An Investigation of Early Sudden Gains in Cognitive-Behavioural Therapy for Depression: Client and Within-Therapy Predictors of Change

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ABSTRACT

Research into discontinuous change patterns across therapeutic treatment has indicated that clients who experience non-linear change patterns (e.g., 'depression spikes', 'transient worsening', and 'sudden gains') have comparatively better outcomes in therapy (Haas, Hill, Lambert & Morrell, 2002; Hayes, Laurenceau, Feldman, Strauss & Cardaciotto, 2007; Illardi & Craighead, 1994; Thompson, Thompson & Gallagher-Thompson, 1995). The focus of the current study is on the discontinuous change patterns that have been identified as *sudden gains*, where a client shows a large symptom improvement from session to session of therapy (Tang & DeRubeis, 1999). Research into the phenomena of *sudden gain*s has indicated that they are associated with better outcomes within therapy and post therapy; they may help identify clients who will respond favourably to therapy; and that they may provide further clarification around change mechanisms and processes within therapy. The current study had two aims: (1) to investigate the client factors that may predict sudden gains in Cognitive Behavioural Therapy (CBT) for depression; and (2) to investigate the within-therapy factors that may be associated with sudden gains within CBT for depression.

Through an overarching depression study at the School of Psychology, Massey University, a final sample of 28 clients experiencing their first episode of Major Depression (MDE) were recruited. They participated in 20 free sessions and two follow-up sessions of CBT for depression. Depression severity was measured at every session using the *Beck Depression Inventory – Second Edition (BDI-II).* Attributional style was measured at six time points using the *Attributional Style Questionnaire (ASQ).* Homework was measured at up to 18 time points using the *Homework Rating Scale – Second Edition (HRS-II) – Client Version.* A longitudinal multi level design method was used to analyse the data. 42.9% (n=12) of the clients experienced a sudden gain and these clients experienced a faster rate of improvement in depression severity across treatment. Clients' attributional style at intake moderated the relationship between sudden gains and rates of change in depression severity across treatment. No moderating relationship was found with either initial symptom severity or co-morbid status at intake and sudden gains and improvement in therapy. Within therapy variables such as attributional style change and homework beliefs across therapy did have a moderating effect on the relationship

between sudden gains and improvement in depression severity across treatment. Clinical considerations and implications for future research are discussed.

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CHAPTER ONE

DEPRESSION AND COGNITIVE BEHAVIOURAL THERAPY: AN INTRODUCTION

Introduction

Depression is one of the world's most debilitating health problems. Murray and Lopez (1997) described depression as the number one source of disability worldwide. This chapter will review the clinical diagnosis of depression. It will consider the individual and social cost of depression and the prevalence rate in New Zealand will be outlined. This chapter will also provide an overview of the aetiology (or causes) of depression. It will explore the cognitive theoretical perspective of depression, introduce cognitive behavioural therapy (CBT) and outline the empirical evidence supporting the use of CBT in the treatment of depression.

Definition of depression

Depression is a commonly used term that usually refers to the depressive disorders as defined by the Diagnostic and Statistical Manual-4th Edition-Text Revision (DSM-IV-TR; APA, 2000). Depressive disorders are a subset of the mood disorders category and include Major Depressive Disorder and Dysthymic Disorder. The clinical diagnosis of depression is contingent on the presence of at least one Major Depressive Episode (MDE). The main feature of an MDE is a period of time lasting at least two weeks during which there is depressed mood or the loss of interest or pleasure in nearly all activities. The DSM-IV-TR diagnostic criteria for MDE include such symptoms as feelings of worthlessness or guilt, concentration difficulties, changes in appetite or weight, sleep and psychomotor activity; decreased energy, difficulty thinking or making decisions; and/or recurrent thoughts of death or suicidal ideation, plans or attempts. To diagnose an individual with depression s/he must experience at least five of these symptoms. They must persist for most of the day, nearly every day for at least two weeks. The episode must also be accompanied by significant impairment or distress in at least one important area of functioning (e.g., social, occupational) (APA, 2000).

Prevalence in New Zealand

There are a large number of people that are affected by depression. According to the DSM-IV-TR (APA, 2000) the lifetime prevalence of major depression is 10-25% for women and 5-12% for men. In New Zealand there have been two recent major studies (discussed below) that have investigated the prevalence of depression in the New Zealand population. The first of these was conducted by the Mental Health and General Practice Investigation (MaGPie) research group from the University of Otago. This study collected information in regards to the prevalence of common mental health disorders among patients who attend NZ general practices. This research showed that mental health difficulties were indeed common among the patients who attended general practices and that approximately half of those patients had been identified as having some type of psychological difficulty in the previous year (The MaGPIe Research Group, 2003). Another important piece of research was the Te Rau Hinengarau: The New Zealand Mental Health Survey, which released its initial findings in September 2006 (Oakley-Browne, Wells, Scott & McGee, 2006). The study was commissioned by the Ministry of Health and over 13,000 interviews were carried out to examine the prevalence of mental illness in the general New Zealand population. Findings showed that mood disorders and depression are common in the New Zealand population. Specifically, the lifetime prevalence rate of any mood disorder in the general New Zealand population was estimated at 20.2% (Oakley-Browne et al., 2006). The lifetime prevalence rate for major depressive disorder was estimated at 20.3% for women and 11.4% for men (Oakley-Browne et al., 2006). Overseas research (e.g., Piccinelli & Wilkinson, 2000) had demonstrated that depression is more commonly experienced by females than males. The higher rate of depression among females is said to be explained by a number of factors, including artifactual determinants (e.g., the tendency for females to report more depressive symptoms than males); risk factors (e.g., familial environment and adverse experiences in childhood) prior depression and anxiety disorders (e.g., females are at an increased risk of depression and anxiety at a younger age) and social roles and cultural norms (e.g., having to fulfil a greater number of different societal roles than men generally) (Piccinelli & Wilkinson, 2000).

The impact and cost of depression

Depression does not only pose a significant cost to the individual, but it has major implications for society. A major World Health Organisation study (Ustun, Ayuso-Mateos, Mathers & Murray, 2004) predicted that depression would be the second leading cause of disability worldwide, next to ischemic heart disease. Depression can lead to a number of direct and indirect costs that can create a significant economic burden, including but not limited to medical resources and professional expertise expended in treating depression, loss of earnings and reduced production due to work absenteeism, early retirement and premature mortality (Berto, D'llario, Ruffo, Di Virgilio, & Rizzo, 2000; Luppa, Hienrich, Angermeyer, & Koing, 2007). Furthermore, as well as having a high rate of incidence, depression is a condition that is marked by relapse, reoccurrence and chronicity (Scher, Segal & Ingram, 2004).

An Australian study (Hawthorne, Cheok, Goldney & Fisher, 2003) found that those with major depression reported worse health status, took more time off work, reported more work performance limitations, made greater use of health services and reported poorer health-related quality of life. It has been claimed that allocating resources to help make the treatment of depression more effective and efficient would result in marked financial and social net benefit (Hawthorne, et al., 2003). Even small gains in functional capacity for depressed individuals could potentially benefit the country as a whole (Australia). For instance, it has been estimated that Australia could benefit by approximately one billion dollars a year (Goldney, Fisher, Wilson & Cheok, 2000).

Aetiology of depression

As with many other mental health problems, there is no absolute consensus of what causes depression. Instead, years of research have identified that there is a wide range of predisposing factors, both genetic and environmental, that are significant predictors of depression (Sullivan, Neale & Kendler, 2000). Stress has long been recognised in the literature as playing an important part in the development and course of different mental illnesses. Ingram and Luxon (2005) describe 'stress' as major or minor life events that disrupt the mechanisms that maintain an individual's stability in terms of physiology, emotion and/or cognition. Mazure (1998) found that approximately 50% of individuals diagnosed with depression have experienced

severe life stress before the onset of depression. Although these findings show that stress plays an important part in the development of depression it does not explain why the other 50% of people also develop depression in the absence of severe life stress. Therefore, vulnerability factors must also play a significant role. A *diathesis* or *vulnerability* is typically described as a pre-dispositional factor, or set of factors (e.g., genetic, biological, psychological) that make possible a disordered state (Ingram & Luxon, 2005). So-called *diathesis-stress* models suggest that all people have some level of predisposing factors for any given mental health difficulty (Ingram & Luxon, 2005). However, each individual will have his/her own point at which they will develop a given disorder. This point will depend on the degree to which the predisposing factors exist and the degree of stress experienced by the individual (Ingram & Luxon, 2005; Monroe & Simmons, 1991). These types of models also recognise that the moderating factors (e.g., psychological, biological, demographic, developmental etc.) that contribute to the development of a depressive disorder or other psychiatric illness are dynamic rather than static.

The cognitive theory model and the stress diathesis model

The cognitive model of depression is an explanatory model that posits that people who are depressed have a stable set of *core beliefs* or *schema* that develop as a result of experiences early in life (Beck, 1964). These schema are said to predispose these depressed individuals to negative interpretations and unhelpful thinking styles (i.e., *systematic cognitive errors*), also known as automatic thoughts, in response to certain situations. For example, if a person is exposed to chronic negativity, stress or abuse in their childhood, the schemas that s/he develops may guide her/his attention to negative rather than positive events, which consequently lead to enhanced recall of negative events and experiences, and information could be distorted to fit these schemas (Scher, et al., 2004). A person's schema is their basis for screening out, differentiating and coding stimuli, therefore they will determine how people structure their experiences. Cognitive mechanisms refer to faulty (or unhelpful) information processing (Scott & Freeman, 2010). As depression becomes worse, it is theorised that people become less able to acknowledge that their negative interpretations are false. Errors in cognition or information processing errors are theorised to maintain dysfunctional states, these occur when individuals selectively screen out information in their environment that supports or refutes their view of themselves, their world

and their future (also known as the cognitive triad). One example of an information processing error is *over-generalising* – this involves drawing a general rule or conclusion on the basis of one or more incident and applying this concept along other situations and incidents that may be related or not. Another example is *dichotomous thinking* – where an individual tends to put all experiences into two opposite categories; for example flawless or defective (Beck, Rush, Shaw & Emery, 1979).

Like many other stress-diathesis models, the mere presence of a negative self-schema would not be sufficient to precipitate depression. Rather, the cognitive model of depression suggests that these schemas will lie dormant until activated by relevant experiences, events or stimuli (Beck, 1964). Empirical data evaluating the cognitive stress-diathesis model has grown considerably over the last 15 years (see Scher et al., 2004 for review of literature). This body of literature largely supports the diathesis-stress theory proposed in cognitive models of depression (Scher et al., 2004).

Other cognitive based theories of depression

Another important cognitive model is the 'hopelessness theory of depression'. Abramson, Alloy and Metalsky, (1989) suggest that people who are more vulnerable to depression will typically (1) attribute negative life events to stable (likely to persist over time) and global (likely to influence many parts of life) causes, (2) infer that current life events will lead to negative consequences, and (3) believe that negative events in their life mean that they are somehow flawed or worthless (Abramson, Alloy, Hogan, Whitehouse, Donovan, Rose, Panzarealla, & Raniere, 2002). Those who develop this inferential style are more likely to make negative inferences about the cause, consequences and self-implications of a negative life event, and therefore develop 'hopelessness' which may eventually lead to what Abramson and colleagues (2002) call 'hopelessness depression'. Beck (1964) had proposed that hopelessness is a hallmark of depression, and it consequently inhibits a person's ability to generate solutions to problems and adaptive beliefs. Both Beck's model and the *hopelessness theory of depression* are diathesis-stress models in which cognitive style is the diathesis. The combination of negative life events and cognitive style are seen to contribute to the cause of depression.

Cognitive behavioural therapy

Cognitive Behavioural Therapy (CBT) is a well established and widely used time-limited treatment for depression which evolved from Albert Ellis's Rational Emotive Behaviour Therapy (1962) and Aaron T. Beck's cognitive theory (CT) (Beck, 1964). Over thirty years ago a key manual (Beck, et al., 1979) was developed that integrated cognitive therapy with behavioural therapeutic techniques in the treatment of depression. A cognitive-behavioural model of depression would consider several aspects of an individual's life as important (including cognition, behaviour, mood and physiology) in contributing to his/her experience of depression. The main emphasis of CBT is to give individuals the cognitive and behavioural tools that will assist their 'adaptive functioning' and improve their overall well-being (Kazantzis, 2006). CBT is a relatively short term structured therapy that depends on a strong therapeutic alliance and collaborative empiricism between the therapist and the client. Techniques used in cognitive therapy are aimed at testing an individual's specific misconceptions and 'maladaptive' assumptions (Beck, et al., 1979). CBT is aimed at teaching clients the following: to monitor their automatic negative thoughts; to recognise the connections between cognition, affect and behaviour; examine the evidence for and against distorted automatic thoughts; and learn to alter and identify the 'dysfunctional beliefs' that predispose the person to distort their perceptions of themselves (Beck et al., 1979).

CBT as a modality of psychological treatment is very popular in New Zealand. Kazantzis and Deane (1998) found that in a sample of 221 New Zealand psychologists 55% identified CBT as their primary therapeutic modality. Another 33% of respondents classified themselves as eclectic of which 80% identified CBT as one of their therapeutic modalities of choice. Merrick and Datillio (2006) point out that there is a worldwide trend from 'consumers' (e.g., service providers, service users, individuals seeking treatment) for professional and economic accountability. They assert that in New Zealand the health and insurance industry and individual clients seek brief, cost effective solution based interventions that carry some empirical validation. Not only is CBT popular with professionals but it is also popular with the individuals who engage in therapy. Merrick and Datillio (2007) suggest a number of reasons why CBT is so widely accepted in New Zealand, including the fact that CBT has empirical support, is brief and time-limited, is

present-focussed, solution-focussed, is collaborative and gives the client(s) a sense of control over their recovery, and generally CBT has acceptance and is promoted in the field of medicine and psychiatry.

Evidence-based practice and cognitive behavioural therapy

In many countries, including New Zealand, there has been a call for psychotherapeutic interventions to show effectiveness and efficacy (Emmelkamp, Ehring & Powers, 2010; Merrick & Datillio, 2007). The psychotherapy research field has increasingly been able to demonstrate the efficacy and effectiveness of specific procedures for specific DSM-IV disorders (e.g., depression, social anxiety, post-traumatic stress disorder) (Emmelkamp, et al., 2010). The methodological approach that is commonly utilised to define what is 'best practice' or empirically supported in the field of clinical psychology is the randomised controlled trail (RCT). RCT studies are ultimately large in scale and they randomly assign individuals to a treatment of interest or to a comparison condition of some kind (Chambless & Hollon, 1998). This research methodology into the *effectiveness* of psychological treatments was proposed by Division 12 of the American Psychological Association Task Force. A treatment would be given the label *possibly efficacious* if it was found to be more effective than no treatment in a single RCT. If these findings were subsequently replicated in another RCT, conducted by an independent research team, the treatment may then be referred to as *efficacious*. Furthermore, if an RCT sets conditions in its methodology that control for non-specific processes or another bona-fide treatment, this treatment is said to be *efficacious and specific* in its mechanisms of actions (Chambless & Hollon, 1998; Chambless & Ollendick, 2001). Therefore, this *evidence-based practice* approach is normative and emphasises group means rather than individual cases.

Randomised controlled trails of therapies such as CBT and Interpersonal Therapy (IPT) have yielded favourable results as they are generally brief, definable and structured. Cognitive behavioural therapy has been shown to be an efficacious and effective psychological treatment for depression across a variety of clinical settings (Butler, Chapman, Forman, & Beck, 2006; DeRubeis & Crits-Christoph, 1998). Due to its popularity, CBT has been researched extensively in clinical populations, and results indicate that it is at least as effective as anti-depressant medications in respect

to symptom reduction and reduction of acute distress (Hollon & Beck, 2004). A recent overview (Beck, 2005) highlighted the breadth of research on different cognitive models of psychopathology. There is a substantial body of evidence for the model of depression for which Cognitive Therapy has been applied as a treatment.

The 'evidence-based practice' approach is advantageous as it means that there are clear objective criteria for clinical decision-making when deciding which treatment would be particularly effective for individuals with specific DSM-IV defined mental diagnoses (Emmelkamp et al., 2010). Furthermore, it is argued that manualised treatments such as CBT or IPT are easy to disseminate for clinicians and are useful for further research and training. However, there are also critiques to using such a methodological approach to assess the effectiveness of treatment approaches. One major concern is the homogenous nature of the populations that these studies are drawn from. Bennett (2009) questions the RCT findings and their applicability to the New Zealand population. The majority of the large scale RCT studies have been carried out in the US and have drawn their participants from populations with relatively limited ethnic diversity (Bennett, 2009). Not only does the diversity of geography, ethnicity and culture need to be considered when making 'best practice' inferences about treatment, but the reality of co-morbidity of psychiatric illnesses needs to be considered. For instance, in New Zealand the most common co-morbidity is between mood and anxiety disorders. It was found that 49.6% of people who presented with a mood disorder had also experienced an anxiety disorder within a 12 month period (Scott, McGee, Oakley-Browne & Wells, 2006). Furthermore, individuals who develop substance use disorders are likely to have a concurrent mental health difficulty (Emmelkamp & Vedel, 2006).

The evidence-based approach is based on 'disorder specific' thinking. It ultimately assumes that patients with a certain diagnosis will respond in a uniform way to a certain treatment (Emmelkamp et al., 2010). In practice, people with mental health difficulties such as depression will often have complex presentations, usually with one or more concurrent DSM-IV-TR diagnoses. Therefore, manualised treatments for specific diagnoses may not be applicable to clinical reality. Powers and Emmelkamp (2009) have noted that very few practitioners are inclined to use the treatment manuals that are designed for RCTs. Likewise, Addis and Krasnow (2000)

found that only 7% of psychologists reported using treatment manuals regularly, and furthermore, only 20% reported being clear on what a treatment manual actually was. Emmelkamp and colleagues (2010) emphasise that there is a clear need for more 'theoretically driven' rather than 'disorder specific' decision making in prioritising treatment targets.

Empirical validation of cognitive behavioural therapy for depression

There is a breadth of research on cognitive models of psychopathology and a substantial body of evidence that supports the cognitive theory of depression (see Beck, 2005 for review). Meta-analytic techniques are often used to quantify the efficacy of a treatment in terms of an effect size (ES). An ES indicates the magnitude of an observed effect in a standard unit of measurement. Effect sizes can be relatively high (ES≥0.8), medium (0.5≤ES>0.8), low (0.2≤ES>0.5), or said to have no effect (ES<0.2) (Cohen, 1988). A number of meta-analytic outcome studies have demonstrated that CBT is highly effective for depressive disorders when compared to waitlist or control conditions (e.g., Butler, et al., 2006). A review by Butler and colleagues (2006) identified over 300 published outcome studies on cognitive therapy and over 16 meta-analyses. Data on mean ES was used to assess specifically, (i) the *effectiveness* of cognitive therapy, (and what disorders is it most effective for) and (ii) the durability of gains made in therapy. They found that, according to a number of meta-analytic studies, compared to waitlist or placebo treatments, CBT is highly effective for depressive disorders. However, it is only marginally superior and/or equivalent to other active treatments.

Studies asserting the effectiveness of CBT have been challenged by claims of overstated findings, publication bias, allegiance effects and presumed lack of bona-fide control (Beck, 2005; Cuijpers, Smit, Bohlmeijer, Hollon and Andersson, 2010). Cuijpers and colleagues (2010) argue that previously reported effect sizes are overestimated because of publication bias (i.e., the tendency for increased publication rates among studies which show a statistically significant effect of treatment). Cuijupers et al., (2010) examined the effect sizes of 117 previous trials with 175 comparisons between psychotherapy and control conditions. After using several statistical adjustments to take into account publication bias, the overall effect size for cognitive behavioural therapy fell from 0.69 to 0.49 (Cuijpers et al., 2010).

This suggests that the effect of psychotherapy for depressed adults appears to be overestimated due to publication bias. Another factor that may contribute to the overestimation in meta-analyses for psychotherapy is the quality of the study. Cuijpers, vanStraten, Bohlmeijer, Hollon and Andersson (2010a) found that when comparing studies that meet strict quality criteria (i.e., participants meet diagnostic criteria for depressive disorder, a treatment manual is used, the therapists were trained, treatment integrity was checked, intention to treat analyses were used, $N \geq 50$, randomisation was conducted by an independent party, and assessors of outcome were blinded) the standard mean effect size found for the high quality studies ($d = 0.22$) was significantly smaller than those studies that did not meet these quality criteria ($d = 0.74$, $p < .001$). This study further suggests that the effects of psychotherapy for adult depression have been overestimated in meta-analytical studies.

