater depression levels (T2 and T3 respectively) • Similarly, in line with hypotheses at each time point greater depression levels prospectively predicted greater cognitive and behavioural avoidance

6.1.1 Discussion of Findings in Relation to the Main Thesis Questions

6.1.1.1 What are the Rates and Timings of Sudden Gains and Depression Spikes in Every Day Clinical Practice, and What is Their Relationship with Treatment Outcomes in CBT and non-CBT Therapies?

In study one (chapter two) we observed 19% of individuals experienced a sudden gain and depression spikes occurred in 24% of individuals in everyday clinical practice settings. Individuals in high- (sudden gains 21.4%; depression spikes 27%), compared to low-intensity (sudden gains 18.3%; depression spikes 22.1%) treatments were more likely to experience sudden gains and depression spikes. To our knowledge this is the first study to examine rates of sudden gains and depression spikes across low- and high-intensity therapies in the same setting using consistent definitions of sudden gains and depression spikes, in a large sample. This is important as other sudden gains research uses slight variations in definitions of sudden gains (Shalom & Aderka, 2020) and depression spikes definitions differ from Hayes et al.'s (2007) original depression spike criteria (e.g. Keller et al., 2014; O'Mahen et al., 2021).

For sudden gains, the overall rates across treatments are in line with other studies looking at sudden gains in everyday clinical practice and research settings, but notably lower than the average (34.6%, range 14.3-62.2%; Shalom & Aderka, 2020). The rate differences between low- and high-intensity therapies are similar to findings seen in the literature where typically sudden gains occur to a lesser extent in group-based treatments (Norton et al., 2010; O'Mahen et al., 2017; Thorisdottir et al., 2018). It is of note that the rate of sudden gains in HiCBT is in the range of sudden gains in RCT samples (Shalom & Aderka, 2020). It is possible that within group-based treatments in IAPT, which are psychoeducation based, there is less personalised therapeutic support and

potentially therefore less opportunity to generate sudden gains. Our results also concur with wider literature findings that sudden gains are more likely to occur early in treatment (Shalom & Aderka, 2020). However, a common finding in the psychotherapy literature is that a large amount of change occurs in the early stages of therapy and this early response is associated with favourable treatment outcomes (e.g. Ilardi & Craighead, 1999; Lambert, 2005; Tang & DeRubeis, 1999). Consistent with this, a recent meta-analysis found a large effect (Hedges' *g* = 0.87) of early treatment response (most commonly conceptualised as changes between baseline and week four of therapy) and posttreatment depression and anxiety outcomes (Beard & Delgadillo, 2019). Therefore it is possible that the association between early sudden gains and treatment outcomes are an artefact of early treatment response.

Another consideration when investigating discontinuous patterns of depression change are that they are only a small window into a wider trajectory of depression symptom change across therapy. Other person-centred statistical approaches can be used to identify individuals who experience similar trajectories of change throughout treatment (Jung & Wickrama, 2008). Growth mixture modelling (GMM) techniques (Muthén et al., 2002) allow for the identification of meaningful, homogeneous groups within a larger heterogeneous population. If individuals who experience sudden gains or depression spikes are more salient than others, these patterns would emerge as distinctive classes. GMM has been used to examine different trajectories of symptom change in the psychotherapy literature. For instance, Stulz et al. (2007) used GMM to examine early treatment progress (up to the sixth treatment session) in routine outpatient psychotherapy. They found five distinct patterns of early symptom change, including early improvements and initial

impairments in symptoms. As expected those who experienced early improvements had the most symptom change compared to the other groups, whereas individuals who experienced initial impairments showed little change in the early stages of treatment. In a more recent example, Senger et al. (2022) observed three distinct symptom change patterns (no response, early response, slow change) in individuals receiving CBT for persistent somatic symptoms. Individuals with an early change response experienced better treatment outcomes. Using GMM techniques to identify patterns of symptom change can also allow for the comparison of different trajectories and their association with short and long term treatment outcomes. Furthermore, this may also have clinical utility for those who do not show an immediate treatment response. Senger and colleagues highlight that understanding the trajectories and treatment outcomes of individuals who make slower change throughout therapy can help clinicians to encourage individuals who become demoralised by this to continue with treatment (Senger et al., 2022). Other research finds that experiencing any discernible pattern of change, even if it is not early in treatment, is associated with good treatment outcomes (Vittengl et al., 2013). This is particularly encouraging for those who do not experience early changes like sudden gains.