Overall, the question of whether a particular psychotherapy is superior to others has created controversy (e.g., Luborsky, 1995; Wampold, 2001). The 'dodo bird verdict' argues that all therapies are equally effective (from a line in Alice in Wonderland, "All have won and all must have prizes"). Research (e.g., Messer & Wampold, 2001) that supports this 'verdict' cites meta-analytic evidence. However, others critique the fact that these meta-analyses tend to aggregate the outcomes for all treatments across all disorders which potentially obscures any real differences between specific psychotherapeutic treatments for specific disorders (Butler et al., 2006). Although there are good arguments that the benefits of CBT for depression have been overstated, there is still good evidence to suggest that CBT does produce benefits for some clients and this should not be discounted.

Research shows that CBT works (Butler, et al., 2006; Hollon & DeRubeis, 2004), but how it works is somewhat unclear. Beyond determining whether CBT is effective, researchers also want to find out what the mechanisms, moderators and predictors of response to cognitive therapy. Some researchers (e.g., Lambert, 2005; Wampold, 2001), have argued for the important role of 'non specific' or 'common factors' across all therapies. In the 1970s Frank (1973) drew attention to the common factors that different modalities of therapy share. Four non-specific therapeutic factors were postulated by Frank (1973): 1) an emotionally charged, confiding therapeutic relationship; 2) a healing setting; 3) a rationale providing plausible

explanation for the symptoms and logic for the recommended procedure; and 4) a treatment procedure believed by both the client and the therapist to be restorative. Lambert and Ogles (2004) have summarised the variety of different common factors into three categories, 'support factors' (e.g., therapeutic alliance), 'learning factors' (e.g., feedback, rationale) and 'action factors' (e.g., mastery, practice). Wampold (2001) suggests that there is little evidence that specific treatment ingredients lead to change (or what he calls the 'medical model of therapy') but strong evidence for a 'contextual model' (i.e., a model that relies heavily on common factors as the primary agents of change in therapy). However, Lambert and Ogles (2004) emphasise that specific therapeutic techniques should not be labelled irrelevant, but it should be remembered that their power for change will be limited if essential common factors such as a therapeutic alliance are not in play.

In recent years there has been a focus on process variables in therapy and some clinically observable and measurable phenomena have been highlighted in the research that may help answer the questions such as "how does therapy work?" "what determines/predicts the efficacy of therapy?", "how do we target therapy more effectively?". Researchers (see Hayes, Hope & Hayes, 2007) are making more use of longitudinal methods to attempt to answer these questions. This type of research employs more frequent assessments across treatment, the study of individual trajectories over time, rather than group patterns, and pays attention to non-linear patterns of change that may not fit in with a traditional dose-response curve (Hayes, et al., 2007). One such phenomenon that has received attention in recent research is *sudden gains*. For instance, Tang and DeRubeis (1999) found that some depressed clients who were engaged in CBT for depression showed substantial symptom improvement in a single between-session interval. Furthermore, these types of gains were associated with better long term outcomes and lower incidence of relapse. Another phenomenon is *depression spikes*. Hayes, Feldman, Beevers, Laurenceau, Cardaciotto and Lewis-Smith (2007a) found that periods of transient worsening were associated with lower post-treatment depression. This research highlights the importance of measuring outcome variables throughout the course of therapy rather than just relying pre- / post- measures to establish whether the therapy works. Furthermore, non-linear patterns of change and discontinuities in individual time course data may point to segments of the therapy that can reveal

potentially important processes of change (Hayes, Laurenceau, Feldman, Strauss & Cardaciotto, 2007b). Several studies have highlighted potential cognitive mechanisms that could account for the active ingredients in CBT. Teasdale, Scott, Moore, Hayhurst, Hope and Paykel's (2000) research suggests that CBT will modify the form of thinking (e.g., all-or-nothing thinking style), consistent with the notion that CBT helps clients gain compensatory or meta-cognitive skills, which in turn predicts better outcomes at the end of therapy. Furthermore, Fresco, Segal, Buis and Kennedy (2007) found that those individuals who responded to CBT showed significantly greater gains in *de-centring*[1] compared to anti-depressant responders, and that higher levels of de-centring post-treatment was associated with lower rates of relapse. This line of research suggests that cognitive variables may play an important role in symptom change. However, Hollon, Stewart and Strunk (2006) warn that much uncertainty remains around whether the benefits of CBT come about because the causal processes that generate the risk are resolved (e.g., schemas), or whether the individual has developed compensatory skills to offset the causal processes. Furthermore, uncertainty also remains around whether benefits of CBT reflect the mobilisation of cognitive resources or other mechanisms of action. Therefore, although common factors have shown to play an important and integral role in therapeutic outcome (see Wampold, 2001), recent studies have indicated that there is some evidence for the role of cognitive meditational change processes operating in CBT and therefore specific ingredients of CBT may play a role in this change.

Conclusion

This chapter has outlined the clinical presentation of depression, its prevalence, and its costs to both individuals and wider society. The large costs associated with the illness highlights the importance of finding effective treatment solutions for those who experience depression. Stress-diathesis models postulate that there is no single cause to depression. The cognitive theory of depression is consistent with a stress-diathesis model, emphasising the interaction of cognitive style and/or schema and negative life events/stress in the development of depression. This chapter outlined the cognitive model of depression and the cognitive-behavioural treatment model. Cognitive behavioural therapy is a popular modality of psychological treatment in

[1] De-centering is an individual's ability to observe their thoughts and feelings as transitory events in the mind that do not necessarily reflect reality (Sauer & Baer, 2010)

New Zealand and it has thrived under conditions that call for 'evidence based practise'. This chapter has outlined the advantages and disadvantages of RCT based research. It acknowledges that research conditions in RCTs may not reflect clinical reality. Researchers have called for more 'theoretically driven' rather than 'disorder specific' decision making when prioritising treatment targets (Emmelkamp et al., 2010). This chapter also acknowledges that previous empirical evidence on the benefits of CBT for depression may have been overstated. The argument for and against specific cognitive ingredients in the change mechanisms of therapy has been outlined. Although some researchers argue that the majority of change is due to 'common factors' such as the therapeutic alliance recent studies have demonstrated some evidence of cognitive mechanisms affecting change within the therapeutic process (e.g., the development of meta-cognitive skills). Furthermore, the trend towards asking *how* change comes about in therapy has led to researchers to make use of longitudinal methods to reveal important processes and mechanisms of change. Exciting findings within the literature around CBT for depression outcome studies are phenomena such as 'sudden gains' and 'depression spikes'. For example, studies show that those who experience 'sudden gains' have comparatively better treatment gains at the end of therapy and they are less likely to relapse than those who do not experience 'sudden gains' (Tang & DeRubeis, 1999). It is asserted that these types of phenomena may be a key to pin pointing important processes and mechanisms of change throughout CBT (Hayes, et al., 2007a).

CHAPTER TWO

HOW DO WE MEASURE OUTCOME IN COGNITIVE BEHAVIOURAL THERAPY FOR DEPRESSION?

Introduction

The ways in which psychotherapeutic outcomes are empirically measured and assessed have changed over time. Traditional means of outcome assessment, which have predominantly focussed on pre- to post- symptom changes, have found CBT to be an effective treatment for a variety of mental health problems when compared to comparison or control groups (Newnham & Page, 2007). However these pre- versus post- symptom approaches to outcome assessment are limited by their ability to modify practice for a particular client in 'real time'. Additionally not all clients are the same and not all benefit from treatment in the same way (Newnham & Page, 2007). This chapter introduces two models of psychotherapy process-outcome research, the *dose-response model* and the model of *clinical significance*. Outcome research into CBT as a therapy has moved beyond merely demonstrating whether a treatment works, but rather it tries to understand why it works and why it does not. While traditional research methodologies have focused on average group outcome, there has been a shift towards client-focused longitudinal research, such as sudden gains methodology, which investigates an individual's response to treatment (Hayes et al., 2007; Newnham & Page, 2007).

Dose-Response Model of Psychotherapy Outcome

The most appropriate amount of psychotherapy to address any one mental health problem is of interest to clinicians, consumers and those that fund mental health care. The *dose-response* relationship (based on the medical model) examines whether patients with different characteristics need different amounts of therapy, and do otherwise 'equal patients' show different outcomes when given different levels of a particular type of therapy (Feaster, Newman & Rice, 2003). Howard, Kopta, Krause and Orlinsky (1986) conducted a meta-analysis on 2,431 patients from published research spanning a 30 year period. They reported a pattern across studies reflecting the relationship between the amounts of therapy and improvement in therapy. This was a positive relationship characterised by a negatively accelerated curve (i.e., the

greater the amount of psychotherapy, the greater chance of improvement with diminishing returns at higher doses). The type of therapy analysed in this study was predominantly psychodynamic or interpersonal. It was found that approximately 15% of patients improved before the first session of therapy, 50% of patients typically improved at 8 sessions, 75% at 26 sessions, and 85% at 52 sessions (Howard et al., 1986). Therefore, the greater the amount of psychotherapy the greater the chance of improvement with diminishing returns at higher doses. This relationship is important for practical reasons, for example in terms social policy, it helps to answer the question of *how much* therapy is needed for clients to obtain adequate benefit from therapy (Lambert, Hansen & Finch, 2001). The findings from Howard et al., (1986) study support the findings that treatment produces benefits for clients that surpass simply spontaneous remission rates. It also demonstrated that clients receiving therapy make substantial gains early on in treatment (Lambert & Ogles, 2004).

Limitations of the dose response model

While the dose-response model has contributed useful information in terms of answering questions about how much therapy is required, it is not without its limitations. For example, there were methodological limitations within the Howard and colleagues (1986) study. First of all, the therapeutic approaches within the study were predominantly psychoanalytic and interpersonal, questioning whether the same effect would be observed with other forms of therapy (Phillips, 1988). Furthermore, the study was lacking any consistent outcome criteria. It was problematic to 'lump' such a number of people (over 2,000) together to show a general pattern of change. Additionally, the outcome criteria in this study were inconsistent regarding aspects of 'client functioning' that were measured, and the magnitude of change necessary to indicate significant clinical improvement was vague (Kadern, Lambert & Andrews, 1996). Different criteria may be important for different populations, diagnoses, or individuals. Thus, rather than a general recovery curve, it may be better to rely on more specific curves based on variables of particular interest (Lambert & Ogles, 2004). For example, Kopta, Howard, Lowry and Beutler (1994) suggest that recovery rates may vary according to whether clients are experiencing acute or chronic distress. They administered the Symptom Checklist-90-R (Derogatis, 1983) to 854 outpatient clients. The symptoms were then separated into three groups (*acute*

distress, *chronic distress* and *characterological distress*) based on analysis of the test scores. It was found that for those identified as experiencing 'acute distress', clients reached a recovery level within 10 sessions, for those identified experiencing 'chronic distress' the number of sessions was extended to 14, and for characterological items fewer than 59% of the clients recovered on any of these items at 52 sessions (Kopta et al., 1994). Furthermore, Barkham, Rees, Shapiro, Stiles, and Agnew's (1996) study found that some classes of symptoms responded more quickly than others and that there were faster rates of change on acute symptoms as opposed to chronic symptoms. Recent studies (e.g., Barkham, Connell, Stiles, Miles, Margison, Evans & Mellor-Clark, 1996; Stiles, Barkham, Connell & Mellor-Clark, 2008), have challenged the usual thinking behind the dose-response relationships in psychotherapy research. It is important to keep in mind that the usual interpretation of dose-effect curves resembles concepts based on the medical model (i.e., where the dose-effect is the physiological response observed when otherwise 'equal' individuals are given differing amounts of a compound) rather than a holistic view of mental health (Howard et al., 1986; Kopta, 2003).

One of the major criticisms of the dose-effect relationship is that it assumes change is linear. The reliance on pre- and post- estimates of patient improvement (rather than session-by-session ratings of improvement) makes it difficult to pinpoint the exact time of recovery (Lambert & Ogles, 2004). Furthermore, reliance on pre- / post-measures to assess change throughout treatment can miss vital information that may be occurring within therapy. Naturalistic studies (e.g., Feaster et al., 2003) that investigate client change in psychotherapy show that many of the reasons for termination during therapy are outside of the researchers' control. For example, where a third party makes the decision to terminate or when a therapist decides further treatment will not benefit the client. In these situations termination is seen as an endogenous variable (Feaster et al., 2003).

Models such as the *responsive regulation model* (Stiles, et al., 2008) emphasise that clients are active decision makers rather than passive recipients of mental health services. This model suggests that in routine practice, a client's level of improvement and the treatment duration are mutually regulated so that the end of treatment would come when a client has improved to a 'good enough level' (Stiles et al., 2008). The *responsive regulation model* suggests that clients and therapists will act together as

rational decision makers to decide when a client reaches their 'good enough level' and can terminate therapy. Feaster et al. (2003) point out that when outcome variables (e.g., depression severity) are measured at the termination point of therapy, growth curves that include this observation time will be biased. It is argued that clients have different factors at different time points in therapy that will either increase or decrease their well-being and it is likely that the decision to terminate for a client will occur when a 'positive shock' occurs during the period of therapy. These positive shocks are likely to raise the client's wellbeing, potentially biasing the information measured (Feaster et al., 2003). The argument is that outcome should not be assessed just at the point of termination, because this is dependent on the therapeutic process. It is asserted that to 'fix' this endogeneity problem in naturalistic studies, fixed time points of assessment need to be administered that are independent of when the therapy terminates (not simply pre-/post- measures) (Feaster et al., 2003). Therefore, session-by-session measurement of outcome across therapy may provide more answers on why and how therapy works. Another methodological approach in client-focussed research has addressed is whether change in outcome measurements is clinically meaningful for the individuals who engage in therapy and will be addressed below.

Clinical significance methods of assessing outcome

Research that focuses on the treatment of mental health difficulties such as depression have increasingly emphasised the importance of demonstrating that interventions not only show a statistically reliable difference from a control condition, but also demonstrate that the intervention has had a 'real' impact on the individual (i.e., that the intervention has *clinical significance*) (McGlinchey, Zimmerman & Atkins, 2008). There are several disadvantages of relying on statistical significance tests to evaluate treatment efficacy. First of all, statistical significance tests provide no information about the variability of the response to treatment within the sample (within-group variation) (Jacobson & Truax, 1998). As information reported would be based on group means and variances, information cannot be gathered regarding an individual client (Ogles, Lunnen & Bonesteel, 2001). Secondly, by determining whether an intervention has had a statistically significant effect (i.e., disproved the null hypothesis) it tells us nothing about the size, importance or clinical significance of the effect. *Clinical significance* is typically regarded as the assessment of meaningful

change due to treatment (Beutler & Moleiro, 2001; Kazdin, 1999, 2001; McGlinchey et al., 2008; Sheldrick, Kendall & Heimberg, 2001). Clinical significance refers to whether the intervention makes a 'real' difference in 'everyday life' of the client, or to others with whom the client interacts (Kazdin, 1999). The concept of clinical significance came out of concern for social validity (i.e., that is whether the effects of an intervention were deemed as important by society) (Kazdin, 1977). Part of this question asks whether the effects of a treatment are clinically or practically important (Ogles, et al., 2001).

Clinical significance methods in outcome studies

There are several methods that have been developed to evaluate clinical significance. However, Kazdin (2001) makes the point that "much more work is needed to show the connection between the construct [clinical significance] and a specific measure used in research" (p.456). Characteristics of clinical significance include; (a) treated clients making statistically reliable improvement as a result of treatment; (b) treated clients are distinguishable empirically from 'normal' peers following treatment; or (c) a combination of return to functioning and reliable improvement (Ogles et al., 2001). One example of such a model is the *Jacobson-Truax Method* (JT method). Jacobson and Truax (1998) assert that clinical meaningfulness can be inferred if the post-treatment status of (*initially dysfunctional*) patients was more similar to *non-dysfunctional* than *dysfunctional* populations. The JT method contains two steps. The first step is to define a cut-off point that separates the *functional* and *dysfunctional* population. The second step compares individual's change from pre- to post-therapy to the standard error of measurement of the outcome (i.e., ± 1.96 SE), referred to as the Reliable Change Index (RCI). These two steps are then used to classify patients into one of four categories, 'recovered' (i.e., individual has passed cut-off A and RCI in the positive direction), 'improved' (i.e., individual has passed RCI in the positive direction but not cut-off A), 'unchanged' (i.e., individual has passed neither criterion), or 'deteriorated' (i.e., individual has passed RCI in the negative direction) (Atkins, Bedics, McGlinchey & Beauchaine, 2005). Clinical significance data are advantageous because they can provide rich information about the individual clients that are involved in treatment. This type of data allows statistical tests to provide information about within-group variation (Ogles et al., 2001).

Conceptual limitations of clinical significance methods

Although there are many operational definitions and methods of assessing 'clinically meaningful change' it does not mean that this is what these methods are actually measuring. Kazdin (2001) cautioned that it is not clear whether the methods that researchers use to measure *clinical significance* reflect any genuine differences in the everyday life of clients. Symptom change is primarily used as the main criterion for clinical significance (Kazdin, 1999). In Axis I disorders such as depression the patho-physiological mechanisms are not fully understood; this leaves researchers to rely on signs and symptoms of the disorder to determine whether remission has occurred (McGlinchey, et al., 2008). The DSM-IV-TR (APA, 2000) definition of remission from depression equates to absence of symptoms. However, the emphasis on symptom change may only reflect the perspective of the investigator rather than reflect what is actually important for the client from their own experience (Kazdin, 2001). Kazdin (2001) asserts out that the criterion used needs to match the goals of treatment, and if one construct is overburdened to represent important changes, we may rely on a construct that is not even important in many therapeutic situations. Domains other than symptom change may have a lot more importance when defining clinical significance that is meaningful to the client. It is argued that current methods of clinical significance do not go far enough, more important domains could possibly include quality of life (a factor commonly mentioned in the context of positive mental health), or impairment (McGlinchey et al., 2008). In contrast to symptom change, 'impairment' describes a client's ability to meet demands and obligations in everyday life, his/her ability to interact with others, and restrictions the individual may encounter when s/he is in particular situations, settings and activities in which s/he would normally participate (Kazdin, 2001). Kazdin (1999) asserts that it is worth distinguishing between actual change and perceived change, where actual change represents changes in symptoms or functioning as reflected on objective and standardised tests and perceived change is the perspective of the client or those with whom the client interacts with. It can be the difference between 'being in control' and 'feeling in control' (Kazdin, 1999). In addition, a measure or criterion for clinical significance should to some extent match the clinical problems and goals of treatment. These arguments remind us that symptom reduction may not be the only goal in treatment for the client. All presenting issues are different, just as people are

different. Psychological problems such as depression are conceptualised as being on a continuum of severity (Emmelkamp et al., 2010), therefore developing cut-off points to determine whether a client's change is meaningful can be arbitrary (Kazdin, 2001). More conceptual clarity is required regarding the construct of clinical significance and what constitutes a clinically meaningful difference in a client's life after therapy (Jensen, 2001; Kazdin, 1999; 2001; McGlinchey et al., 2008; McGlinchey, Atkins & Jacobson, 2002).

Methodological limitations of clinical significance methods

Several possible methodological difficulties have been identified in attempts to assess outcome using clinical significance methods. One concern is that clinical significance relies on the validity of the measures that are used to assess clinical change (Sheldrick, et al., 2001). Some instruments can be one-dimensional, whereas people in treatment typically present with multidimensional clinical problems. When clients enter treatment they do not always appear 'dysfunctional' on outcome measures. This can be because of measurement error, the lack of sensitivity to measures, or temporary fluctuations in symptoms (Ogles, et al., 2001). Another potential limitation of assessing clinical significance is the possibility of rater bias. In particular, some self report instruments may be too reactive to be used for judging clinical meaningfulness (Kazdin, 2001; Ogles et al., 2001). A third problem involves regression towards the mean whereby those clients who have high pre-treatment scores on an outcome measure may be more likely to make large improvements (Ogles, Lambert & Masters, 1996). If the measures used in psychotherapy outcome research are not psychometrically sound it limits the usefulness of any data they yield (Jacobson & Truax, 1998). Furthermore, the absence of normative data for 'functional' and 'dysfunctional' populations on many commonly used outcome measures will deter the development of standardised cut-off points (Jacobson & Truax, 1998). Another existing problem is that there is great variation in the application of the methods that examine clinical significance (Atkins et al., 2005; Follette & Callaghan, 2001; Sheldrick et al., 2001). Even when investigators use the same methods or similar populations of clients, the parameters and cut-off scores used in formulae can differ significantly (Ogles et al., 2001). As there is no uniformity in the process it questions the utility and meaningfulness of the

conclusions drawn from this kind of research, especially when comparing different studies (McGlinchey et al., 2008).

Conclusions

This section has highlighted two different methodological approaches; the *dose-effect relationship* and *clinical significance* which measure how clients' change over time within treatment. Howard and colleagues' (1986) original study on the dose-response relationship has given rise further research to address the question of 'how much therapy is enough?' Gains appear to be made early in therapy at a faster rate than those made later on in therapy. However, Howard and colleagues' (1986) original study had many methodological limitations. The outcome criteria were ill-defined and tended to lump a large heterogeneous sample together. This approach was based on the medical model, relied on pre- verses post-measures of outcome, and assumed that change is linear. However, research that measures change session-by-session has shown that this change is rarely linear (Kadern, et al., 1996; Tang & DeRubeis, 1999). This section then considered the advantages and disadvantages of clinical significance methods that determine whether a client's change within therapy makes a difference in his/her everyday life or for others with whom s/he interacts. However, the over-reliance on symptom change as the main criterion in clinical significance models questions whether these methods are actually capturing the construct of clinical significance and measuring what is actually important for clients. These models (e.g., Jacobson-Truax method) of clinical significance also rely on the psychometric properties of the measures they use, and assume that normative data are available for functional and dysfunctional populations, despite such norms being difficult to obtain for many assessment tools. Both the *dose-response model* and the *clinical significance* approach to outcome research have significant limitations in terms of 'meaningfulness' to the individual client and their ability to be sensitive enough to pick up individual differences and patterns in the therapy process as change occurs. In recent times, researchers have made use of longitudinal design methods to investigate important processes in therapy and potential mechanisms of change (Hayes, et al., 2007). These types of studies incorporate more frequent assessments, the study of individual trajectories over time, and the identification of discontinuities and nonlinear patterns of change (Hayes et al., 2007). Measuring improvement in psychotherapy session-by-session is a different way of assessing the pattern of

change and may be more clinically meaningful than other approaches (Tang & DeRubeis, 1999).

CHAPTER THREE

THE PHENOMENON OF SUDDEN GAINS IN COGNITIVE BEHAVIOURAL THERAPY FOR DEPRESSION

Introduction

This chapter discusses the emergence of the phenomenon of *sudden gains* in the cognitive-behavioural treatment outcome literature and discusses the importance of sudden gains. The exploration and modelling of change is of great importance to psychotherapy researchers. Two main research traditions have moved away from assuming that change is linear and steady; they are instead concerned with how change unfolds during treatment (Stulz, Lutz, Leach, Lucock & Barkham, 2007). The first is *client focused research,* which is concerned with the early identification of clients at risk for treatment failure, feedback to therapists and outcome management. The second, *process and outcome research,* which focuses on the relationships between psychotherapeutic processes and outcomes over the course of treatment (Orlinsky, Ronnestad & Willutzki, 2004). This approach has produced an abundance of process-outcome research that investigates generic change processes and specific therapeutic tasks (Stulz et al., 2007). One branch of this research is concerned with *sudden-gains* or major discontinuous changes that occur between sessions. These two types of research (i.e., 'client focussed research' and 'process outcome research') are not mutually exclusive. They are both detailed explorations of the change processes during treatment. They help to answer the question of whether there are different subgroups that follow certain trajectories of change and the client factors that may predict such patterns (Stulz et al., 2007).

Discontinuous change patterns

Different types of discontinuous change patterns have been identified to predict symptom improvement in CBT for depression. Illardi and Craighead (1994) identified an *early rapid response* pattern that was characterised by a substantial decrease in depression symptoms by week four of therapy, after which these changes level off. Thompson, Thompson and Gallagher-Thompson (1995) examined session by session changes in mood for patients receiving therapy for depression. They noted that although the average rate of change across patients was linear and steady, many patients had unsteady rates of change (i.e., increases or decreases in self-reported

depressive mood). They showed that those who had unsteady change also had the greatest rate of improvement (during treatment), indicating that individual session-by-session change is a significant factor in psychotherapy outcome research. Another phenomenon that has been researched is *transient worsening* (i.e., a client's symptom severity score significantly becoming worse from session to session) it has been found that these *depression spikes* have predicted lower post-treatment depression (Hayes et al., 2007a). Additionally, Hayes and colleagues (2007a) showed that clients' weekly diaries indicated that these *spikes* were associated with more cognitive-emotional processing during this period, or arousal, than those that did not experience the *spikes*. Early research in this area has also demonstrated that early positive response in psychotherapy predicted final treatment status and follow-up status. Haas and colleagues (2002) found that clients that were *rapid early responders* made up the bulk (80% at termination) of patients who made clinically significant gains. Therefore, discontinuous and non-linear change throughout therapy is significant and appears to be associated with better outcome for those clients who receive therapy.

The discovery of 'sudden gains'

Tang and DeRubeis (1999) discovered another pattern of change in early sessions of CBT for depression that many patients experience which they termed *sudden gains* This refers to a pattern whereby a client shows a large symptom improvement which occurs between two consecutive sessions, which does not reverse. Tang and colleagues (Tang & DeRubeis 1999; Tang, DeRubeis, Beberman & Pham, 2005) reported plots of individual time course data that suggest about 39-46% of clients will experience a sudden-gain and this pattern is discontinuous and does not suggest gradual or linear change. Several sudden-gain studies have confirmed that sudden-gainers average greater improvement across treatment than do non-sudden gainers (Davies, Leach, Lucock, Stiles, Iveson & Barkham, 2006; Greenfield, Gunthert & Haaga, 2011; Hardy, Cahill, Stiles, Ispan, Macaskill & Barkham, 2005; Kelly, Roberts & Ciesla, 2004; Stiles, Leach, Barkham, Lucock, Iveson, Shapiro, Iveson & Hardy, 2003; Tang & DeRubeis, 1999; Tang, et al., 2005). Using Tang and DeRubeis (1999) criteria for sudden-gains or adaptations thereof, researchers have found that sudden-gains are associated with: better outcomes at the end of treatment in supportive-expressive therapy (Tang, Luborsky & Andrusyna, 2002); systematic behavioural

family therapy (Gaynor, Weersing, Kolko, Birmaher, Heo & Brent, 2003); CBT for atypical and recurrent depression (Vittengl, Clark & Jarrett, 2005); behavioural activation therapy for depressed cancer patients (Hopko, Robertson & Carvalho, 2009); and in routine practice of varied theoretical approaches with a diverse client population in three clinics run by a large British National Health Service Trust (Stiles et al., 2003). Sudden gains are not restricted to depressed clients but can occur in the treatment of other disorders such as bulimia nervosa and alcohol abuse (Wilson, 1999); social phobia (Hofmann, Schultz, Meuret, Moscovitch & Suvak, 2006); posttraumatic stress disorder (Doane, Feeny & Zoellner, 2010); and generalised anxiety disorder (Present, Crits-Christoph, Gibbons, Hearon, Ring-Kurtz, Worley & Gallop, 2008). In sum, empirical evidence suggests that sudden gains occur in a range of therapies for a number of psychiatric difficulties and they are not random or clinically meaningless. Rather they are maintained over time and associated with better outcomes.

It has been reported that the majority of sudden gains occur in the early phases of treatment (Kelly et al., 2004). The suggestion that a client's progress in therapy is neither gradual, nor smooth as indicated by mean dose-effect curves, but instead marked by sharp discontinuities is an important change in our expectancies of outcome patterns in CBT for depression (Stiles et al., 2003). Previous research (see below), has found that sudden gains that occur in the early sessions of therapy are related to better outcome than those that occur in later sessions. Stiles and colleagues (2003) claimed that sudden gains that occurred before session 16 were associated with better outcomes than those that occurred after session 16. Kelly and colleagues (2004) reported that those who experienced sudden-gains in the first third of treatment and not later showed greater reductions in depressive symptoms than those who experienced sudden-gains later in treatment. Similarly, Busch, Kanter, Landes and Kohlenberg (2006) found that those clients who had experienced early sudden gains (i.e., within the first 10 sessions of therapy) compared to those who experienced sudden gains after session 10 had lower scores on the *Beck Depression Inventory – Second Edition* (BDI-II; Beck, Steer & Brown, 1996) at the end of treatment. These findings highlight the significance of sudden-gains that occur early in treatment. The recognition of sudden gains is useful as they have the potential pin-point the few therapy sessions that may have critical importance in therapeutic

outcome. Furthermore, those researching change mechanisms in therapy can more adequately concentrate their resources for detailed analyses of these sessions (Tang et al., 2005), thus facilitating a new way of researching psychotherapy outcomes.

Possible mechanisms behind 'sudden gains'

As noted above, there is evidence that sudden gains are related to treatment outcome (e.g., Tang & DeRubeis, 1999). However, there has been some debate about the mechanisms of change behind sudden-gains in CBT for depression. What happens between the pre-gain session and the sudden-gain session that helps to explain why clients experience sudden gains? One hypothesis is that symptom severity is normally volatile and that this does not mean anything for long term outcomes. Additionally, there is a question of whether sudden-gains are actually attributable to factors inside of therapy? Could it be positive or negative life events outside of therapy that are causing these sudden-gains to occur? Research into the interaction between sudden gains and significant life events outside of therapy has concluded that there is no connection between the two (Hardy et al., 2005; Manning, Hardy & Kellett, 2010). These findings indicate that other processes and techniques within the therapy may account for the occurrence of these sudden gains. In a review of rapid response literature, Illardi and Craighead (1994) concluded that 60 to 80% of symptom reduction occurs in the first four weeks of cognitive therapy. It is argued that because rapid response to treatment occurs in the early weeks of therapy that the cause of these improvements is more likely to be due to non-specific therapeutic factors (e.g., therapy rationale, therapeutic alliance and assignment of homework) rather than specific cognitive techniques, because these are administered later on in treatment (Illardi & Craighead, 1994). It is argued that early response might just indicate a client's readiness to change and a response to common factors rather than specific interventions (Lambert, 2005). This conclusion has been challenged by Tang and DeRubeis (1999), who point out that first of all that eight sessions of CBT are usually administered in the first four weeks of therapy, and secondly they argue that cognitive techniques are used and administered as early as session two in therapy. They also suggested that Illardi and Craighead's (1994) analysis of the group Beck Depression Inventory scores (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) over the time course was not appropriate because this method obscured significant session-by-session symptom reductions. Illardi and Craighead's (1994)

methodological approach analysed average symptom time courses. Tang and DeRubeis (1999) argue that the validity of this approach depends on the homogeneity of the patients' time course pattern. Therefore, if each individual's time course pattern is different, the group mean time course would be misleading. Tang and DeRubeis (1999) assert that it is more important to measure time course data for every individual client as this approach can identify sessions that precede significant change. The sudden-gains approach to measuring individual patients' change over time draws upon arguments of functional analysis research which has long argued that the best way to detect underlying causal relationships is to carry out detailed analysis of a participant's change over time (Barlow & Herson, 1984).

There is also some debate about the types of therapeutic mechanisms that may be behind early rapid change in therapy, namely are they behavioural or cognitive? This question will be addressed later on. Several studies have provided some clues about the mechanisms behind sudden gains (see Chapter Four). Research carried out by Goodridge and Hardy (2009), using qualitative methodology, explored the relationship between the client's insight and understanding of their problems and the occurrence of sudden gains using the *assimilation model*. The study found that at the time a client was experiencing a sudden gain there was also corresponding positive affect and a high level of cognitive involvement. Therefore, this study generally supports Tang and DeRubeis (1999) findings of high cognitive activity in the pre-gain session (i.e., the session before the sudden gain). However, Goodridge and Hardy's (2009) findings and analyses also showed a more 'incremental' pattern of cognitive change and insight, with a high level of insight and cognitive activity found in the after-gain session. This suggests that the processes of change within psychotherapy may be more gradual, as clients were more likely to develop full insight after the gain. Strunk, Brotman and DeRubeis (2010) explored the 'predictors' of early inter-session symptom gains within CBT for depression. They focussed on process variables such as, therapeutic alliance, therapist adherence to CT techniques and the client's facilitation/inhibition of these techniques. Two important elements of therapists' adherence emerged as the strongest predictors of symptom improvement i.e., the use of cognitive methods and the therapist's ability to negotiate the content of the session with the client and/or structure the session (Strunk et al., 2010). Additionally, client facilitation/inhibition of therapist

adherence also predicted subsequent symptom change. However, alliance scores did not predict early symptom change, rather high alliance scores were predicted by early symptom change (Strunk et al., 2010). The findings from this study suggested that a therapist's adherence to CBT techniques and structure in therapy and the client's facilitation of the therapist's adherence subsequently predict early change in therapy. These researchers also highlight the reciprocal relationship between symptom change and therapeutic alliance. At present, research into the active mechanisms behind early treatment response and long-term outcomes is growing but is in the preliminary stages and would warrant further investigation.

Pre-treatment client predictors of sudden gains

In psychotherapy one of the critical questions in the research field is; "what treatment, by whom is most effective for a particular individual with a specific problem, and under which set of circumstances?" (Jarrett, Eaves, Grannermann & Rush, 1991; Paul, 1967). Therefore it is relevant to ask which clients, which treatment, and under which set of circumstances will a person most likely experience an early sudden gain? Although much research has been produced recently that shows sudden gains do occur in the treatment of CBT for depression and also in non-cognitive treatments (Kelly et al., 2004), there appears to be some gaps in the research produced that accounts for the predictors of early sudden gains in CBT for depression. Tang and DeRubeis (1999) assert that pre-gain sessions and control sessions in their sudden gains research did not differ significantly on therapist adherence to CBT techniques, therapeutic alliance, facilitative conditions or therapeutic competence. Additionally predictor variables such as education level, gender, number of previous depressive episodes or melancholic subtypes were also not associated with initial BDI-II scores (Kelly et al., 2004). Furthermore Hardy et al. (2005) found that the sudden gains that were experienced were not associated with the patient's marital status, gender, first session alliance, or the amount of good or bad life events a person has experienced. There were no significant differences found between sudden gainers or non-sudden gainers on personality subscales (see Hardy et al., 2005).

Possible client factors that may predict the occurrence of sudden gains

There has been relatively little attention paid to the factors and characteristics of the client that predict outcome in cognitive therapy of depression (Hamilton & Dobson, 2002). Information gained from investigating client variables that influence outcome would ultimately help facilitate treatment planning and therefore increase treatment efficiency. Pre-treatment symptom severity and diagnostic profile are variables that are likely to influence the course and outcome of CBT for depression and therefore may inform the clinician about whether the client is likely to experience an early sudden gain. The relationship between pre-treatment severity of depression and outcome in cognitive therapy is complicated (Hamilton & Dobson, 2002). Shapiro, Barkham, Rees, Hardy, Reynolds & Startup (1994) discovered a significant interaction between pre-treatment severity and therapy duration. Those with high pre-treatment severity scores (BDI scores above 26) showed a significantly greater amount of improvement in a 16 session treatment condition relative to those in an eight-session condition. Therefore, those with more severe depression benefited from a longer term therapy. Hamilton and Dobson (2002) emphasise that because more severely depressed individuals respond differently to cognitive therapy for depression that their less depressed counterparts, severity is an important factor to control for when examining outcome.

Another factor that is likely to affect outcome in manualised cognitive-behavioural treatment for depression is the complexity of the client's diagnostic profile. Co-morbid depression with other Axis One and Two disorders are likely to be associated with increased symptom severity (Gelhart & King, 2001; Laberge, Gauthier, Cote & Plamondon, 1993) and reduced functioning, therefore treatment of patients with co-morbid disorders is likely to be more challenging (Singer, Dobson & Dozois, 2008). For example, those with major depressive disorder and dysthymic disorder, with co-morbid anxiety were found to be more likely to have worse outcomes than those who were not anxious (Gelhart & King, 2001). Overall the client's status before treatment is likely to influence how they will respond to treatment and therefore, may affect the probability that they may experience a sudden gain within CBT for depression.

Conclusion

The mechanisms that dictate the presence of sudden gains within therapy for individual clients remain unclear. However, they are nevertheless of great critical importance in terms of both safety and effectiveness. Previous research in the area of sudden-gains and CBT for depression have shown that they are not random or clinically insignificant, but rather that they are associated with better overall outcomes in the long term (Hardy, et al., 2005; Kelly et al., 2004; Tang & DeRubeis, 1999; Tang et al., 2005; Tang et al., 2007). Investigating sudden gains in CBT for depression is useful because sudden-gains may pin-point the few therapy sessions that may have critical importance. With the emergence of brief intervention models it is useful to identify who will most benefit from short-term treatment. There appears to be some gaps in this area of research that accounts for the client factors that predict sudden gains in CBT for depression. Identifying clients that are more likely to experience sudden-gains in early session of CBT for depression will help to inform clinicians who will most benefit (and maintain these benefits) from short-term CBT. The rationale behind measuring sudden gains that occur in the early sessions of CBT is guided by the research in this field that emphasises that gains made in the first half of treatment are associated with more positive outcomes (Busch, et al., 2006; Kelly et al., 2004; Manning et al., 2010). Therefore, further investigation into the client factors that may predict those clients who may experience an early sudden gain and the potential mechanisms behind these gains are of great significance.

CHAPTER FOUR

WHAT ARE THE POSSIBLE MECHANISMS BEHIND SUDDEN CHANGE IN THERAPY?

Introduction to 'cognitive change' in cognitive behavioural therapy for depression

The central principle of Cognitive Behavioural Theory (CBT) is that the alleviation of depression is linked to the mediation of cognitive processes (Garratt, Ingram, Rand & Sawalaui, 2007). It is assumed that changes in cognition are associated with improvement of depressive symptomatology. Cognitive Behavioural Therapy can be effective in reducing depressive relapse. However, the processes and mechanisms though which symptomatic improvement in CBT for depression is achieved are not well understood. Modifying cognitions that are 'dysfunctional' (e.g., negative automatic thoughts) is seen as an essential ingredient to the efficacy of Cognitive Therapy (CT) both in the short-term and the long-term (Jarrett, Vittengl, Doyle & Clark, 2007). The meaning that depressed people assign to their experiences is said to influence their affect and the course of their depression (Clark, Beck & Alford, 1999). Cognitions, within the cognitive theory of depression, include both self talk (e.g., thoughts and attitudes) and images. These are also known as *cognitive products* (Clark et al., 1999). These cognitive products can span many domains (e.g., attributional style, automatic thoughts, dysfunctional attitudes and cognitive distortions). These, and similar constructs, are the focus of many outcome and validity studies. These variables can be used as indicators of possible changes in an individual's functioning schema, which are useful as schema change cannot be directly observed (Garratt et al., 2007). Consequently, cognitive change can be measured by assessing a variety of constructs such as attributional style, dysfunctional beliefs and the like. This chapter aims to introduce the theory behind cognitive change (i.e., improvement) in depression and different available methods of measuring cognitive change within therapy. It also considers the temporal relationship between symptom change and changes in cognition. Subsequently, the phenomena of sudden gains are discussed (see Chapter Three) and the possible mechanisms that may account for sudden gains within therapy are reflected upon.

Attributional style and the hopelessness model of depression

One of the elements or constructs representing an individual's style of thinking within depression is *attributional style* or *explanatory style* (DeRubeis & Hollon, 1995). Attributional style is based on the *hopelessness theory of depression* (Abramson et al., 1989). The hopelessness theory of depression describes *hopelessness depression* as a subtype of depression and captures two core elements as contributing factors; (a) the tendency to have negative expectations about the occurrence of highly valued outcomes (negative outcome expectancy), and (b) the expectation that one will be helpless to change negative outcomes (a helpless expectancy). Therefore, *hopelessness depression* is regarded as being influenced by an individual's expectations that bad things will happen to them and that s/he will be helpless to change the negative consequences of these events. According to the hopelessness theory there are three main attributions that people make that modulate whether they will become hopeless, and in turn develop the symptoms of hopelessness depression when encountered with negative life events: 1) they make inferences about why the event occurred; 2) they make inferences about the consequences that will result from the event; and 3) they make inferences about the self given that the event occurred (Abramson et al., 1989). There is a significant body of empirical research (e.g., Abramson et al., 1989; Robins & Hayes, 1995) that links a negative attributional style to depressive symptoms. It is hypothesised that some individuals will tend to attribute negative events to stable and global factors and, in turn, view these events as very important (also known as a *pessimistic explanatory style*; see Gillham, Shatte, Reivich & Selgiman, 2001), whereas other individuals may not. They in turn are more likely to become hopeless and consequently develop the symptoms of hopelessness depression (Abramson, et al., 1989). Abramson and colleagues (1989) use the term *hypothesised depressogenic attributional style* to describe this tendency. Attributional style and depression are understood within a distress-diathesis model which recognises that cognitive styles and negative life events are not dichotomies and both work on a continuum. The continuum view suggests a titration model where the less negative a person's cognitive style is, the more stressful a life event has to be in order to interact with that cognitive style and contribute to the formation of depressive symptoms (Abramson et al., 1989). The *hopelessness theory* model intends to function as an organising rationale for the source of predictions about therapeutic

interventions and propose a hypothesised etiological chain to *hopelessness depression*. Abramson and colleagues (1989) indicate that each link of this chain demonstrates a point for intervention. To treat current episodes of depression, it is suggested that therapeutic strategies be employed aimed at undermining hopelessness and restoring hopefulness (Abramson et al., 1989). The hopelessness theory suggests that by introducing therapeutic strategies that target hopelessness depressive symptoms it is hypothesised that these symptoms will reduce and thus decrease the likelihood of relapse. Therefore, the *hopelessness theory* of depression suggests that cognitive style may bring about a change an amelioration of depressive symptoms.