With regard to depression spikes, this study is the first to explore the rates of depression spikes in both low- and high-intensity non-EBCT treatments in everyday clinical settings. Study one showed that depression spikes also occur in naturalistic settings and with similar rates to other studies which examine depression spikes in non-exposure based treatments (Abel, 2014; O'Mahen et al., 2021). Although the overall rate of depression spikes in this study was lower than in Hayes et al.'s (2007) seminal depression spikes study

(62%), we found depression spikes were more likely to occur in high- (27%) than low-intensity (22.1%) treatments. This suggests, similarly to Hayes et al.'s hypothesis, that depression spikes may occur when there is greater in-depth processing of depressive content which is more likely to occur in high- than low-intensity treatments in IAPT. However, in Hayes et al.'s study this occurred during the middle of treatment where there intended therapeutic strategies were used to generate depression spikes. In the current study, we found depression spikes were more likely to occur in the early sessions of treatment, which is contrary to other research that generally finds they occur during the middle of treatment in non-EBCT studies (session 9/18, Abel, 2014; session 5/12, O'Mahen et al., 2017; session 3 or 4/10, O'Mahen et al., 2019) and in EBCT (Hayes et al., 2007). This may be because some of those in high-intensity therapy had been "stepped up" from a course of low-intensity therapy and thus were essentially midway through treatment early on in their course of high-intensity therapy. Alternatively, it is possible depression spikes occur when more intensive therapeutic strategies are being used. Further research examining why depression spikes occur in low- and high-intensity therapies is needed.

Regarding the relationship to treatment outcomes, both sudden gains and depression spikes were associated with beneficial dimensional depression outcomes in study one, regardless of treatment modality. These results are consistent with other findings that show sudden gains in everyday clinical practice and RCT settings have beneficial depression outcomes across treatments (Shalom & Aderka, 2020). The literature examining the association between depression spikes and treatment outcomes is mixed; some find beneficial associations between depression spike and depression treatment

outcomes (Hayes et al., 2007), other research suggests they could be associated with unfavourable depression outcomes (O'Mahen et al., 2021), whereas some find depression spikes are not associated with outcome (O'Mahen et al., 2017; O'Mahen et al., 2019). Further examination of how depression spikes are related to treatment outcomes is needed across different samples. Additionally, this study extended the current literature and found both sudden gains and depression spikes are also associated with favourable anxiety and functioning outcomes across all treatments. One previous study found sudden gains were not associated anxiety and functioning outcomes and treatment end (Stiles et al., 2003), but these individuals did not have a primary diagnosis of depression. No previous studies have examined the association of depression spikes and other end of treatment outcomes. These results are particularly encouraging and suggest that when there is a focus of depression reduction in treatment this can also be beneficial to other symptoms commonly associated with depression, such as anxiety and problems with functioning. However, it is of note that using different methods of assessing treatment outcome did yield slightly different results. For instance, although individuals who experienced a sudden gain, compared to those who did not, were more likely to reliably improve than deteriorate or experience no change across outcomes (depression, anxiety and functioning), they were also less likely to have clinically significant change in anxiety and functioning outcomes. For depression spikes, individuals were more likely to improve than experience no change, but were also more likely to have deterioration of depression symptoms than improve. From these findings and those of study three, which examined client processes and therapist strategies related to depression spikes, it is unclear why individuals experience depression spikes. One possibility that we

were unable to capture in study three was whether negative life events outside of therapy may have an impact on a depression spike. It is also possible that positive life events may impact on sudden gains. It would be useful to gather further information about life events during therapy and this can be done in non-burdensome way such as asking a short question about positive and negative life events experienced between therapy sessions. Not only may this help us to further understand variability in depression symptoms like sudden gains and depression spikes, this immediate feedback may support therapists during the therapy session.