Attributional style and cognitive change

Explanatory style or attributional style as measured by the *Attributional Style Questionnaire* (ASQ; Peterson, Semmel, von Baeyer, Abramson, Metalsky & Selgiman, 1982) plays a significant role in the mechanisms of change in cognitive therapy (Barber, Abrams, Connolly-Gibbons, Crits-Christoph, Barrett, Rynn & Siqueland, 2005; DeRubeis, Evans, Hollon, Garvey, Grove & Tuason, 1990; Jarrett, et al., 2007; Selgiman, Castellon, Cacciola, Schulman, Luborsky, Ollove, & Downing, 1988). The ASQ was developed to measure attributional style, based on Abramson and colleagues (1978) theory of helplessness depression. The ASQ is a self report measure that allows researchers and clinicians to gain knowledge about their clients' cognitions and also measure outcomes over the course of treatment. When filling out the ASQ, an individual is presented with 12 hypothetical situations, six of these negative and six positive. Participants are then asked to generate a cause for each of these situations and rate, on a scale of one to seven, the extent to which the cause reflects internal, global and stable factors. The internal, global and stable rating for each negative situation (18 total) can be averaged and form a composite index of *failure* and *success* (i.e., *negative* and *positive*) attributions relative to depression (Peterson et al., 1982). Initial psychometric properties of the ASQ have been found to be satisfactory, and the expected dimensions have been correlated with depressive symptoms. Cronbach's alphas for the positive and negative composite scores are 0.75 and 0.72, respectively (Peterson et al., 1982). However, the internal reliability of the single dimensions were not ideal; they ranged from 0.44 to 0.69 with an average of 0.54. Although the psychometric properties of the ASQ are sound, and research (DeRubeis et al., 1990; DeRubeis & Hollon, 1995; Jarrett et al., 2007; Selgiman et al.,

1988) demonstrates that attributional style can change over the course of CBT for depression, some caution should be exercised when using these types of measures to track cognitive change.

It is important to recognise/acknowledge that changes in cognition can involve not only *what* people think but more importantly *the way* people think or analyse information. The cognitive mechanisms that underlie depressive biases have been under-researched for a variety of reasons. In most of the research to date, the measures (e.g., ASQ, Peterson et al., 1982; Dysfunctional Attitudes Scale: DAS, Beck, Brown, Steer & Weissman, 1991; Automatic Thoughts Questionnaire: ATQ, Hollon & Kendall, 1980) that are used reflect the content of people's thoughts rather than the actual processes or mechanisms of thinking. The cognitive theory of depression proposes that negative thoughts and cognitions interact with changes in affect (i.e., a negative schema is most likely to be activated when people experience shifts in emotion). A limitation of using these measures in research is that it is hard to know if an endorsement made by an individual on a self-report questionnaire would be similar to the moment-to-moment thinking that occurs before, simultaneous with, and after changes in affect or mood (Jarrett, et al., 2007). For example, participants may fill out cognitive measures when they are in a good mood and their *dysfunctional schemata* may be deactivated at this time. This could question the external validity of such measures. Alternative techniques, such as *mood induction* (i.e., prompting a sad emotion as part of the research design) may be useful for comparing changes in cognitions within CBT (Barber & DeRubeis, 2001). Segal, Gemar, and Williams' (1999) study claimed that depressive patients who received cognitive therapy, compared to those receiving pharmacotherapy, experienced fewer *dysfunctional attitudes* after receiving sad mood induction. Overall, empirical research lags behind theoretical speculation when it comes to understanding how schemata are structured, activated and deactivated and also how these cognitive structures are related to maladaptive or biased information processing (Dozois & Dobson, 2001). As schema change cannot be directly observed cognitive change measures are useful as they do provide some indication of cognitive change. The ASQ has been used in process outcome research and findings have demonstrated that explanatory or attributional style does change over the course of therapy and also has a relationship

with symptom change (Barber & DeRubeis, 2001; Gotlib, Lewinsohn, Seeley, Rhode & Redner, 1993).

Cognitive change and symptom change

A study by Jarrett and colleagues (2007) examined how changes in cognitive content and changes in depressive symptoms were related during CBT for depression. Participants (N = 155) met criteria for recurrent major depressive disorder with clear inter-episode recovery, 56.8% of the participants had been treated with pharmacotherapy and 59.4% with psychotherapy. The study used psychometric instruments such as the ASQ and the DAS to measure changes in cognition over the course of therapy. It was found that at pre-treatment there was no difference between those who responded to therapy and those who did not respond to therapy in terms of their cognitive content. However, by session 17 those who had responded to therapy had lower DAS scores and less *depressogenic* ASQ scores than non-responders. Pre-treatment cognitive content did not predict response to cognitive therapy. So improvement in cognitive content observed during the middle of treatment may reflect a developing response to cognitive therapy. Overall, it was found that patients who showed improvement in their cognitive content also tended to show improvement in depressive symptoms (Jarrett et al., 2007). In this particular study (Jarrett et al., 2007), regression analysis showed that reductions in depressive symptoms accounted for changes in cognitive content, rather than the other way around. Neither early change in depressive symptoms nor early change in cognition predicted the change in the other (Jarrett et al., 2007). The pattern of correlates suggested change in negative cognitive content parallels but does not account for, or predict, changes in depressive symptoms. The results from their study suggest that if the primacy hypothesis[2] is correct, then it would appear that the effects of cognitive content on depressive symptoms do not occur on a timescale of weeks or months as has been assessed in this and other studies. It is rather suggested by Jarrett and colleagues (2007) that they may occur on a more 'micro' timescale of days or perhaps moments. Therefore, measuring depressive symptoms and cognitions on a session-by-session basis may capture more accurately the temporal relationship between

[2] The 'primacy hypothesis' asserts that negative cognitive products, such as core beliefs/schema are a core feature of the depressive experience and they have a significant influence on other depressive symptoms (Clark, Beck & Alford, 1999)

symptom change and cognitive change. It also suggests that it may be within the session-to-session timeframe that these cognitive and depressive shifts are being made.

Sudden gains and cognitive change

The mechanisms that help to explain cognitive change within therapy remains unclear. There is still a question of whether the benefits of CBT are a consequence of the amelioration of the causal processes that generate risk or the development of compensatory strategies that will offset them (Scott & Freeman, 2010). There are two competing models that attempt to explain the process of change in CBT; the *schema change model* and the *compensatory skills model* (Persons, 1993). These two models differ in their predictions about the timing of schema change and their statements about the generality of the lessons learned in CBT. The *compensatory skills model* proposes that cognitive therapy helps clients by teaching them cognitive or behavioural skills for use when they experience negative emotional experiences (Barber & DeRubeis, 2001; Baron, Baron, Barber & Nolen Hoeksema, 1990). According to the compensatory skills model, the timing of schema change occurs much later in therapy (even after therapy has finished) following the repeated application of compensatory skills (Persons, 1993). The model also proposes that patients learn general skills in therapy (e.g., the ability to examine available evidence to test particular cognitions within a difficult situation). There are several studies (e.g., Fresco, et al., 2007; Teasdale, et al., 2000) that have indicated that changes in the processing of depression related information may be an important mechanism behind positive change in the treatment of depression. Teasdale and colleague's (2000) study suggested that CBT helps clients acquire meta-cognitive skills that in turn prevent depressive relapse. They reported that CBT was found to modify the form of thinking in chronic depression and produce an overall reduction in *all-or-nothing* thinking style that in turn mediated the relapse prevention effect (Teasdale et al., 2000). Similarly, Fresco and colleagues (2007) found that CBT responders showed greater gains in '*de-centring*'[3] compared with anti-depressant responders and additionally, that higher levels of de-centring post-relapse was associated with lower relapse rates at 18 months.

[3] De-centring is an individual's ability to observe his/her thoughts and feeling as transitory events in the mind that do not necessarily reflect reality (Sauer & Baer, 2010).

In contrast, the *schema change model* (see Persons, 1993) proposes that CBT helps clients by teaching them their central underlying attitudes or schemata are *distorted* and/or *maladaptive*. According to the schema change model, schema change will occur during CBT, even within short-term therapy. This model also suggests that patients learn new beliefs that are highly specific to their particular underlying cognitive vulnerabilities (e.g., "I am not as worthless as I thought" would be an example of a new schema that may develop during CBT). Although these are both competing models, it is important to point out that the two are not necessarily mutually exclusive and they could both apply. Indeed, both may be occurring during the course of therapy for a particular client (Persons, 1993).

There are also similar models that purport to explain schema change (see Garrett, et al., 2007). There is an *accommodation model of change* where it is proposed that depressogenic schema can be changed in fundamental ways (Hollon, Evans & DeRubeis, 1990). An alternative model would be the *activation-deactivation model of change* which suggests that rather than profoundly changing the nature of cognitive structures, the depressive structures stay intact but become deactivated over the course of treatment. These models of proposed cognitive change are important when considering the mechanisms behind sudden gains. The *primacy hypothesis* (Clark et al., 1999) suggests that negative cognition and biased information processing have a significant influence on the symptoms of depression. The assertion is derived from the cognitive theory of depression (Beck, 1964). If the primacy hypothesis were true, then cognitive change would precede symptom change in early sudden gains. Therefore, one would predict that the schema change model would explain a rapid change in cognitions as there would have been a fundamental shift in schema. However, there is much debate about making this conclusion. It is argued that the very fact that early sudden gains exist is evidence that cognitive change is not influenced by specific therapeutic ingredients but rather common factors across all therapies (Illardi & Craighead, 1994; Lambert, 2005; Longmore & Worrell, 2007). Additionally, it is argued that cognitive change is no more influenced by *cognitive therapeutic techniques* than *behavioural activation techniques* (Jacobson, Dobson, Truax, Addis, Koerner, Gollan, Gortner, & Prince, 1996). Overall, the question of "what are the mechanisms behind early sudden gains?" is still largely unanswered by empirical research.

Common factors versus specific effects: What accounts for the symptom change that is experienced with sudden gains?

There are two fundamental assumptions that underlie the proposed mechanisms of cognitive therapy (Whishman, 1993). The first assumption is that cognitive change must co-vary with symptom reduction. The second suggests that cognitive change is specific to cognitive interventions. Therefore, the central rationale of CBT is that cognitive changes alleviate depression and cognitive change is due to specific cognitive interventions used within therapy. Wampold (2001) conducted a meta-analysis of component studies that appeared in the literature between 1970 and 1998. The effect size between the outcomes of treatment, with the *active component* included, and, without the active component included was calculated. The aggregate effect size across the 27 studies was -.20, indicating a trend in favour of the treatment without the component, but which was not statistically significant from zero. From this, it is argued that *specific ingredients* in therapy are less important than *common factors* in terms of outcome in therapy. Messer and Wampold (2002) argue that the purpose of specific ingredients is to construct a coherent treatment that therapists believe in, and provide a convincing rationale to clients. They emphasise that specific ingredients cannot be studied independently from the healing context in which they occur. Wampold (2001) suggested that common factors accounted for nine times more variability in outcome than specific ingredients, while his meta-analysis estimated that specific therapeutic effects accounted for only 8% of the variance. On the other hand, Tang et al., (2005) found in their study that sudden gains were immediately preceded by substantial within session cognitive gains, therefore suggesting that there is a greater cognitive change in the pre-gain session compared to the control session. Therefore, this indicates that therapeutic techniques that target change in cognition may play a role in sudden gains within therapy. In one sudden gains study it was reported that participants who had early sudden gains (within the first 3-4 sessions) had not received instructions in cognitive techniques prior to these gains (Kelly et al., 2004). '*Common factors'* is a term that recognises that many therapies have active ingredients, that are not unique to a particular therapeutic modality (e.g., therapist confidence, therapeutic relationship) but are none-the-less efficacious (Lambert, 2005). Lambert (2005) argues that it is common factors rather than specific therapeutic ingredients that produce rapid early response in therapy.

He asserts that whatever the mechanisms behind rapid early response to therapy are, they occur quickly, questioning whether specific cognitive ingredients are able to be practically applied so early on in therapy. Ilardi and Craighead (1994) also observed that 60-70% of symptom improvement in CBT occurs in the first four weeks and they assume that cognitive modification techniques are not extensively utilised within the early sessions of therapy. However, Tang and DeRubeis (1999) argue this point, acknowledging that the first four weeks of CBT treatment have two sessions per week in the first four weeks. Therefore up to 40-60% of the CBT sessions are carried out in arguably enough time for cognitive techniques to impact symptoms. DeRubeis et al., (1990) examined the temporal pattern of change in cognition and depressive symptomatology in the context of a randomised trial in which patients received either cognitive therapy (with or without imipramine pharmacotherapy) or did not receive cognitive therapy (but received imipramine pharmactotherapy). Several different measures of depression-relevant cognition (including the ATQ, ASQ and the DAS) and symptomotology were obtained before and after treatment and at the midpoint allowing for analyses of the effects of early changes in cognition and depressive symptoms. Findings from this study suggested that these sets of cognitive structures do play a meditational role in cognitive therapy. However, the role they play is not a causally sufficient one, because the relation of change on these variables to subsequent symptom change was not found in pharmacotherapy.

Cognitive verses behavioural techniques that account for symptom change in sudden gains

Within the *medical model* (e.g., see Wampold, 2001), component studies are considered to be a way of isolating the specific ingredients that are critical to the success of psychotherapy (Messer & Wampold, 2002). Jacobson and colleagues (1996) conducted a study in which a component analysis for CBT for depression was carried out to find that, surprisingly, they reported that *behavioural activation* alone is equal in efficacy to the *cognitive component* in the therapy. These findings seem to run contrary to the primacy hypothesis generated by the cognitive model of depression (Jacobson et al., 1996). Dimidjian, Dobson, Hollon, Schmaling, Kohlenberg, Addis, Gallop, McGlinchey, Markley, Gollan and colleagues (2006) found that, when treating severe depression, behavioural activation is comparable in efficacy to anti-depressant medication, and it significantly outperformed the cognitive component

alone. These results underscore the value of sustained use of behavioural strategies such as goal setting, self-monitoring, activity scheduling and the like (Dimidjian et al., 2006). In regards to early sudden gains, Kelly, Roberts and Bottonari's (2004) study also questioned Tang and DeRubeis' (1999) suggestion that cognitive mechanisms are facilitating early sudden gains, as they found that there was no significant change in self-esteem indicating that sudden gains were not preceded with improvements in thoughts about the self. However, this conclusion itself has its own limitations as it relies on measures of self-esteem rather than more specific indices of cognitive style (e.g., attributional style or amount of dysfunctional attitudes). Additionally, Hollon (2000) criticizes the findings of Jacobson and colleague's (1996) component analysis study. He points out that cognitive therapy typically integrates behavioural and cognitive strategies in an ongoing and interactive fashion throughout therapy. Furthermore, in most instances cognitive therapists will introduce behaviour change strategies in the context of testing specific beliefs, and encourage clients to use behavioural strategies to examine the accuracy of their underlying beliefs and attitudes. DeRubeis and colleagues (1990) also acknowledge that trying to identify mediating variables of symptom reduction in cognitive therapy for depression is difficult, as depressive symptoms, and the mechanisms of effective treatments, are integrally intertwined or reciprocally caused.

Conclusion

As discussed in Chapter One, CBT has been shown to be an effective treatment for depression. What is less clear is why or how it works. The move towards measuring more time points across therapy (rather than just pre- / post- measurements) has led to the discovery of several phenomena of discontinuous change patterns over time. One of these patterns of change is *sudden gains*. As covered in Chapter Three, sudden gains are of great significance for individual clients as they are associated with better outcomes and a lower chance of relapse. There is some evidence to suggest that change in cognition coincides with symptom change in sudden gains (Goodridge & Hardy, 2009; Tang & DeRubeis, 1999; Tang et al., 2005). However, this evidence is limited. This chapter has acknowledged debates in the literature regarding the question of what accounts for change in CBT. The first argument concerns whether specific effects and techniques in therapy are less important than the common elements across all therapies (see Wampold, 2001). Component studies have also

questioned the value of specific cognitive techniques (see Jacobson et al., 1996). However, these lines of research have their limitations. Preliminary research that has looked into the mechanisms behind early sudden change in therapy has supported cognitive change as a potential mechanism behind the presence of sudden gains in therapy (Tang & DeRubeis, 1999; Tang et al., 2005). This chapter has aimed to highlight the importance and value of further investigating the mechanisms behind early sudden change in therapy.

CHAPTER FIVE

THE ROLE OF HOMEWORK IN COGNITIVE BEHAVIOURAL THERAPY

Introduction

It is suggested that the direction of further research focus on the mechanisms behind the change in sudden gains for cognitive therapy. One area that has not been addressed in the sudden gains research is the relationship between sudden gains and homework. Homework assignments allow clients to make use of time between therapy sessions by completing activities that are targeted towards the agreed upon goals for therapy (Kazantzis & Ronan, 2006). Homework is a critical component of CBT. In their depression treatment manual, Beck and colleagues (1979) posit that homework is a type of trial in which clients' gains new insights about their thoughts, emotions, and behaviours that lead to negative thinking that can trigger and maintain their depression. Not only can homework help clients to get better and stay better, but it may also help the client to implement solutions to problems, increase self awareness, practice cognitive, behavioural and emotional skills, reinforcing what was learned in session, testing ideas and preventing relapse (Beck & Tompkins, 2007). As a process variable of therapy, homework may account for some of the variance that helps to explain why some clients experience rapid change from session to session.

The efficacy of homework

Data from correlational research studies in this area have demonstrated that psychotherapy that incorporates homework tasks yields better treatment outcomes than psychotherapy without homework (Kazantzis & Lampropoulus, 2002). Kazantzis, Deane and Ronan (2000) carried out a meta-analysis (27 studies, N = 1702) which aimed to examine the effects of homework assignments on treatment outcome and the relationship between homework compliance and therapy outcome. They reported that homework had a positive and significant effect on therapeutic outcome (weighted mean effect size of .36). Additionally, Kazantzis and colleague's (2000) meta-analysis found that those groups that showed high levels of homework compliance also demonstrated improvements in their therapy (weighted average correlation, $r = .22$).

Homework: Quality and quantity

One major question in research carried out in this area is whether compliance with homework leads to reductions in symptom severity. When considering the question of compliance it is important to remember that not all clients carry out the homework that they are assigned to do at the same standard. Focussing on more than just *how much* homework (quantity) is carried out is important. Theory dictates that what a client has learnt from carrying out the homework task (quality) is also vital in shifting beliefs and behaviour and therefore symptom improvement (Beck & Tomkins, 2007). Schmidt and Woolaway-Bickel (2000) examined the effects of homework compliance on outcome in CBT for panic disorder. They found that therapist and independent raters' estimates of the quality of participants' work, relative the quantity (amount) of the work, were better predictors of outcome. It is suggested that recovery is facilitated by engagement in activities that provide information that is at odds with pre-existing beliefs (Schmidt & Woolaway-Bickel, 2000). It is hypothesised that the more an individual engages with a task, the higher the level of emotional processing, and consequently the greater the shift in unhelpful existing beliefs.

Homework as a skill: An explanation for long term change?

Homework can be seen as a way for a client to gain adaptive skills that will contribute towards his/her improvement within therapy. A *compensatory skills model* (Badigo, Halperin & Barber, 1999) in cognitive therapy demonstrates that depressive symptomotology is reduced as a result of the client's utilisation of a set of skills which are learned throughout therapy. According to this model cognitive therapy for depression will not change a client's tendency to generate automatic negative thoughts, but rather provides the client with a set of adaptive (compensatory) skills that help the individual to deal with these thoughts when they arise (Badigo et al., 1999). The skills that are acquired can be behavioural and/or cognitive. For example, meta-cognitive skills can include the ability to generate accounts or explanations for events other than automatic depressive thoughts (Badigo et al., 1999). According to this model, long term change comes about when the client confronts challenging and stressful life events (in the future/post-therapy) using the skills gained in therapy, protecting themselves from the emergence of depression

(Badigo et al., 1999). It is proposed that over time these skills will become more automatic and eventually lead to a change in the clients underlying schemata (Badigo et al., 1999). Niemeyer and Feilxas (1990) found that clients who showed greater skill in using thought records were more likely to maintain gains in self-rated depression at six month follow-up.

The importance of cognitions and beliefs in homework

The process of homework directly involves the patient's belief system. It is theorised that when clients engage in homework tasks, their sense of mastery and progress towards a goal helps to facilitate a reduction in unhelpful thinking and therefore lasting positive cognitive change (Kazantzis & Daniel, 2009). Cognitive theory suggests that the effects of behavioural antecedents and consequences are moderated by cognitions. In cognitive therapy it is important to work with clients to identify beliefs which help both the client and therapist understand how the client's depression developed and how it is maintained (Garland & Scott, 2005). These beliefs in turn shape the client's experiences within, and out of, the therapy session. Homework effectively serves as a bridge between the client's experiences out of therapy and within therapy. As such, homework offers the perfect opportunity to work on understanding and modifying the impact of these beliefs (Garland & Scott, 2005). The active process of exploring the client's thoughts, emotions and behaviours around the completion (or non-completion) of assigned homework can be one of the most effective ways of accessing conditional and unconditional beliefs that may play a role in the development and/or maintenance of the client's difficulties/depression (Garland & Scott, 2005). Within the process of assigning and designing homework within therapy the client is likely to form several beliefs, including: beliefs about the task, beliefs about the tasks relevance to their goals in therapy, beliefs about its consistency with their understanding of the problem, and integration with their own coping strategies (Kazantzis & L'Abate, 2005).

There has been some research (e.g., Addis & Jacobson, 2000; Fennell & Teasdale, 1987) in this area to support the notion that compliance with homework may reflect a change in cognitions/beliefs which in turn affect symptom severity. It has been demonstrated that clients who accept the CBT treatment rationale are more likely to

have successful outcomes (Addis & Jacobson, 1996; Fennell & Teasdale, 1987). Addis and Jacobson (2000) found that acceptance of the treatment rationale during the first three episodes of treatment predicted change in depressive symptom severity midway through treatment and overall treatment outcome. They also reported that homework compliance predicted early depressive symptom severity change in depression across the course of treatment. However, there was no evidence to suggest that homework compliance mediates the relationship between acceptance of treatment rationale and within-treatment change or outcome.

Another cognitive item that has been explored in this area is the role of clients' expectations about therapy. Existing literature suggests that a clients prognostic beliefs or anticipation of relief (expectancy of change in therapy) play a role in the subsequent response to treatment across a number of different disorders (Dozois & Westra, 2005; Greenberg, Constantino, & Bruce, 2006; Westra, Dozois, & Marcus, 2007). In an investigation between pre-treatment expectancy in anxiety change and early homework compliance to initial and overall change in treatment for anxiety, Westra and colleagues (2007) found that the role of homework compliance early in therapy and early symptom change acted as possible mediators between expectancy (i.e., beliefs about outcome) and outcomes in CBT for anxiety. The study found that those clients who had higher baseline expectancy for change were more likely to achieve homework compliance, which in turn was associated with initial improvement. Therefore, pre-treatment positive expectancies for change may be one way to impact treatment improvement by influencing a client's engagement in the therapeutic process (Westra et al., 2007). Goodridge and Hardy's (2009) qualitative study into sudden gains shows that there is some level of insight within the sessions that precede the sudden gain. However, they found the rate of assimilation (of insight) is incremental and gradual which suggests that clients do not gain full insight into their problem until links generalise across situations, feelings, cognitions and behaviour. It could be argued that homework as a way of generalising and practising what is learned in therapy across these domains may have some relationship with session by session symptom change. These findings highlight the importance of cognitive constructs to treatment outcome.

Assessment of homework compliance: The Homework Rating Scale-Second Edition (HRS-II)

The Homework Rating Scale – Second Edition (HRS-II; Kazantzis, Deane & Ronan, 2005) was developed on the basis of theoretical and empirical foundations for homework assignments in CBT. The HRS-II is a 12-item self-report measure designed to assess a number of aspects within the process of designing homework, engaging in the process of homework, and reviewing homework (Kazantzis, et al., 2005). Items in the HRS-II specifically measure *rationale, comprehension, specificity* and *collaboration.* The measurement of these factors acknowledges that clients may have attitudes, rules or beliefs about the homework activity, and about their ability to complete it (Kazantzis et al., 2005). According to cognitive behavioural theory, it is likely that these beliefs, rules and attitudes around homework are reflective of the client's own experiences of themselves, the world and other people. It is argued that, if sufficiently explored, a client's beliefs about engaging in a homework task will be consistent with her/his presentation and conceptualisation (Garland & Scott, 2005). The HRS-II asks clients to rate the extent to which they understand the homework assignment (*comprehension*), understand the reasons why they are carrying out the homework assignment (*rationale*), the extent to which they play a role in designing the homework assignment (*collaboration*), and whether they found the guidelines for the homework assignment to be specific enough (*specificity*) (Kazantzis, et al., 2005). A client could have beliefs about the specific homework assignment (e.g., beliefs about the cost and/or benefits about engaging in the task) or broader beliefs that feed into their case conceptualisation (e.g., beliefs about their ability to cope with the task, perfectionism, etc.). At both levels, these beliefs, attitudes and rules provide valuable information that inform treatment, and need to be kept in mind by the therapist. Not only is it important to consider the beliefs that the client may hold about a task that they will carry out in the future, but also their beliefs about homework tasks that they have already carried out. Reviewing homework within the therapy session is a way of systematically incorporating homework into therapy and furthermore enables the client to synthesise their experience and form conclusions based on what they learnt from carrying out the task (Kazantzis et al., 2005). Two items on the HRS-II were designed to examine this degree of synthesised learning. It asks the client to rate the extent to which they found the homework task matched the goals of therapy

(*match therapy goals*) and found the activity helpful to their overall progress in therapy (*progress*) (Kazantzis, et al., 2005).

Bjornholdt (2006) carried out a factor analysis on both client's and therapist's ratings on the HRS-II. The first factor that was found comprised the items *pleasure, mastery, progress, quantity and quality*. It is said that the first three items (*pleasure, mastery, progress*) measure the client's beliefs about the perceived benefit of the homework while the last two items (*quantity, quality*) are measures of homework completion. The second factor that was found in this study comprised the items *rationale, comprehension, collaboration, specificity* and *goals*. These second factor variables are said to represent a client's beliefs about the process of assigning/designing homework (Bjornholdt, 2006).

Conclusion

This chapter has discussed homework as a potential process variable that may have a relationship with the phenomenon of sudden gains. There is both empirical and theoretical support to indicate that compliance with homework is related to better outcome in therapy. This chapter introduced the theoretical ties between homework and a client's belief system. When a client engages in homework it in turn affects his/her beliefs about the process of therapy and therefore is likely to affect outcome. As a process variable, homework may facilitate cognitive change which may lead to rapid symptom improvement or sudden gains. Therefore, investigating the possible link between homework and early sudden gains in CBT for depression may provide valuable information about the possible mechanism(s) responsible for sudden gains in CBT for depression.

CHAPTER SIX

AIMS AND RESEARCH QUESTIONS OF THE CURRENT STUDY

This chapter will introduce the overall research questions of the current study. It will also outline the specific hypotheses. This chapter will then proceed to present the key rationale for focussing on each particular area of research. There are two overarching research questions or aims within this study. Additionally there are seven main hypotheses that are investigated within the present study, which are presented below:

Research questions:

1. What are the client factors that may predict whether a client will obtain a sudden gain within CBT for depression?
2. What are the within-therapy factors that are associated with sudden gains within CBT for depression?

Specific hypotheses:

I. As clients progress through therapy, their overall levels of depression will decrease

II. Those clients who experience an early sudden gain are more likely to have better outcomes at the end of therapy than those clients who do not experience an early sudden gain

III. Those clients who experience an early sudden gain are more likely to have a less depressogenic attributional style at the start of therapy than those clients who do not experience an early sudden gain

IV. Those clients who experience an early sudden gain are less likely to have co-morbid diagnoses at the beginning of therapy than those who do not experience an early sudden gain

V. Attributional style change throughout therapy will moderate the relationship between sudden gains and outcome in therapy

VI. Client beliefs about the homework process throughout therapy will moderate the relationship between sudden gains and outcome in therapy

VII. Client beliefs about their progress in homework throughout therapy will moderate the relationship between sudden gains and outcome in therapy

Rationale for focus on cognitive behavioural therapy for depression

As discussed in Chapter One, depression is a significant and wide-ranging health problem. There were several reasons for the focus on major depression in this study. Not only is there a high estimated lifetime prevalence rate of major depression within New Zealand (Oakley-Browne et al., 2006), it can have high costs for the individual and their family and debilitating consequences on a societal and economic level. The substantial impact that depression has on society makes it a relevant focus for this study (see Chapter One for summary). Additionally, since the publication of Beck and colleagues' (1979) depression treatment manual, there has been a considerable quantity of international research into the effectiveness and efficacy of CBT for depression. This significant research base has focussed on whether CBT is an effective treatment for depression, however, recent outcome studies have begun to focus on the mechanisms of treatment and change in therapy (Kazdin, 2007). Therefore, the focus on major depression and CBT can help to build on the large body of research that already exists.

Although research into the phenomenon of sudden gains has branched out to other forms of psychiatric illnesses (e.g., generalised anxiety disorder, social phobia, posttraumatic stress disorder etc.) and treatment modalities (e.g., systematic behavioural family therapy, supportive-expressive therapy, etc.) the initial discovery of early sudden gains (Tang & DeRubeis, 1999) was found in the context of cognitive behavioural treatment for major depression. Additionally, the majority of sudden gains research has focussed on depression and CBT. Therefore, it was appropriate to continue to build on this research base and to see whether similar phenomena would be found within a New Zealand population.

A large incentive to focus on a cognitive behavioural approach to treatment is its acceptability within New Zealand. As reviewed in Chapter One, CBT has been comprehensively validated as an effective treatment for depression. One rationale for the focus on CBT as a treatment modality is the fact that a remarkable majority of practicing psychologists in New Zealand operate from primarily a cognitive-behavioural perspective (Kazantzis & Deane, 1998). As a large proportion of

psychologists in New Zealand operate from this modality, it seems appropriate that the current research help to provide guidance and information around early sudden gains and their role within therapy.

Rationale for focus on early sudden gains

As reviewed in Chapter Two, there are significant disadvantages within previously dominant models of assessing change, such as the dose-response and clinical significance methods. Chapter One introduced the argument for measuring change session-by-session to help identify important sessions and significant change points within therapy. Early research into discontinuous change patterns across therapy have indicated that clients who experience non-linear change patterns (e.g., 'depression spikes', 'transient worsening', 'early rapid responding') have better outcomes in therapy (Haas et al., 2002; Hayes et al., 2007; Illardi & Craighead; 1994; Thompson et al., 1995). One of these discontinuous change patterns that have received lots of attention is 'sudden gains' (Tang & DeRubeis; 1999). Previous research in the area of sudden-gains and CBT for depression have shown that they are not random or clinically insignificant, but rather that they are associated with better overall outcomes in the long term (Hardy, et al., 2005; Kelly et al., 2004; Tang & DeRubeis, 1999; Tang et al., 2005; Tang, DeRubeis, Hollon & Amsterdam, 2007). The rationale behind measuring sudden gains that occur in the early sessions of CBT is guided by the research in this field that emphasises that gains made in the first half of treatment are associated with more positive outcomes (Busch, et al., 2006; Kelly et al., 2004; Kelly et al., 2007). There are several reasons why the focus on early sudden gains within this study is important: sudden gains are associated with better outcomes within therapy and post-therapy; they may help to identify those clients who will respond favourably to therapy; and they may provide further clarification around change mechanisms and processes within therapy.

Rationale for understanding the client factors that predict sudden gains

With the emergence of brief intervention models it is useful to identify who will most benefit from short-term treatment. There appear to be some gaps in this area of research that accounts for the client factors that predict sudden gains in CBT for depression. Identifying clients that are more likely to experience sudden-gains in early sessions of CBT for depression will help to inform clinicians who will most

benefit (and maintain these benefits) from short-term CBT. Furthermore, if clients can be identified who may not typically make rapid improvements in symptomatology it may provide justification for the utilisation of *pre-therapy* treatment modalities such as carrying out motivational interviewing to increase a client's expectations about the value of the therapeutic process.

Rationale for understanding the mechanisms operating behind sudden gains

Sudden gains within CBT for depression are related to treatment outcome (see Chapter Three for summary). However, there is still uncertainty around what mechanisms and processes are contributing to this change within therapy (Kopta, Lueger, Saunders & Howard, 1999). This section aims to give a rationale for the methodological approach chosen within the current study and justification for the focus on the potential moderators focussed on within the study. In recent years there has been a call for the return of individual analyses (rather than the focus on group means) when studying the discontinuities of symptom change across treatment and considering questions of 'why' and 'how' this change is occurring (Barkham, Stiles & Shapiro, 1993; Hayes, et al., 2007a; Hayes, et al., 2007b; Krause, Howard & Lutz, 1998). It has been suggested that focussing research attention on phenomena such as 'sudden gains' can help guide researchers to the segments of therapy that are likely to identify and reveal predictors, moderators and mediators of the change process within therapy (Hayes, et al., 2007a).

There are several disadvantages to traditional ANOVA-based forms of data analysis in psychotherapy research (see Chapter Eight). These methodological designs assume that everyone in a treatment (or control) group responds exactly the same to either the treatment (or control) conditions. However, it is well known that there are individual differences in the ways that clients respond to therapy. Traditional ANOVA-based approaches treat these individual differences as sampling or measurement error rather than meaningful individual variability in change over the course of treatment (Laurenceau, Hayes & Feldman, 2007). Therefore, it was important to find an analytic technique that not only focuses on differences between individuals, but simultaneously models between-person and within-person change. It was therefore decided to use a multi-level model approach when analysing the data gathered (the rationale for this data analytic approach is outlined in Chapter Eight).

As outlined in Chapter Four, there has been some debate about the mechanisms behind change in CBT for depression. One dominant challenge to the cognitive mediation hypothesis that underlies the core theory of change within CBT for depression (Beck et al., 1979) is the argument that 'common factors' rather than 'specific ingredients' in therapy are responsible for change (Messer & Wampold, 2002; Wampold, 2001). Another dominant argument within this research area is based around the findings from 'component analysis' studies (see Chapter Four). Researchers in this area have suggested that specific cognitive components in therapy are not necessarily responsible for the bulk of symptom change within therapy, and that 'behavioural components' on their own are of comparable efficacy (e.g., Dimidjian et al., 2006). A few studies have indicated that cognitive changes are associated with symptom change when clients experience sudden gains in therapy (Goodridge & Hardy, 2009; Tang & DeRubeis, 1999; Tang et al., 2005). However, this evidence is limited. Therefore, the current study aims to build on evidence for the moderating role of beliefs (i.e., cognitive constructs) in cognitive therapy for depression, and to investigate whether changes in beliefs moderate the relationship between sudden gains and outcome in therapy. 'Cognitive change' in the current study is operationalised as change in attributional style as measured by the Attributional Style Questionnaire and clients beliefs about homework within therapy as measured by the Homework Rating Scale – Second Edition (HRS-II; Kazantxis et al., 2005). Within this study homework is conceptualised as a reflection of the 'process variable' in therapy. Literature in this area emphasises that the process of designing and assigning homework within therapy is directly linked to a client's belief system (Kazantzis & Daniel, 2009; Kazantzis & L'Abate, 2005; Garland & Scott, 2005). Research studies have also supported this notion (e.g., Addis & Jacobson, 1996; Fennell & Teasdale, 1987; Westra et al., 2007). A unique aspect of the current study is that it explores the relationship between client beliefs about the homework process and early sudden gains in CBT for depression. This has not been explored before in the current research literature.

Summary

This chapter has outlined the research questions, the hypotheses and the rationale for the present study. It has provided a summary of the main arguments within the introductory chapters of the current study and gives a rationale for the current areas

of focus based on theory and research in the area of CBT for depression. Chapter Seven, Eight and Nine will introduce the research design employed in the current study, the quantitative analytic techniques employed and the results from the current study.

CHAPTER SEVEN

METHODOLOGY

Introduction: 'The Depression Study'

The present study is set within the context of a wider research project within the School of Psychology at Massey University in Albany named the 'depression study' or also known as the 'Cognitive Behavioural Therapy Homework Project'. The study was set up to examine the process of homework within cognitive behavioural therapy for depression (Kazanzis, MacEwan & Dattilio, 2005). This chapter aims to give an overview of the 'depression study' sufficient to understand and replicate the current project.

Participants

The sample consisted of 28 participants (N=28), 10 were males and 18 were females of an average age of 44.75 years (range from 20 to 62 years old). Twenty-four out of the 28 participants identified themselves as European/Caucasian (85.7%). The other four participants identified as New Zealand Indian, American, Scottish and Australian. All clients included within the study met diagnostic criteria for major depressive disorder (MDD) as their primary diagnosis. Clients were initially assessed using the *Composite International Diagnostic Interview Version 2.1*(CIDI; Kessler & Ustun, 2004) and some clients met additional diagnostic criteria for other psychiatric diagnoses (*n*=13) including, generalised anxiety disorder, social phobia, PTSD, panic disorder, specific phobia, OCD, brief psychotic disorder, alcohol dependence, alcohol abuse, agoraphobia, bulimia nervosa, conversion disorder, hypochondriasis, pain disorder, nicotine dependence, and nicotine withdrawal. These clients were later assessed by the treating therapist to meet criteria for a primary diagnosis of MDD without meeting any exclusionary criteria. Each client completed a full course of CBT for depression for up to 20 sessions (average of 17.79 sessions) with some clients completing the two additional booster sessions at two months (*n*=11, 38%) and six months (*n*=4, 14%) following therapy.

Therapists and Treatment Fidelity

Seven female therapists participated in the study. They all identified as of European/Caucasian decent. The mean age of the therapists was 36.86 years (range from 23 to 50 years old). All therapists were Doctor of Clinical Psychology students, who had attended two advanced block courses in the theory and practice of CBT for depression (i.e., 'Theory and Practice of Cognitive Behaviour Therapy' and 'Cognitive Behaviour Therapy for Depression'), run by Massey University. This was additional to their standard clinical training towards their doctorate. The therapists were trained specifically in homework administration which consisted of two days training in homework protocol (as per Kazantzis, MacEwan, & Dattilio, 2005) run by Dr Nikolas Kazantzis, an experienced registered clinical psychologist and senior lecturer at Massey University (currently at La Trobe University in Melbourne) and primary investigator of the 'depression study'.

Therapists were required to demonstrate clinical competence whilst they were delivering CBT for depression throughout the study. During the time that the project was running therapists received weekly group supervision from a registered clinical psychologist with experience in clinical supervision for the practice of CBT. Before therapists were allowed to see more than three clients they were required to obtain three ratings above 40 on the Cognitive Therapy Scale (CTS; Young & Beck, 1980) as rated by the group supervisor. To ensure that competence in homework administration was maintained, randomly selected video-recorded therapy sessions were viewed by independent postgraduate psychology students using the Homework Adherence And Competence Scale (HAACS; Kazantzis, Wedge, & Dobson, 2005). These adherence scores were fed back to the therapists in supervision and any adherence issues were problem solved with the group supervisor.

Procedure

Recruitment of participants

Participants were recruited from the public in the wider Auckland area for participation in the *Depression Study* via multiple forms of media advertisements (i.e., suburban newspapers, campus magazines) (See Appendix A for examples).

Pamphlets were also designed for distribution to University health centres and local General Practitioners' offices.