When examining moderators of the association between discontinuous change and treatment outcomes, in study one we found baseline clinical severity moderated the association between sudden gains and depression spikes and treatment outcomes. In particular individuals with greater baseline severity benefited the most from experiencing a sudden gain or depression spike in treatment. The literature examining moderators of discontinuous change is limited and in a recent meta-analysis looking at sudden gains across treatments and disorders, Shalom and Aderka (2020) did not find pre-treatment symptom severity moderated sudden gains across disorders and psychotherapies. Although in the current thesis and wider literature there is a focus on examine baseline demographic and clinical characteristic as moderators of discontinuous change, it may be more beneficial to understand whether there are client related prognostic factors at baseline that can be utilised during periods of depression variability in treatment. Other research suggests focussing on clients strengths (capitalisation models) can cultivate motivation to engage with therapy (Flückiger et al., 2009; Grawe, 1997) and this may be particularly important during times of depression variability, especially

periods of worsening. Another example is psychological flexibility, which has found to be important in processing new information and learning of new skills (Kashdan & Rottenberg, 2010). Further research examining specific individual characteristics at baseline may help personalise the strategies used during periods of discontinuous change in treatment to maximise treatment effectiveness.

It is particularly interesting that treatment modality did not moderate the association between patterns of discontinuous change and treatment outcomes in the clinic based sample in study one. For sudden gains, this is contrary to Tang and DeRubeis' (1999) original hypothesis which posited that sudden gains in CBT in particular are likely to lead to beneficial treatment outcomes because of cognitive changes. However subsequent sudden gains research also shows that sudden gains occur across therapies (both cognitive and non-cognitive therapies) and are associated with beneficial treatment outcomes across treatment types (Shalom & Aderka, 2020). For depression spikes, this is the first study to examine the association between depression spikes and treatment outcomes across low-and high-intensity therapies and further replication is needed. We note that these individuals were not randomly allocated to treatment, but if our results are replicated they perhaps suggest that some treatments may be more likely to bring about discontinues patterns of depression change. This concurs with Shalom and Aderka's (2021) hypothesis that treatment related factors may facilitate changes in the slope of depression change and cultivate a sudden gain. A limitation of study one was that it was only possible to examine immediate end of treatment outcomes because IAPT does not routinely follow-up with clients, and these data are often difficult to gather in regular clinical practice. Other research investigating patterns of

discontinuous change in RCT settings suggests there may be treatment differences at longer term follow up. For instance in a recent study, O'Mahen et al. (2021) found individuals who experienced sudden gains in CBT had significantly lower depression scores at 6- and 18-month treatment outcomes compared to their counterparts in BA. Further, they found depression spikes in CBT compared to BA were associated with non-significantly higher depression scores at 18-months follow up. Further research is needed to ascertain the longevity of the clinical benefits that have been found to be associated with experiencing discontinuous change in depression treatment, especially so for depression spikes where the research is limited.

6.1.1.2 What are the Key Client Processes and Therapist Strategies Preceding and Following Discontinuous Change in Depression Symptoms, and Are they Moderated by Treatment Type (CBT/BA)?

In the current thesis we found little evidence that key within-therapy client processes or therapist strategies prospectively predicted discontinuous change in the form of either a sudden gain or depression spike (see study two and three results in Table 6.1). Our sudden gains findings accord with the wider sudden gains literature which has also failed to find robust predictors of sudden gains, including baseline client demographic characteristics (Aderka et al., 2021; Zilcha-Mano et al., 2019) and within-therapy processes, such as cognitive (Abel et al., 2016; Hunnicutt-Ferguson et al., 2012; Lemmens et al., 2021) or behavioural (Lemmens et al., 2021) client processes or improvements in the therapeutic relationship (Lutz et al., 2013) in depression treatments (including CBT and BA). Within the depression spikes literature this is the first study to examine within-therapy processes predicting depression spikes in a trial that directly compared CBT and BA. Although none of the theoretically relevant

processes we examined in the pre-spike or life events in the spike session were found to be associated with depression spikes here, further replication is needed in other samples. There is some suggestion in the literature that periods of discontinuous depression change within treatment may just be an extension of natural fluctuations in depression symptoms which also occur outside of treatment (Aderka & Shalom, 2021). Rather than discontinuous change being the result of treatment-related factors (including changes in client or therapist processes), treatment may cause small perturbations in depression symptoms which, when a threshold is met, leads to a shift in depression symptoms (i.e. a sudden gain or depression spike). In their research group, Aderka and Shalom have found that depression symptom fluctuations predict sudden gains in therapy for PTSD, OCD and diverse disorders (Shalom et al., 2018), and pre-treatment symptom variation predicted sudden gains in internet delivered treatment for social anxiety disorder (Shalom et al., 2020). Although beyond the scope of the current thesis, research may wish to examine the association with pre-treatment depression symptom variability and patterns of discontinuous change in treatments for depression in particular. Although this research (study two and three) and other null results examining predictors of discontinuous change (Aderka & Shalom, 2021) may suggest we cannot elucidate factors that surrounding discontinuous change, it is important to continue to examine predictors of discontinuous change to understand why they occur. There are a number of relevant methodological considerations that will be discussed in turn.

One reason why predictors of symptom discontinuities were not identified in studies two and three could be the methodology used to identify processes of change. We coded therapy sessions preceding sudden gains and depression spikes using the CHANGE coding system. Coding systems are commonly used

to identify processes of change surrounding discontinuous depression change (e.g. Abel et al., 2016; Lemmens et al., 2021; Tang & DeRubeis, 1999) and have advantages over other methods, such as self-report, as we can capture multiple process variables across the therapy session. Another advantage is that coding systems allow for a more proximal measurement of processes, compared to assessing processes at baseline. Nevertheless, we can gather information only on the processes the coding system concentrates on. A range of transtheoretical processes are identifiable using the CHANGE coding system which means we can examine common and specific therapy processes, but from the literature we still do not know which processes of change we should focus on. Some suggest that cognitive processes are important to examine, particularly surrounding sudden gains (Tang and DeRubeis, 1999) and it is unclear, theoretically, which processes to examine when studying depression spikes in CBT and BA, which do not use therapeutic strategies to instigate depression spikes. Other research examining cognitive processes of change surrounding sudden gain sessions have used the 'Patient Cognitive Change Scale' (Lemmens et al., 2021; Tang & DeRubeis, 1999; Tang et al., 2005) which examines seven categories of cognitive change including awareness and acceptance of cognitive changes and belief changes. This differs from the CHANGE coding system which examines broader categories of cognitive change (e.g. overgeneralisation, cognitive flexibility, cognitive emotional processing). The findings from study four looking at cognitive and behavioural avoidance changes outside therapy, suggest that we need to look at person-specific variables that are contextually relevant and it may be that the processes in the CHANGE coding system are too broad. Another drawback of coding systems is that they only capture what is being said during the therapy session.

It is also possible that changes occur outside of the therapy sessions and this would not have been picked up during coding of therapy sessions in studies two and three unless the client had directly spoken about this. In addition to coding of psychotherapy processes future research may also want to use other methods in conjunction such as weekly self-report measures or ecological momentary assessment (EMA) approaches to examine change outside of therapy sessions. Moreover, because the coding of therapy sessions was conducted following the end of the trial it was not possible to ask clients what they think may have contributed to changes in depression symptoms. We used quantitative methods in the current thesis, but using qualitative methods during therapy to further understand depression change may provide rich information and consideration of other factors not captured by coding systems.