Interested participants left answer phone messages and were informed that that they would be contacted by a 'depression study' coordinator to arrange an initial phone interview. There were three doctoral-level study co-ordinators (including the author). Each was a psychology postgraduate with research, administration, statistical skills and experience necessary for the requirements of the study. The study co-ordinators acted as the contact point between the participants/clients and the therapists and carried out safety monitoring across the 'depression study'.

Initial Screening

Applicants undertook a telephone interview by a postgraduate psychology student (see Appendix B for interview format). Participants were screened to ensure that they meet specific inclusion criteria. Participants met criteria for their first episode of Major Depression as defined by the DSM-IV-TR (APA, 2000), were aged between 18 and 65 years, not taking any medication that may affect the central nervous system, not concurrently participating in any other psychotherapy or counselling for depression, not experiencing psychosis or borderline personality disorder, and had no imminent risk of self harm. The telephone screening interview format included standardised questions based on the DSM-IV-TR criteria for major depressive episode. Participants were asked whether they had any current thoughts or intent to harm themselves or others. Additionally, a risk assessment was carried out at any time in the interview where participants expressed any degree if risk of harm to themselves or others.

Secondary Screening

Participants who met preliminary criteria for involvement in the 'depression study' were invited to attend a pencil and paper intake assessment at the Massey University Centre for Psychology. At this point the participant was sent via mail an information sheet on the study and a confidentiality agreement (see Appendix C). Any participant that was deemed unsuitable for the study was referred to the appropriate community health or mental health service/resource.

In the second phase of screening participants were oriented around the Centre of Psychology. They then completed a 50 minute computerised Composite International Diagnostic Interview (CIDI; Robins, Wing, Wittchen, Helzer, Babor & Burke, 1989) at the Massey University Centre for Psychology and also completed the BDI-II (Beck, Steer & Brown, 1996), and the ASQ (Peterson et al., 1982). Clients were then invited back to be interviewed by a therapist to ensure that they met diagnostic criteria for a major depressive episode (MDE). Therapists used a semi-structured interview guide and completed the Suitability for Cognitive Therapy Scale (Safran & Segal, 1990) and the Social and Occupational Functioning Assessment Scale (SOFAS; Goldman, Skodal, & Lave, 1992) after the initial clinical assessment interview. Figure 7.1 below outlines the number of clients within each stage of the screening process.

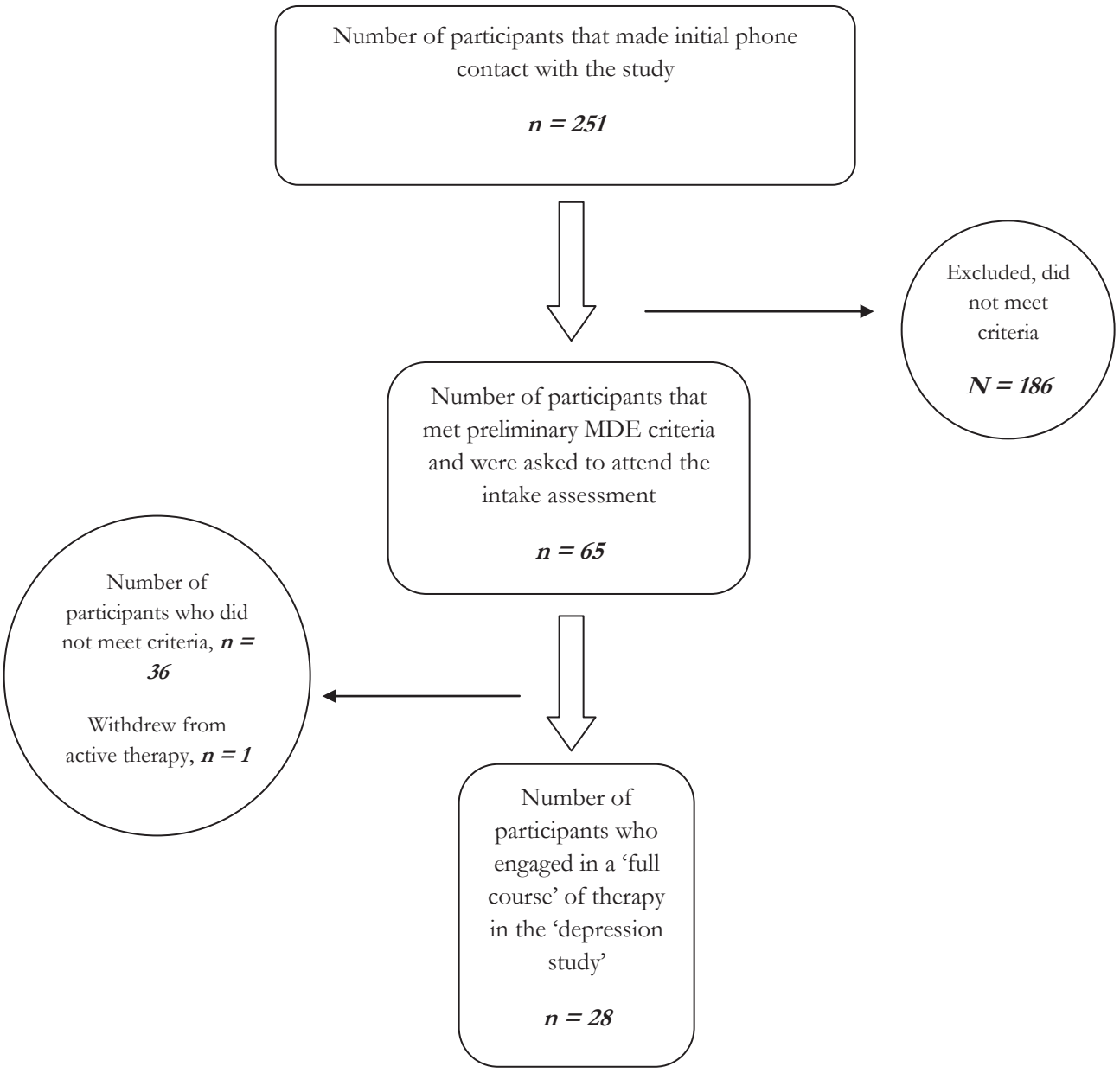

Figure 7.1: Number of participants at each stage of the recruitment process

Therapy

The therapy stage of the 'depression study' was carried out from 2007 to 2009 and clients were recruited at different times throughout this period. After providing written informed consent, participants received up to 20 sessions of standardised protocol CBT for depression over a 16 week period. Treatment was individualised for participants and the first 8 sessions were offered twice weekly. The last two

sessions were spaced bi-weekly. Follow up sessions occurred at two months and six months post treatment. The average number of therapy sessions across the total number of participants (N=28) was 17.79 (range from 6 to 20 sessions). The *depression study* had a large focus on enhancing homework completion. Therapists adhered to protocol (see Kazantzis, MacEwan et al., 2005, pp 380-400) that saw them a) *reviewing* clients' homework, reinforcing efforts made and conceptualising with the client barriers that lead to non-completion, b) assisting the client to *design* new homework that is relevant to the content of the session and makes sense to the client and finally c) *assigning* the homework in a way that will facilitate the client completing the homework (e.g., problem solving barriers to completion). Therapy would generally involve psycho-education, behavioural tasks (e.g., activity scheduling) and cognitive tasks (e.g., using thought records to challenge negative thinking).

The final sample size consisted of 28 people who received treatment in the 'depression study' (N=28). Clients were selected from an initial applicant pool of 251. Client participation was voluntary and therapy was provided free of charge.

Measures

Outline of relevant measures

The pre-treatment assessment included structured clinical interviews to determine the presence or absence of Axis I and II disorders. Participants were asked to complete the Composite International Diagnostic Interview (CIDI), the Beck Depression Inventory – Second Edition (BDI-II) to assess depression severity, and the Attributional Style Questionnaire (ASQ) to assess current attributions. Participants were then seen by the treating therapist for the status of their current condition. These clinicians assessed current functioning using the Social and Occupational Functioning Assessment Scale (SOFAS from the DSM-IV-TR).

Depression severity, attributions and functioning were assessed at Session 0 (intake), Session 5, Session 8 and Session 20. Depression severity was measured by the BDI-II at the beginning of each session. The primary outcome measure was sustained symptom reduction on the BDI-II over subsequent assessments. Secondary outcome measures were rates of remission, recovery, and maintenance of recovery

from depressive symptoms. *Remission* was defined as BDI-II less than 10 for two consecutive assessments (2 consecutive weeks). *Recovery* was defined as a BDI-II less than 10 for three consecutive assessments (8 consecutive weeks).

Table 7.1

Outline of relevant measures used at each time point in current study

Time point	Measure used
Intake/Session 0	Beck Depression Inventory – Second Edition (BDI-II; Beck, Steer & Brown, 1996) Attributional Style Questionnaire (ASQ; Peterson & Villanova, 1988) Composite International Diagnostic Interview (CIDI; WHO, 1997)
Sessions 5, 8, 20 and 2 month and 6 month follow up	Attributional Style Questionnaire (ASQ; Peterson & Villanova, 1988)
Sessions 0-20 including 2 month and 6 month follow up	Beck Depression Inventory – Second Edition (BDI-II; Beck, Steer & Brown, 1996)
Sessions 2-20	Homework Rating Scale – Second Edition (HRS-II, Kazantzis, Deane & Ronan, 2005)

Beck Depression Inventory – Second Edition

Depressive symptom severity was measured using the Beck Depression Inventory – Second Edition (BDI-II; Beck, et al., 1996) which was administered at initial assessment and from session one through to session twenty. The BDI-II is a widely used 21 item self-report measure of depressive severity and has been shown to have strong psychometric qualities (Beck, et al., 1996). Each item is rated on a '0' to '3' point scale with possible scores ranging from 0 to 63. Scores have been grouped into different ranges of severity, i.e., 0-13 = 'minimal range'; 14-19 = 'mild range';

20-28 = 'moderate range'; and 29-63 = 'severe range'. The BDI-II has shown great internal consistency (α = .92) among a sample of depressed outpatients and college students (α = .93) (Beck et al., 1996).

Composite International Diagnostic Interview

The Composite International Diagnostic Interview (CIDI) Version 2.1 (WHO; 1997) was administered to participants at the assessment phase to identify co-morbid diagnoses. The CIDI was used to measure the clients' pre-treatment co-morbid Axis I and II diagnoses. The CIDI was originally developed by the World Health Organisation (WHO) and the United States Alcohol, Drug Abuse, and Mental Health Administration (ADAMHA). The CIDI is useful in this current study as it can be used to generate a preliminary diagnosis of MDE and identify potential co-morbid Axis I psychiatric difficulties. Although an individual's response on the CIDI does not qualify as a formal diagnosis, it does provide useful information that can be followed up with a clinical interview with the clinician. Additionally, the CIDI can be administered by 'lay people' with limited clinical training (Rosenman, Levings, & Korten, 1997).

Attributional Style Questionnaire

Attributional style was measured by using the extended Attributional Style Questionnaire (ASQ; Peterson & Villanova, 1988). The ASQ is a widely used measure of the depressive cognitive style associated with the hopelessness theory of depression. The ASQ was administered at the assessment phase to measure the clients' pre-treatment attributional style. The attribution measures in this study were the composite scores derived from the ASQ. Scores for the three attributional dimensions (i.e., internality, stability and globality) were summed for the negative and positive events on the ASQ. Thus, higher scores on the 'composite negative' (CoNeg) scale represented a stronger tendency to relate negative events to internal, stable and global causes. Higher scores on the 'composite positive' (CoPos) scale, thus represents a stronger tendency to relate positive events to internal, stable and global causes. To create a combined composite score ('CPCN') the 'composite negative' score is subtracted from the 'composite positive' score. Thus lower scores represent a stronger tendency to attribute negative events, relative to positive ones, to internal, stable and global factors.

As well as being administered at intake the ASQ was administered at session five, session eight and session 20. These (early) sessions in particular were chosen as a result of current literature in the research area that suggests that most therapeutic change happens early in therapy (e.g., Illardi & Craighead, 1994). This made it possible for change in attributional style to be measured during therapy and therefore a change in cognition to be measured.

Homework Rating Scale – Second Edition

The Homework Rating Scale (HRS-II; Kazantzis, et al., 2005) is a 12 item questionnaire rated on a 5 point rating scale (ranging from '0' = not at all to '4' = extremely) which is used to measure client's beliefs about homework (i.e., between session activities) over the course of therapy. Both the client version and the therapies version were used in the 'depression study'. Research by Munro (2006) had demonstrated in a sample of 56 participants that the HRS-II had an inter-rater correlation coefficient (ICC) of .82, whilst the majority of individual item ICCs were greater than .60. Similarly, in a sample of 74 clients' DVD recorded therapy sessions of CBT for depression, this study demonstrated that it had an overall ICC of.82 between clients and therapist versions of the scale. Additionally, factorial validity for a four factor model was adequate (.45, N = 104) = 101.6, $p < .001$ for the client version of the scale whilst poor fit was shown for the therapist and independent observer versions (Kazantzis, Bjornholdt, Munro, Dobson, Merrick, Fletcher & Jones, 2006).

Ethical considerations

The research for this thesis was carried out under the umbrella of a larger project which had received ethical approval from the Northern X Regional Ethics Committee (NTX/06/08/085).

In fulfilment with obligations outlined from the ethics committee, ongoing monitoring of participant safety took place during active therapy. To ensure client safety, clients needed to be seen as generally improving as a result of therapy. This was operationally defined by BDI-II scores (i.e., that the BDI-II scores were generally positive). It was agreed that if more than 50% of the clients engaged in active therapy were not observed to be improving that the 'depression study' would

be discontinued. All participants were required to give informed consent (see Appendix C for copy).

Summary

This chapter has introduced the methodological design of the current study. It has described how the sample was recruited, the screening criteria and the primary assessment tools. Chapter Eight will introduce the analytic strategy used within the current study to answer the research questions as outlined in Chapter Six.

CHAPTER EIGHT

DATA ANALYSIS

Introduction

This chapter aims to provide an introduction to the analytic strategy used in this study using quantitative methods. The chapter will introduce multi-level modelling (MLM) as the primary analytic technique within the current study. It will also outline the rationale and advantages of using this statistical approach. The treatment and screening of data will be described as well as a strategy for checking for model assumptions. Additionally, this chapter will introduce how *sudden gains* are defined within this study, and the rationale for defining these phenomena this way. Finally, this chapter outlines the multilevel model building process and how this was carried out within the current study.

Structure of the data collected within the 'depression study'

The data within the *depression study* has been collected over a number of levels, for example, by individual client, by therapist, and by session. Additionally, both *within subject* and *between subject* variations are of interest in this study. For example, the current study is interested in identifying those clients who experience early rapid change in therapy and the factors that make them different from those clients who do not. It could be said that the data are "nested" within themselves and therefore are congruent with a multi-level analytic strategy that simultaneously analyses relationships that occur within clients and between clients at different levels of analysis. Traditionally psychotherapy data has been analysed using statistical approaches such as ANOVA (analysis of variance) and MANOVA (multivariate analysis of variance) that aggregate scores without consideration of within-subject effects. This is a disadvantage if one is interested in individual subject trajectories. Hedeckler (2004) provides an example comparing a psychological study comparing MLM analysis and a 'traditional' MANOVA approach. This study demonstrated that the MANOVA approach reduced the sample size by a third compared to the MLM approach. Additionally, it missed out a critical relationship whereby some individuals had a poor response to medication. Multi-level analysis is advantageous as it allows 'individual growth models' to be developed rather than focussing on group means

over time. Therefore, trajectories can be produced for individuals within the study, and relationships can be derived within an individual's data over time ('*Level 1'* relationships) and for relationships in the data between individuals ('*Level 2'* relationships) (Hedeckler, 2004; Kwok, Underhill, Berry, Luo, Elliot & Yoon, 2008).

Definition of 'multi-level analysis'

Multi-level models can be conceptualised as a series of regression models that are interrelated, which explain different sources of variation at multiple levels of analysis (i.e., they have a hierarchical structure) (Hoffman & Rovine, 2007). A data set is said to have a hierarchical structure when one or more observations (e.g., clients/participants) are "nested" within another unit (e.g., therapists) (Reise & Duan, 1999). This type of analysis has been given a number of different names and definitions across the literature, for example, Applied Longitudinal Data Analysis (ALDA; Singer & Willett, 2003), Hierarchical Linear Models (HLM; Raudenbush & Bryk, 2002), random coefficient models (Longford, 1993) and mixed effect models (Littel, Milliken, Stroup, Wolfinger & Scabenberber, 2006). For the purposes of this study, the analytical approach will be defined and named as 'multi-level modelling' or MLM.

Advantages of multi level modelling

There are numerous advantages for using this analytic approach in the current study. First of all the sample size of 'the depression study' was made up of 28 participants. Not all participants completed all 20 therapy sessions and not all participants completed the two and six month follow up sessions in this study. MLM's assumptions regarding sample size are flexible enough that they allow for unbalanced designs, attrition and missing data. It is normal for attrition to occur in longitudinal studies and is typical in therapy as a whole. The MLM approach allows *intent to treat* samples to be analysed (Kwok et al., 2008; Quene & van den Bergh, 2004). Secondly, time is treated as a continuous variable. This is advantageous because it is acceptable to have different waves of data for each client, and to have unequal spacing of data (Hedeckler, 2004; Kwok et al., 2008). Within the current study, depression severity data was collected at every session; in contrast information regarding attributional style was collected at intake, session 5, 8 and 20 and data collected regarding homework could only be practically gathered from session 2

onward. Therefore, there were unequal quantities and unequal spacing between different measures used in the study. MLM analysis is advantageous as it does not assume that variances and co-variances are equal across time as in ANOVA (Hedeckler, 2004).

The treatment of data

Statistical software used

Data was analysed using the Statistical Package for Social Sciences (SPSS) for windows, Version 17.0 (SPSS Inc., 2008).

Treatment of time

Singer and Willett (2003) describe three essential requirements for building a multi-level model. It must have at least three waves of data (the current study has 22), that is data must be collected across a minimum of three time points. Secondly, the model must have a reliable outcome measure which changes systematically across time. Finally, a sensible measure of time is also required (e.g., weeks, age, etc.). In the current study time was represented by therapy sessions and the outcome measure used across every session was the BDI-II (Beck, et al., 1996), which measured depression severity. There were a number of different ways that time could be coded in this study. For example, one could count the number of days or weeks between each session. In this study it was decided that time would be coded by session, the intake session would be centred as session '0' as no active therapy had taken place at this point in time. Each additional session would be coded as session '1, 2, 3, 4, 5...20' progressively. Since sessions were carried out bi-weekly in the first four weeks of therapy it needed to be considered whether the first 8 sessions should be coded as '0.5, 1, 1.5, 2...4'. Although this coding would account more accurately for time, it does not reflect that sudden gains occur from session to session (rather than as a construct of time). Furthermore, when a client attends a session bi-weekly they are engaged in two hours of therapy across two sessions and are required to complete homework between these sessions. Therefore coding time in whole intervals more accurately reflects this dose effect. It is also important to note that there were individual differences between clients regarding the amount of time

between sessions and it was not always possible for sessions to be conducted bi-weekly because of practical obligations of the clients.

Missing data

It was important to consider missing data within the current sample. Although the assumptions of MLM are robust in that they can accommodate for missing data using maximum likelihood (ML) estimation (Kwok et al., 2008; Singer & Willett, 2003). The validity of using all available data can depend on the nature of the data that is missing (Kwok et al., 2008). In particular it is important to consider whether data are missing completely at random (MCAR), (i.e., when the reason for this missing data are unrelated to any observed or unobserved data) or missing at random (MAR), (i.e., when the missing data depends on the observed data but is unrelated to unobserved data) (Scheffer, 2002). Little's MCAR test can be used in SPSS to see whether there is a significant relationship between the missing data and the observed values (p <.001). This confirms whether or not missing data is missing are random. Case deletion (either list-wise or pair-wise) tends to be avoided in multi-variate analysis as it has the potential to create biased estimates and can limit the representativeness of the sample in question (Schafer & Graham, 2002). An alternative that is often used in MLM to deal with missing data is 'case imputation' (Allison, 2002). Instead of deleting cases, imputation involves filling in missing values. This approach helps to lessen any loss of power caused by the diminished sample size which occurs when cases are deleted (Schafer & Graham, 2002). In this study missing values of the trimmed data set were calculated and replaced using expectation-maximisation (EM) imputation method in SPSS. Table 8.1 outlines the proportions of missing data within the primary measures and demonstrates results from Little's MCAR test within SPSS.