Another consideration is that we examined a limited number of therapist related variables in relation to discontinuous change and perhaps therapist characteristics may be important in facilitating discontinuous change. In study three we did not find therapist strategies (therapeutic relationship difficulty and levels of therapist cognitive and behavioural corrective information) were associated with experiencing a depression spike. In the wider literature, little other research has explored therapist factors related to depression discontinuous change, but a recent study suggests that therapist effects may play a role in the generation of sudden gains (Deisenhofer et al., 2021). Furthermore, in individuals with treatment resistant depression Abel et al. (2016) examined the role of case-conceptualisation, which is the process whereby therapists can understand the patient's problems to generate hypotheses about the development and maintenance of an individuals' depression symptoms and to help inform treatment strategies to use within

therapy to alleviate depression symptoms. They found that clients who had a sudden gain had therapists who demonstrated greater competence in case conceptualisation (Abel et al., 2016). These findings are important because they point to therapist procedure that happens early in treatment and may also be associated with hope for the client. Case conceptualisation may be especially useful because it pulls the client's problems together in a coherent manner and provides them with a clear and focussed directional path for treatment. No other research has examined therapist factors in relation to depression spikes. In the wider psychotherapy literature therapist effects contribute to variability in psychotherapy outcomes (e.g. Crits-Christoph et al., 1991; Firth et al., 2019; Johns et al., 2019; Kim et al., 2006; Okiishi et al., 2003) and further research is needed to understand whether particular therapist characteristics may play a role in generating patterns of discontinuous change in treatment.

Furthermore in the current thesis and the wider discontinuities literature the majority of studies (e.g. Abel et al., 2016; Lemmens et al., 2021; Tang & DeRubeis, 1999; Tang et al., 2005) look at processes of change in isolation. It is possible we have not found robust predictors because we have not looked at the relationships between processes. Limitations with study sample sizes can make this difficult to assess, but one study used sudden gain and depression spike sessions as times to explore processes of change and found when psychological flexibility was limited, maladaptive processes such as avoidance and rumination predicted poorer depression symptoms at 12-months post-treatment (Yasinski et al., 2019). Using other statistical methods, such as network analysis may help to elucidate the relationships between processes. Network approaches have been used in other ways in the psychotherapy field such as to examine early warning signals of mental problems (Fried et al.,

2017) and to predict relapse (Lorimer et al., 2020). Recently, Vittengl et al. (2021) demonstrated the use of network analyses to examine how the interconnection of depression symptoms change over time for depressed individuals receiving CBT. Future research could apply this methodology to examining changes in the relationships between processes prior to and over discontinuous depression change.

Alternatively, it may be the case that prior to discontinuous change there are smaller shifts in processes, but after the change in depression symptoms we may see changes in process. Examining this can allow us to further understand how therapists can respond to and embed the potentially positive changes or learning that might occur during periods of significant symptom fluctuations. Nevertheless relatively few studies have examined this (e.g. Tang & DeRubeis, 1999; Wucherpfennig, Rubel, Hofmann, et al., 2017; Zilcha-Mano et al., 2019), despite the clinical implications of doing so. In the current thesis we found that following sudden gains individuals exhibited lower avoidance levels regardless of treatment modality (study one) and, although not in a treatment context, higher levels of cognitive and behavioural avoidance predicted prospective greater depression levels (study four). In this study we highlighted the importance of examining processes over time and that there may be a natural recovery time. Although additional research is needed, this suggests that looking after the period of depression fluctuations may also be important too. Study four further contributed to our knowledge about what occurs during fluctuations of depression symptoms outside of treatment and highlights the importance of both cognitive and behavioural avoidance in the development of depression, but also as a maintaining factor. This study lends support to the behavioural theory of depression and highlights the role of

avoidance in depression over time. In the current thesis we did not examine differences in processes at the peak of the spike in individuals with depression spikes, because our comparison group were individuals who did not have a depression spike and were yoked to the timing of depression spikes. In order to better understand what is occurring during the peak of the spike a better comparison group may be individuals who experience a sudden loss. Sudden losses (Lutz et al., 2013) are the opposite of a sudden gain where there is a worsening of depression symptoms, but unlike depression spikes the worsening is not temporary. Further research examining this can help us to understand what occurs following an increase in depression symptoms and perhaps elucidate why some individuals' depression symptoms reduce (depression spikes) and others do not (sudden losses).