Table 8.1

Proportions of missing data for primary measures used and results from Little's MCAR tests

Measure	*Percentages missing across sessions*	*Little's MCAR test*
BDI-II	0.17% (session 2)	Non-significant
	0.17% (session 4)	Non-significant
	0.17% (session 8)	Non-significant
ASQ	0%	Non-significant
HRS-II	1.3-1.9%	Non-significant

Assumption checks

According to Singer and Willett (2003), there are three primary assumption checks that need to be carried out (1) *linearity* – that is that the systematic part of the model is specified appropriately and did not leave out any additional terms such as interactions; (2) *normality* – the assumption that the error distribution is normal; and (3) *homoscedasticity* – the assumption that the error variance is constant across observations (Reise & Duan, 1999; Singer & Willett, 2003). The validation of these three critical assumptions within the data set permits the use of regression analysis and procedures such as data cleansing and transformation can be deemed as unnecessary. Additionally, the confirmation of these assumptions can strengthen the confidence in the accuracy in the final results and reduces the risk of making a Type I or Type II error.

By applying the standard recommendations outlined by Tabachnick and Fidell (2007), the 'Normal Probability Plot' ('P-P plot) of the main dependent variable (BDI-II) was checked to see that the residuals formed a diagonal line (normal distribution). Additionally, the assumption of *normality* is confirmed by the random scattering of residuals around the centre of the plot rather than skewed at the bottom or the top. Scatter plots were examined to make sure that they were (approximately) rectangular in distribution, to ensure that the relationship was *linear* rather than curvilinear. Furthermore, in accordance with guidelines from Tabachnick and Fidell (2007), scatter plots were examined to see whether the residuals were distributed in roughly equal widths across the graph, this is to ensure that the assumption of *homoscedasticity* was met.

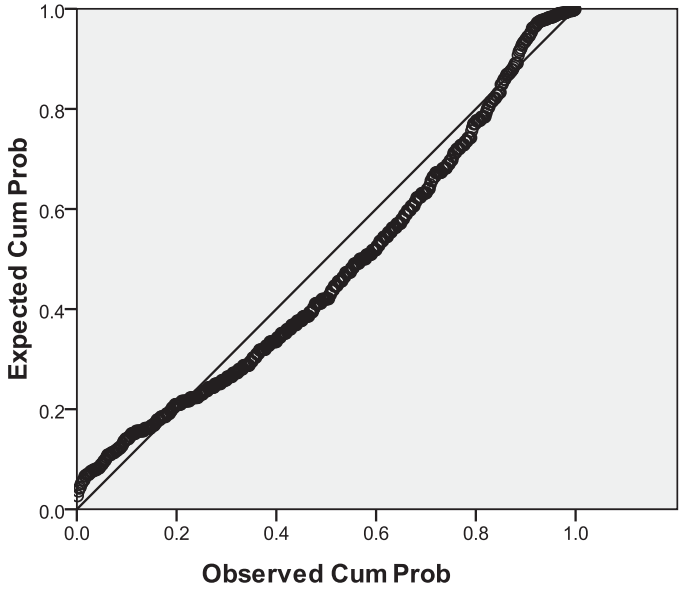

Figure 8.1: Residual plot of depression severity scores (BDI-II change)

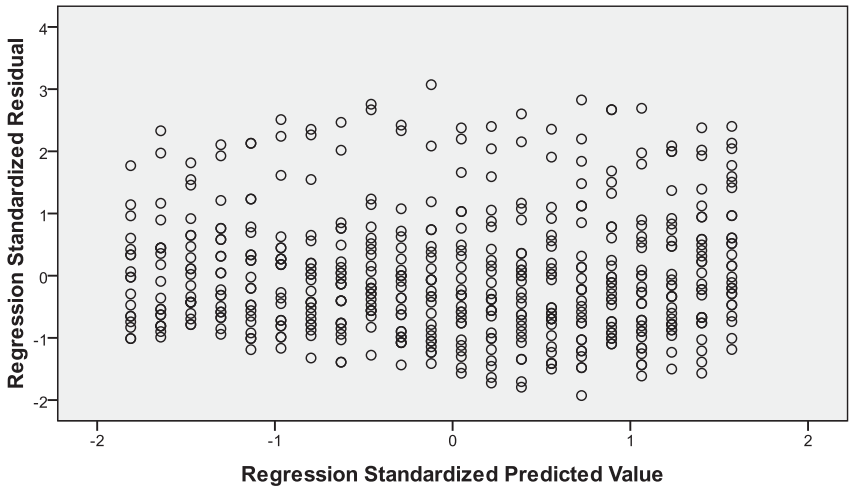

Figure 8.2: Standardised residual scatter plots for depression severity across time (BDI-II change)

Upon visual inspection the residuals in Figure 8.1 show that the residuals follow a roughly diagonal line which indicates minimal deviance from normality. Furthermore, Figure 8.2 shows roughly a rectangular shape, which is centred in the middle and has roughly equal width throughout. These preliminary checks showed that the assumptions of linearity, normality and homoscedasticity are adequately met for the primary dependent variable in this study (i.e., depressive severity). Along with checking the assumptions for the BDI-II as the main dependent variable, other

primary parameters (i.e., ASQ and HRS-II) used in this study were also assessed by regressing them against the dependent variable (BDI-II) (see Appendix E). By visually inspecting these plots there appears to be no major departure in the assumptions of linearity, normality or homoscedasticity that would violate assumptions underlying further regression analysis.

Management and definition of variables

This study utilised data collected from intake (defined as 'Session 0') to 'Session 20'. A total of 21 time points were utilised in this study out of a total of 23 available time points (i.e., two-month and six-month follow up sessions). The rationale for using the trimmed data set (i.e., 'Session 0' to 'Session 20') was that the current study was interested in change over the course of therapy, rather than change after therapy. Therefore, the data set was trimmed.

Defining 'sudden gains'

Sudden gains were originally operationalised by Tang and DeRubeis (1999) using three main criteria. These stated that the gain between *N* (the pre-gain session) and *N+1* (the after-gain session) must meet the following criteria:

(1) The gain (improvement on a depression severity measure, e.g., BDI-II) between *N* and *N+1* should be large in absolute terms. Using the Beck Depression Inventory (BDI) as a measure of depression severity Tang and DeRubeis (1999) specified that the gain should be equal to 7 BDI points or more ($BDI\ N - BDI\ N+1 \geq 7$). Tang and DeRubeis (1999) justified this criterion based on previously reported secondary peaks in frequency distribution plots between BDI score changes. In support of this criterion, Stiles et al., (2003) acknowledged that a 7 point gain on the BDI was close to the BDI's reliable change index (RCI) (see Jacobson & Truax, 1991).

(2) The gain should be large relative to the symptom severity before the gain. Specifically, Tang and DeRubeis (1999) specified that the gain should represent at least 25% of the pre-gain session's BDI score ($BDI\ N - BDI\ N+1 \geq 0.25 \times BDI\ N$).

(3) The gain should be large in relation to fluctuations in depression severity before and after the gain. Tang and DeRubeis (1999) specified that the mean BDI-II score of the three therapy sessions before the gain (*N-2, N-1 and N*) should be significantly

higher than the mean BDI score of the three therapy sessions after the gain (*N+1, N+2 and N+3)* using a two sample *t* test, with an alpha of 0.05.

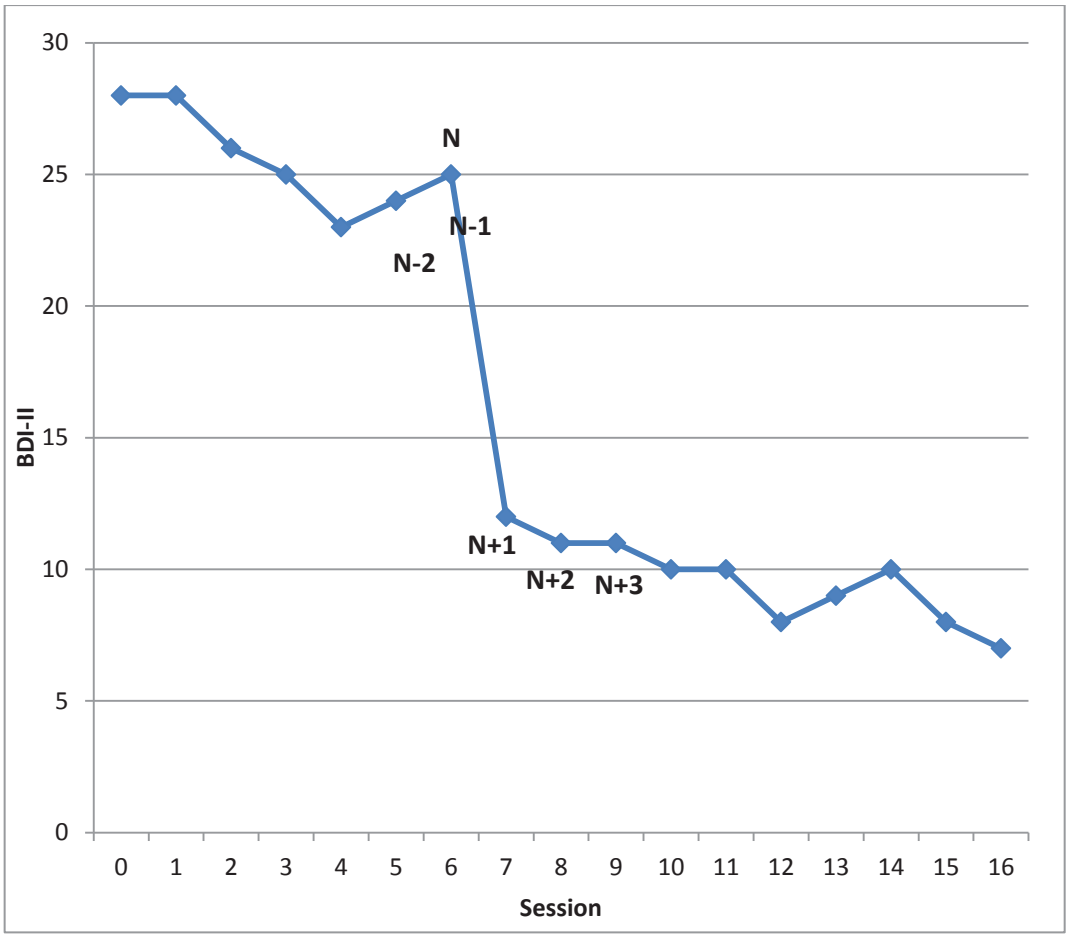

Figure 9.3: Example of a 'sudden gain' as defined by Tang and DeRubeis (1999) original criteria (See Tang & DeRubeis, 1999; p. 896).

Many researchers in this area have acknowledged that these three criteria are somewhat arbitrary, and therefore are subject to criticism and revision (e.g., Hardy et al., 2005; Kelly, Roberts, et al., 2007; Tang et al., 2002; Vittengl et al., 2005). More specifically, in terms of criterion two Hardy et al., (2005) argued that Tang and DeRubeis did not adequately justify why the gain should be at least 25%. They assert that this criterion is problematic as it assumes that the BDI as a measure is a ratio scale. They argue that at best the BDI is an interval scale and therefore is sensitive to arbitrary scaling decisions (Hardy et al., 2005). Several studies have found that when they had omitted this criterion, they yielded no additional sudden gain clients (Hardy et al., 2005; Stiles et al., 2003). Therefore, questions remain as to the value of maintaining this criterion. In regards to criterion three, it has been argued that the

three pre-gain and three after-gain sessions are not independent observations and therefore positive autocorrelation effects are likely to deflate the *t* value, running the risk that the comparison would not be a valid inferential test (Hardy et al., 2005; Vittengl et al., 2005). Another complication can arise when there are cases where only two sessions are available prior to or following the gain. This reduction in degrees of freedom is not often accounted for (Kelly et al., 2007; Tang et al., 2005). The original third criterion (Tang & DeRubeis, 1999) make it impossible to assess potential large improvement that occur very early or very late in treatment. This could substantially reduce the amount of potential sudden gains that can be accounted for. Other studies that have assessed the phenomenon of sudden gains have altered this third criterion (e.g., Tang et al., 2002; Hardy et al., 2005; Vittengl et al., 2005). In particular, Kelly et al., (2004; 2007) have required that a sudden gain be greater than or equal to 1.5 times the individual's standard deviation across all time points. It is argued that this criterion adheres to the intent of the original criterion by screening out individuals with typically large shifts in depressive symptoms, whilst easing the statistical and practical complications of criterion three as described above (Kelly et al., 2007).

In terms of the current study the complications discussed above were kept in mind when defining how sudden gains would be operationalised. The criteria were kept as consistent as possible for practical reasons and so that findings of this study could be used to compare findings from other studies in the area of sudden gains. Therefore, criterion one and two were kept consistent with the original as defined by the Tang and DeRubeis (1999) study. Criterion three was modified in that the *sudden gain* was required to be equal to or greater than 1.5 times the individual's standard deviation across all time points, thus demonstrating that the sudden gain is not simply a *random fluctuation*. This study was particularly interested in *gains* that were attained early on in therapy, as in past research these have been associated with better outcomes post therapy (e.g., Kelly et al., 2004; Tang & DeRubeis, 1999). In this study a *sudden gain* is only accounted for if it occurs within the first 10 weeks (14 sessions) of therapy. The gain is not counted if it is obtained within the first session of therapy as no active therapy has taken place. Additionally, there is a range of time between clients regarding when their BDI-II is first measured and the time that they first engage with a therapist. However, it is counted if it is obtained within session 2 to 14. In other

studies of sudden gains those clients who experienced *sudden gains* but also experienced a reversal of this *gain*, (i.e., in a later session of therapy obtaining a score that is 50% or more of the *original gain*) did not meet the criterion of obtaining a sudden gain (Tang & DeRubeis, 1999). For example, if a client's pre-gain BDI (*BDI N*) score was 30 and their after-gain session (*BDI +1)* was 20 if they obtained a score in a later therapy session that was 25 or more this would be counted as a 'reversal'. Within the current study this criterion was not utilised due to literature that states that *depressive spikes* or *worsening of symptoms* are also associated with better outcomes in CBT (Hayes, et al., 2007a).

Defining variables as time-variant or time-invariant

In MLM variables included in the model need to be defined in terms of what *level* they will be modelled at. That is, are they time-variant and therefore a *Level 1* predictor or time-invariant and therefore a *Level 2* predictor. The BDI-II, ASQ and HRS-II can all be defined as either *Level 1* or *Level 2* predictors. Therefore each variable needs to be considered carefully and defined at a particular level within the model.

BDI-II depression severity

In the current study BDI-II as the primary outcome variable is modelled at *Level 1*. The current study had a rich data set due to the fact that the BDI-II was administered at each time point in therapy for all clients. One of the first steps in MLM analysis is to assess whether variability exists at the lowest level (Singer & Willett, 2003). MLM is only appropriate in cases where there is sufficient and significant variability between groups in terms of the dependent variable. If it is found that there is variance at this level, variables (at the same or higher levels) can then be added to the model in an attempt to explain the variability either within-individuals and/or between individuals (Bryk & Raudenbush, 1992; Singer and Willett, 2003).

Sudden gains

Sudden gains are treated as a *Level 2* (time invariant) predictor in this study. Those clients who met the outlined criteria (see above) for experiencing a sudden gain in this study were given a value of '1' (*sudden gain*) and those who do not meet criteria

for experiencing a sudden gain throughout therapy are given a value of '0' (*no sudden gain*) throughout the person period data set. 43% of the current sample (n = 12) experienced a sudden gain.

Attributional style at intake

Attributional style was measured using data from the composite score of the Attributional Style Questionnaire. Attributional style was collected at five time points (the current study used a trimmed data set with four time points) throughout therapy – intake, session five, session eight, session twenty and follow-up sessions. A client's initial composite ASQ score (that measures both negative and positive attributional style) at intake is used as a *Level 2* variable (time invariant) in the current study. As a *Level 2* variable a client's initial ASQ score acts as a predictor to help answer the research question about the client variables that may help explain the variance in depression severity improvement throughout therapy.

Attributional style change across therapy

The ASQ composite score is also utilised as a *Level 1* (time variant) variable in the current study. As only five time points were available across therapy sessions, it meant that sessions where the ASQ score was not collected would not be included in analyses, meaning that 17 time points would not be included. Another option was to use linear interpolation to extend the ASQ scores over all time points in between measurements (Roth, 1994). The latter option was used in the current study to make use of all available time points.

Co-morbidity

The occurrence of co-morbid psychiatric disorders (as defined by the DSM-IV-TR; APA, 2000), as well as major depression at intake, was measured by the CIDI. Those clients who met criteria for two or more psychiatric disorders (including MDE) were defined as '*co-morbid*' and given a value of '1'. Those clients who only met criteria for major depression and showed no co-morbid diagnoses on the CIDI were given a value of '0'. Co-morbitidy was defined as a *Level 2* (time invariant) predictor within the current study.

Both the client version and the therapist version of this measure were available for analysis. Only the client version was used for analysis in this study as the focus of the thesis is on clients' beliefs of their homework within therapy. Therefore, the client version would give the most personal account of their experience of homework and their beliefs around this process. Secondly, it had to be decided how the HRS-II data would be utilised within this study (e.g., use of single items or total scores). The HRS-II factors as described in Bjornholdt (2006) were considered for this study (outlined in Table 8.2). Residual plots were examined to check for items that violated assumptions of normality, linearity or homoscedasticity (see Appendix E). Also, correlational analyses were carried out to check for strong relationships with the BDI-II as the primary dependent variable in the study. A decision was made to use Factor 1 and Factor 3 as described in Bjornholdt (2006). These factors at face value conceptually were associated with clients' beliefs. Factor 1 as described by Bjornholdt (2006) is split into two categories including *benefits* (pleasure, mastery and progress) and *completion* (quantity and quality). As this study is interested in beliefs about homework rather than the actual quantity (or quality) of homework completed by the client, it was decided that this factor would only include the items *pleasure*, *mastery* and *progress*. Factor 3 was kept as described by Bjornholdt (2006). These two factors were used in the later MLM analysis. These factors were modeled as a *Level 1* (time variant) predictors. Because of practical and theoretical reasons the HRS-II measure could only be used from session 2. Items were multiplied together rather than summed to create an interaction (e.g., Comprehension x Rationale x Collaboration x Specificity x Match with therapy goals = 'Client's Beliefs'). Crossing or multiplying factors together to create interactions has the advantage that it increases external validity (Smith, 2000).

Table 8.2

Factor structure of the HRS-II (Bjornholdt, 2006)

Factor	HRS-II items	Description
Factor 1: 'Benefits and completion'	1	Quantity
	2	Quality
	10	Pleasure
	11	Mastery
	12	Progress
Factor 2: 'Costs and completion'	1	Quantity
	2	Quality
	3	Difficulty
	4	Obstacles
Factor 3: 'Client's beliefs'	5	Comprehension
	6	Rationale
	7	Collaboration
	8	Specificity
	9	Match with therapy goals

Data shape

It needed to be considered whether the assumption of linearity in the multilevel analyses was correct or whether transformations needed to be performed. Specifically, are the BDI-II data actually curvilinear in nature rather than linear as assumed? Singer and Willett (2003) suggest that the primary step in this consideration is to examine the descriptive graphs of each individual in the sample over time (these graphs are displayed in Chapter Nine). In addition to this, a curve fit analysis was carried out in SPSS. This analysis indicated that there was minimal difference between a linear, quadratic or cubic model fit in R-squared values. This suggests that all three may be appropriate to use. These results, taken into account along with the residual scatter plots (see Figure 8.2), which showed random, even scatter, suggestive of linearity lead to the decision that the data should not be transformed but rather kept in its original form. Singer and Willett (2003) assert that the complexity of a cubic transformation of the data may be disproportionate to the

improvement of the model fit. Therefore, keeping a linear form meant the results could be interpreted more clearly.

Reliability analyses

Alpha

Reliability analyses were carried out for the primary dependent measure (BDI-II) and other measures used in the study including the ASQ and the HRS-II. Reliability analyses were carried out at each time point for all of the measures (see Chapter Nine). As the HRS-II is a relatively new measure, it was especially important to check the reliability of this measure.

Preliminary analyses for multi-level model builing

Preliminary regression and correlation analyses

Initial regression analyses were performed that addressed, BDI-II scores over time, ASQ scores over time and HRS-II scores over time (factor scores). Each regression produced OLS-estimates of intercepts (constant) and of rates of change (Beta) for each individual client. The values produced allowed for correlations between variables and depression severity. Correlational analyses were performed between the BDI-II intercepts and rates of change as well as the HRS-II and ASQ intercepts and rates of change. The purpose of this correlational analysis was to help identify which parameters to include in the multi-level model.