In addition to not finding key client processes and therapist strategies preceding and following discontinuous change, we also did not find they were moderated by treatment type (CBT/BA) in studies two and three. Despite this, a strength of the current work was that we used the same trial to examine individuals in CBT and BA in studies two and three. This ensured that treatment related factors such as trial protocol and settings and participant factors were similar. One possibility as to why we did not observe treatment differences is because of our sample sizes. However, the sample sizes in study two (n = 100) and three (n = 88) exceeds similar studies in the literature (Abel et al., 2016; Hayes, Feldman, Beevers, et al., 2007; Lemmens et al., 2021; Tang & DeRubeis, 1999; Tang et al., 2005). The intensive nature of coding may limit sample size in studies of this nature and as previously suggested perhaps other measures such as EMA or therapist measure of change in conjunction with

coding therapy sessions may also elucidate processes of change around periods of discontinuous change in CBT and BA.

The lack of treatment differences may suggest that, contrary to the specific factors debate, there are not specific treatment factors that contribute to depression change in CBT or BA, thus lending more support to the common factors view (Cuijpers, Reijnders, et al., 2019; Wampold, 2015). However, we note that we focused on key specific factors of change in studies one and two according to cognitive and behavioural theory, and further research is needed to further understand the role that common and specific factors play across therapies. Sudden gains were first identified to test the cognitive mediation hypothesis that cognitive change drives depression symptom changes specifically in CBT and can occur in early treatment (Tang & DeRubeis, 1999). Contrary to Tang and DeRubeis' (1999) 'upward spiral' hypothesis, we did not find any evidence of this in study two. We also did not find evidence of cognitive factors driving depression change in BA. It is also the case that CBT utilises behavioural strategies in early treatment sessions and therefore behavioural change could drive depression symptom change, but we found no evidence for this in study two. Similarly, around depression spikes sessions we did not find key cognitive or behavioural client processes or therapist strategies differed between CBT and BA. Overall our results do not accord with the cognitive mediation hypothesis.

Another important consideration that we did not explore in the current thesis is whether particular therapeutic strategies may lead to a change in client processes. As highlighted in the literature review of the thesis (chapter one), there is debate regarding the necessity of using cognitive change procedures to elicit depression change (Longmore & Worrell, 2007) and whether cognitive

change is specific to CBT (Hollon et al., 1987). Most of this literature focuses on cognitive strategies, but it may also be important to focus on the extent to which behavioural strategies in BA and CBT also elicit change in cognitive and/or behavioural processes. Recent experimental research has attempted to isolate cognitive change procedures to examine whether they have a direct impact on cognitive change in depressed individuals (Bruijniks, Los, et al., 2020; Bruijniks et al., 2018) and although they did not find the change procedures directly translated into cognitive changes, this is another potential avenue for future research. Understanding whether specific therapeutic change procedures lead to change in certain client processes could help us be more specific in therapy to target key hypothesised depression maintenance processes.

6.1.1.5 Do Key Client Processes or Therapist Strategies Preceding and Following Discontinuous Change Predict Treatment Outcomes, and Are These Moderated by Treatment Type (CBT/BA)?

Despite examining theoretically important client processes and therapist strategies surrounding patterns of discontinues change, in studies two and three we found few main effects of client and therapist variables upon treatment outcomes. The main effects that were found are difficult to interpret because they are not moderated by sudden gain or depression spike status and therefore simply show that levels of process in that session are associated with treatment outcomes.

Unfortunately we did not find that treatment condition (CBT/BA) moderated the effects of many key client and therapist variables on later outcomes. In an exception to this, in study two we found that the relationship of postgain overgeneralisation with outcome at 18-months post-treatment was

moderated by sudden gain status and treatment type. For individuals who experienced a sudden gain in CBT, higher levels of postgain overgeneralisation was associated with lower depression scores, but in BA higher postgain overgeneralisation was associated with higher depression levels at 18-months post-treatment. There were no other relationships between processes, discontinuous change, treatment type and outcome. Our finding supports Tang and DeRubeis' (1999) 'upward spiral' hypothesis which posited that further cognitive changes after a sudden gain in CBT creates a positive feedback loop to lead to sustained depression improvement at the end of treatment and follow up. Additionally, findings from a recent paper examining the long-term effects of sudden gains in this trial dataset showed that individuals who had a sudden gain in CBT, compared to BA, had better treatment outcomes at 18-months post-treatment (O'Mahen et al., 2021). Our study findings suggest that BA may not target some residual maladaptive symptoms, like overgeneralisation, and engaging in cognitive strategies in the face of overgeneralisation can enhance the benefits of experiencing a sudden gain. However, this is speculative pending replication. Our findings also indicate that therapists should be alert to maladaptive processes (such as overgeneralisation) that persist despite depression alleviation which may be important to target to fully maximise the sudden gain. Research has found during periods of depression remission depressogenic processes can persist for up to three years post-treatment (Conradi et al., 2011). These residual symptoms may act as risk factors for future depression episodes. This finding also highlights the need to understand the optimal times in which to engage in specific therapeutic strategies and perhaps in the face of overgeneralisation a cognitive focus may be beneficial.

Regarding depression spikes, there is some suggestion from a recent study that depression spikes in CBT may be associated with poorer long-term depression treatment outcomes, than depression spikes in BA (O'Mahen et al., 2021). In study three we focused on examining the processes in the spike session, which is in line with Hayes et al.'s (2007) hypothesis that the peak of the spike represents a time for corrective processing to occur. However, we note that there are three points of inflexion in a depression spike and it is possible that another point of examination (e.g. postspike session) may influence treatment outcomes. Our study was the first to examine client and therapist variables during a depression spike and their relation to treatment outcomes in CBT and BA. Further research is needed to understand what depression spikes represent in CBT and BA and the factors that influence how they may be related to treatment outcomes.

An unexpected finding in study two was that the majority of the three-way interactions between sudden gain status, treatment, and processes were in individuals who did not have a sudden gain. Conclusions about whether certain change processes are occurring in individuals who do not have a sudden gain in study two cannot be drawn because these individuals were yoked to the timing of sudden gain. Although the focus of this thesis was to elucidate processes surrounding discontinuous change in CBT and BA, this also highlights the need to examine processes of change in individuals who do not experience discontinuous depression change in treatment, whom we know are vulnerable to disadvantageous depression outcomes (Andrews et al., 2020; Hayes, Feldman, Beevers, et al., 2007; Shalom & Aderka, 2020; Vittengl et al., 2016).

A range of methodological, clinical and theoretical implications of the thesis findings have been discussed in this chapter and these are summarised in Table 6.2.

Table 6. 2

Summary of the Methodological, Theoretical, and Clinical Implications from the Thesis

Methodological Implications	Theoretical Implications	Clinical Implications
• Using a combination of coding manuals (like the CHANGE coding system) in conjunction with weekly self-report measures and EMA methods to examine other processes and change occurring outside therapy • Further research is needed to examine the relationship between processes, and methods like network analysis can be utilised	• Our findings do not accord with the cognitive mediation hypothesis • Little research examines whether depression change may be as a result of behavioural change processes and further research is needed	• Despite depression alleviation therapists should be alert to maladaptive processes that may be important for future depression outcomes • Further research is needed to understand what therapist procedures can be used to maximise the positive effects and minimise any disadvantageous effects of experiencing discontinuous depression change

6.2 Conclusion

In conclusion, the current thesis presents research that furthers our understanding of patterns of discontinuous depression change. We demonstrated how discontinuous change occurs in everyday clinical settings, outside of therapy, and explored theoretically important client processes of change and therapist strategies surrounding depression change in an RCT setting. The thesis provides a basis to help in the generation of hypotheses for future research. Ultimately research examining how treatments work can help to refine and improve the effectiveness of treatments for depression in the future.

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