Multilevel Modelling

A *multilevel model* represents a series of regression models that are interrelated to explain sources of variance from multiple levels of analysis (Hoffman & Rovine, 2007). Multilevel data analysis techniques involve collecting data from at least two sampling units that are hierarchically arranged in such that they are "nested" within one another (Affleck, Zautra, Tennen & Armeli, 1999). In multilevel modelling there are two levels of variables. *Level 1 (or lower) variables* which consist of repeated observations within persons. *Level 1 variables* are organised within *Level 2 units (or upper level variables)* which are the characteristics that differ between individuals in the study (Affleck et al., 1999). A distinguishing feature that multilevel models have is

that they account for the fact that responses from *Level 1* observations are not likely to be independent from *Level 2* units (Affleck et al., 1999; Singer & Willett, 2003).

Each regression equation in the multilevel model has a *fixed* portion, which describes the individuals 'true' trajectory (intercept and slope) over time at *Level 1* and also group-averages at *Level 2*. The regression equation also comprises a *random* component, which describes error associated with the equation (i.e., the variance that is still left unexplained). The random component accounts for the difference between the 'true' change trajectory and what is actually observed. These random components are reduced by adding predictors to later models which explain systematic error. Variables added to multilevel models that are associated with between person differences will reduce the variance at *Level 2*, whilst variables that are associated with within person change will reduce overall variance at *Level 1* (Singer & Willett, 2003).

Model A & Model B

Each multilevel model is first made up of an *unconditional means model (Model A).* The purpose of this model is to assess whether or not the baseline/primary dependent variable (i.e., BDI-II scores) contain enough variance at each level to justify this type of analysis. The model also can give an indication of where this variance is best explained (i.e., within people or between people). If there was not enough variation in this model, there would be no rationale for adding additional predictors to the model. Secondly, multilevel analysis includes an *unconditional growth model (Model B).* Model B introduces *time* (session) as a *Level 1* covariate. In this way the growth trajectory for repeated measurements over *time* are modelled on individual client score trajectories. The purpose of this analysis is to see whether the proposed model has enough variance to justify MLM analysis across time. After building these two models, subsequent models were developed that incorporated increasing numbers of predictors. In this study two separate multilevel models were developed to answer the research questions: *"What client factors are associated with early sudden gains in CBT for depression?"* ("MLM 1") and *"What are the within therapy change factors that are associated with early sudden gains in CBT for depression?"* ("MLM 2"). When considering how to order subsequent variables into these models it is considered best practice to base these decisions on theory (Singer & Willett, 2003; Wallace & Green, 2002).

Multilevel Model 1

Model 1 aims to answer the over all research question:

What are the client factors that may predict whether a client will obtain a sudden gain within CBT for depression?

MLM1 mainly focuses on *Level 2* variables as it asks questions about differences between clients that might moderate the relationship between the occurance of sudden gains and outcome in therapy. Sudden gains as a *Level 2* variable is entered first into MLM1 and is called 'Model C'. This variable is entered first as it is the main focus for the study and is predicted to be a very important factor in terms of change and improvement in BDI-II across therapy (Tang & DeRubies, 1999). The next variable that was entered into MLM1 as a *Level 2* variable is initial ASQ scores.

Abramson et al.'s (1989) 'hopelessness theory' predicted that those with hopelessness depression (or a depressogenic attributional style) would tend to have negative expectations about the occurrence of highly valued outcomes and an expectation that one would be helpless to change negative outcomes. Therefore, this theory assumes/predicts that those with a more depressogenic attributional style are more likely to have negative expectations about their ability to make improvements in therapy. Based on this theoretical rationale, this study hypothesised that those with more depressogenic attributional styles are less likely to make early rapid improvements and vice versa. Attributional style at intake is entered in at 'Model D' before other *Level 2* variables as one of the main tenants of the cognitive theory of depression is that *cognitions* are linked to the alleviation of depressed mood in depression (Beck et al., 1979; Garrett et al., 2007). Since the ASQ is measuring cognitive mechanisms, it is predicted that it will have a more significant effect that other variables (e.g., co-morbid status) and therefore is entered as 'Model D'.

The *Level 2* variable of 'co-morbid status' is entered last into MLM1 as the preliminary analysis showed little varibility in BDI-II change across therpay between those who have a co-morbid diagnosis at the start of therpay and those who do not. Therefore, this predictor is entered as 'Model E' last in MLM1.

Multilevel Model 2

MLM2 aims to answer the overall research question:

What are the within therapy factors that are associated with sudden gains within CBT for depression?

Multilevel Model 2 mainly focuses on *Level 1* variables as it asks about 'time variant' factors that may moderate the relationship between the occurrence of sudden gains and outcome in therapy. 'Model C' is identical in MLM2 as in MLM1 as sudden gains are the focus of the current study. Research suggests that depressogenic attributional style plays a significant role in the mechanisms of change in CBT for depression (e.g., Barber et al., 2005; DeRubeis et al., 1990; Peterson et al., 1982). The Attributional Style Questionnaire (ASQ) is based on Abramson et al.'s (1989) *hopelessness theory* of depression which theorises that cognitive mechanisms (specifically depressogenic attributional style) are linked to change in affect within therapy for depression. Attributional style is utilised as a *Level 1* variable in MLM2 as there is some research to show that attributional style is not a static state, but rather it has been shown to change across the course of therapy (e.g., Jarrett et al., 2007). Attributional style change across the course of therapy is entered at 'Model D' in MLM2 before other variables (i.e., 'homework beliefs' and 'homework progress') as there is greater empirical research and theoretical rationale to indicate that attributional style plays a more significant moderating role in the alleviation of depression in CBT.

The next *Level 1* variable to be entered into MLM2 is 'homework beliefs' (Factor Three as defined by Bjornholdt, 2006), as measured by the HRS-II. The hypothesis that 'homework beliefs' will moderate the relationship between the occurrence of sudden gains and outcome in therapy draws on theory that indicates that engagement in the process of homework is a reflection of clients' beliefs about the overall therapeutic process (Garland & Scott, 2005; Kazantzis & Daniel, 2009). Therefore, homework is seen to be a bridge between clients' experiences out of therapy and within therapy and an important intervention point for the therapist to understand and modify a clients belief's (Garland & Scott, 2005). The current study assumes that beliefs about homework provide greater insight to how clients' cognitions change across the course of therapy. 'Homework beliefs' is entered in MLM2 before

'homework progress' (Factor Two as defiend by Bjornholdt, 2006) as it is assumed to be a better reflection of a client's beliefs about the therapeutic process.

Components of the multilevel model

Within each multilevel model are *fixed effects* which provide several key estimates. The first estimate γ00 represents the intercept of the 'true' change trajectory of the grand mean across all occasions and all individuals. The next fixed effect γ01 represents the estimated differential in intercept for clients in different subgroups of the population. The subsequent fixed effect, γ02 represents the differential in the intercept for individuals in different subgroups, whilst controlling for other variables (Singer & Willett, 2003). The second set of fixed effects, γ10, γ11, γ12 provide estimates of the rate of change in clients' scores across time. γ10 represents a fixed estimate for the rate of change experienced by the average individual within the population, whilst the fixed estimate γ11 represents the differential between rates of change between different subgroups within the population. Correspondingly, γ12 is an estimate of the difference in rate of change for individuals in particular subgroups, whilst controlling for other variables.

Singer and Willett (2003) assert that one of the major reasons for fitting a multilevel model is to examine the random effects or *variance components* within the model. There are several variance components within a multi-level model for change ($\sigma^2\varepsilon$, $\sigma^2 0$, $\sigma^2 1$, $\sigma^2 01$). The *Level 1* variance component, $\sigma^2\varepsilon$ estimates the variance *within-individuals* across all occasions of measurement for all participants in the sample. The *Level 2* variance component, $\sigma^2 0$ represents the variance in true intercept *between-individuals* in the sample. The second *Level 2* variance component, $\sigma^2 1$ represents the residual variance in rate of change (or slope) *between-individuals*. Finally, *Level 2* covariance (between 'true' intercept and 'true' slope) across all occasions and all individuals in the sample is represented by the $\sigma^2 01$ estimate.

Also included in each multilevel model is a series of *Pseudo R* statistics and *Goodness of fit* estimates. These values enable one to compare subsequent models of 'nested' data to determine appropriateness of fit (Singer & Willett, 2003). *Pseudo R square* statistics ($R^2\varepsilon$, $R^2 0$, and $R^2 1$) are determined by calculating the percentage of change between models in terms of the variance components. If the variance components significantly decrease from one model to another, a higher proportion of the

variance will be explained, and this will be noted in the *Pseudo R* statistics. *Goodness of fit* statistics were obtained in the form of three different statistics in this study as recommended by Singer and Willett (2003). The first was a *deviance* statistic, -2Log Likelihood, which is a comparison of the likelihood of observing the sample data actually observed in the current model compared to a saturated or more general model (Singer & Willett, 2003). The second is the *Akaike Information Criterion (AIC;* Akaike, 1973) which adjusts the deviance statistic for the number of parameters in the model. Finally, the *Bayesian Information Criterion (BIC;* Schwarz, 1978) makes adjustments for the sample size as well as the number of parameters in the model (Singer & Willett, 2003). When these values decrease in subsequent models, it represents a greater model fit.

Summary

This chapter has outlined the advantages of utilising multilevel modelling as the primary analytic strategy for the current study. It provides a summary of how the data was treated and outlines the preliminary data analytic techniques that were utilised. This chapter then introduced the multilevel modelling process and described how and why each of the predictor variables was entered into the models. The following chapter will present the results of the multilevel model analysis.

CHAPTER NINE

RESULTS

Introduction

This chapter presents the main results from the multilevel analyses. Before presenting the results from each of the models, a number of preliminary analyses are introduced. First of all, the results from the reliability analyses are presented. Secondly, an assessment of the amount of variance found in the data set is carried out. This entailed introducing each of the primary variables to see whether they showed any significant variation both within clients and between clients over time. Once it was confirmed that there was an appropriate amount of variability to justify further investigation a number of preliminary correlation analyses were performed to introduce the need for multilevel analyses and to highlight significant relationships between variables. Visual analysis of *Level 2* variables was also carried out to help confirm the variability within the data set. These helped to identify which parameters would be appropriate to include in the subsequent models. Finally, the results from the multilevel analysis are presented and each model was reviewed.

Preliminary analyses

Reliability checks

An important initial step is to perform reliability analyses on the measures for the primary variables in the study (i.e., BDI-II, ASQ and HRS-II). This step was critical to ensure that all subsequent interpreted results were well supported and accurate. Therefore, confirming the reliability of the measures used in this study helps support the integrity of the results from this study.

Reliability of the BDI-II

Previous studies have demonstrated that the BDI-II has good internal consistency ($\alpha = .92$) in both clinical and non-clinical samples (Beck et al., 1996). However, it is important to reconfirm this within the context of this study as it was used as the primary dependent variable. To perform the reliability analysis, each session of the BDI-II data was separated into individual data sets across each session and the SPSS reliability analysis function was used. The results from this analysis demonstrate

excellent reliability based on thresholds of acceptability (Nunnally, 1978). These results are outlined in Table 9.1. It can be seen that the BDI-II had an average alpha of .94 and a range of .91 to .96 across all sessions.

Table 9.1

Reliability analysis of the BDI-II across all sessions of therapy

Session	N	A	M	SD
0	28	.92	31.0	11.1
1	28	.93	27.1	11.5
2	28	.93	25.0	11.1
3	28	.95	22.1	12.1
4	28	.95	24.2	11.9
5	28	.96	21.3	13.4
6	28	.94	19.1	11.0
7	27	.95	19.3	11.9
8	27	.95	18.4	12.0
9	27	.94	19.5	12.3
10	26	.94	17.5	11.8
11	26	.93	16.6	10.9
12	25	.93	19.1	10.9
13	25	.93	15.7	10.4
14	23	.94	15.6	10.7
15	22	.94	16.0	11.1
16	22	.93	15.2	10.7
17	21	.92	15.1	9.6
18	21	.91	14.3	8.8
19	20	.94	12.6	10.7
20	18	.91	11.3	8.9

Reliability of the ASQ

Reliability analyses were carried out on the ASQ, more specifically the constructs that make up positive attributional style (CoPos) and negative attributional style (CoNeg). Table F1 can be found in Appendix F which outlines the alpha reliability values across four sessions of therapy. Table F1 shows that the alphas for the negative attributional style construct (CoNeg) were all adequate, above α=.67. However, the alpha values did decrease over time. The alpha values for the positive attributional style construct (CoPos) were not as strong as for the CoNeg construct. However, they did remain over α=.54 and they did improve over time.

Reliability of the HRS-II (client version)

As the HRS-II is a fairly new measure, it was important to reconfirm the reliability of the measure within the specific context of this study. As demonstrated in Table F2 (which can be found in Appendix F), the alpha values for the HRS-II client versions were adequate across time ranging from α=.61 to .93. This indicates that the 12 items that make up the client version of the HRS-II cohere in a way that indicates that they are measuring the same construct.

Assessment of vairance within primary variables

In order for MLM analysis to be effective, there needs to be enough variance within the data set for results to be significant. Therefore, an essential step in the preliminary analysis was to *assess the variance* within the primary variables that were utilised within this study. When an appropriate level of variance is identified within each parameter it helps to confirm the appropriateness of multi-level analysis.

Depression severity – BDI-II

Within the current study, depression severity was utilised as the primary dependent variable and this was measured by the BDI-II. Depression severity scores were collected at intake and before each session of therapy. *Hypothesis One* of this study predicted that clients' depression severity scores on average would decrease throughout therapy. An outline of the change in depression scores across clients is presented in Appendix G (Table G1). Figure 9.1 demonstrates the population average change in depression across 20 sessions of therapy.

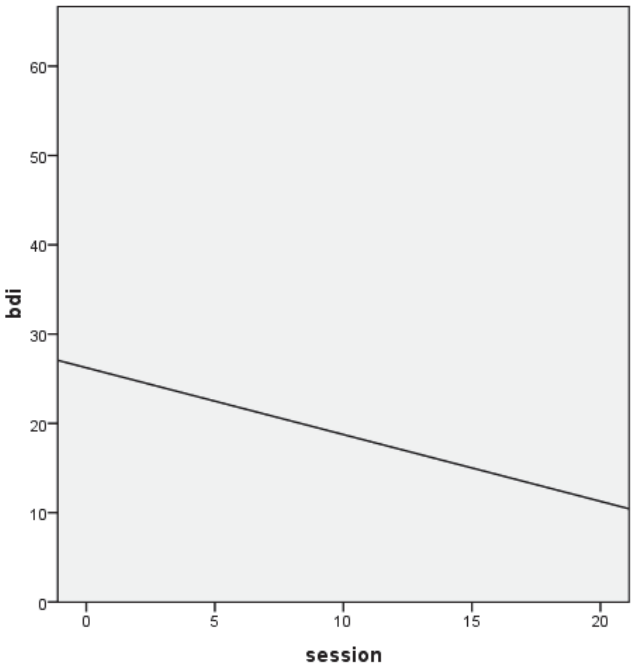

Figure 9.1: Average change trajectory of depression severity across therapy sessions

As can be seen in Figure 9.1 and Table G1 (see Appendix G), a clear pattern of improvement is demonstrated in the current study. All clients (N = 28) improved in their BDI-II scores from intake to the end of therapy. On average clients had a 67.6% decrease in depression severity from intake to the end of therapy, with a range of 22% to 100% improvement. It was shown that these gains were maintained at two-month and six-month follow up sessions. At the two-month booster session clients had improved on average 72% from the intake session (with a range of 33% to 98%) and at six-month follow up clients had improved on average 70% from the intake session (with a range of 8% to 100%).

The above results have demonstrated that on average depression severity improved over therapy sessions. This study was also interested in the individual differences in initial severity levels and change trajectories. One of the primary considerations when investigating how individuals change over time is to examine each of their individual growth plots as they are able to reveal a great deal of information about how each individual changes over time (Singer & Willett, 2003). Figure 9.2 highlights the individual differences in change trajectories across clients.

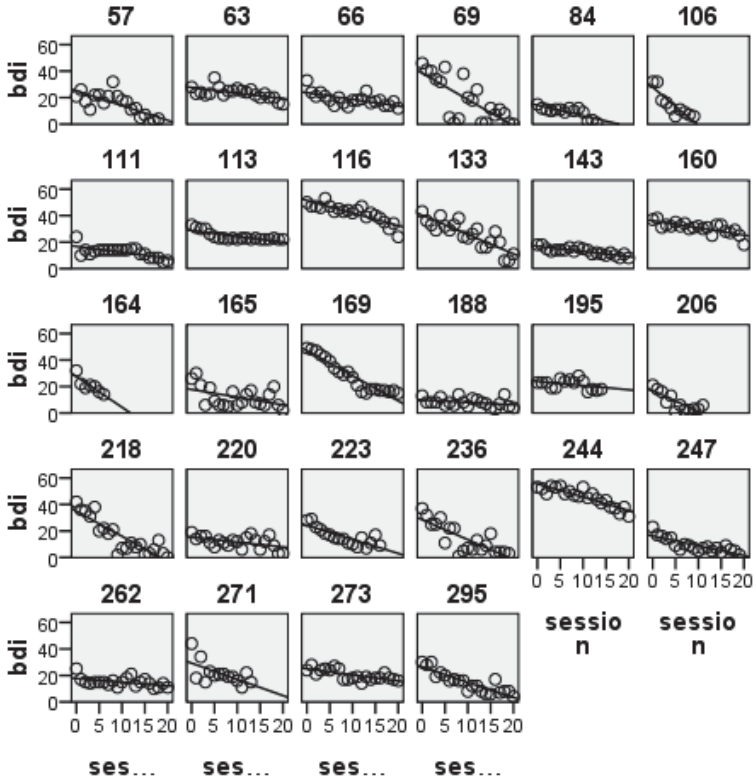

Figure 9.2: Fitted ordinary least squares (OLS) trajectories for each clients BDI-II scores across 20 sessions of therapy.

Figure 9.2 showed that all clients within the study had a reduction in depression severity across treatment. Thus, providing additional evidence that BDI-II scores vary across time. Upon visual inspection of each individual trajectory it is evident that there is individual variance between initial intake scores and rates of change in depression severity. Therefore these analyses provide evidence of within-individual and between-individual variance in BDI-II scores across therapy. The fact that this variability exists provides support for subsequent multi-level analysis and the exploration of additional predictors that may explain this variance. Furthermore, these analyses, which demonstrate both average and individual change trajectories, taken together confirm that the null hypothesis for *Hypothesis One* can be rejected.

Attributional Style –ASQ

Attributional style was measured by the ASQ across different time points in therapy (i.e., intake, session 5, session 8, session 20, 2-month follow-up and 6-month follow-up). Composite scores were used in this analysis to explore change in attributional

style across four time points in therapy (i.e., intake, session 5, session 8 and session 20). Figure 9.3 presents the average change in initial ASQ scores and rate of change across four time points in therapy. Figure 9.4 presents the individual change trajectories of ASQ across four time points in therapy.

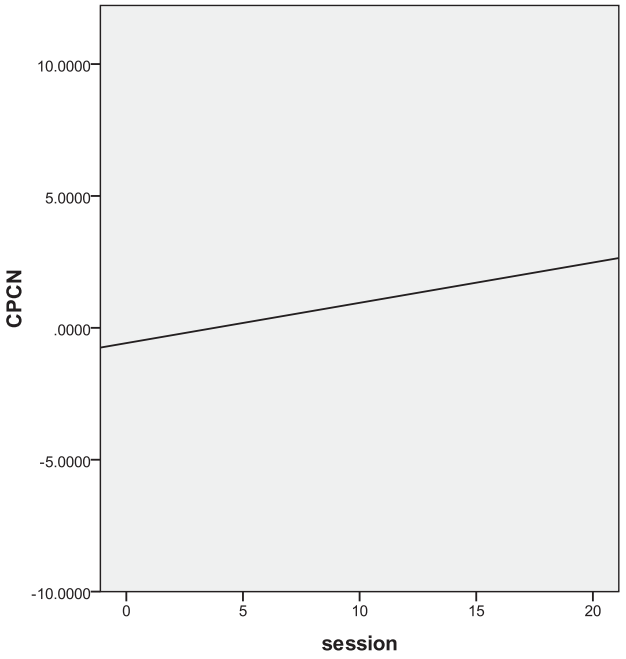

Figure 9.3: Average ASQ scores across four time points in therapy

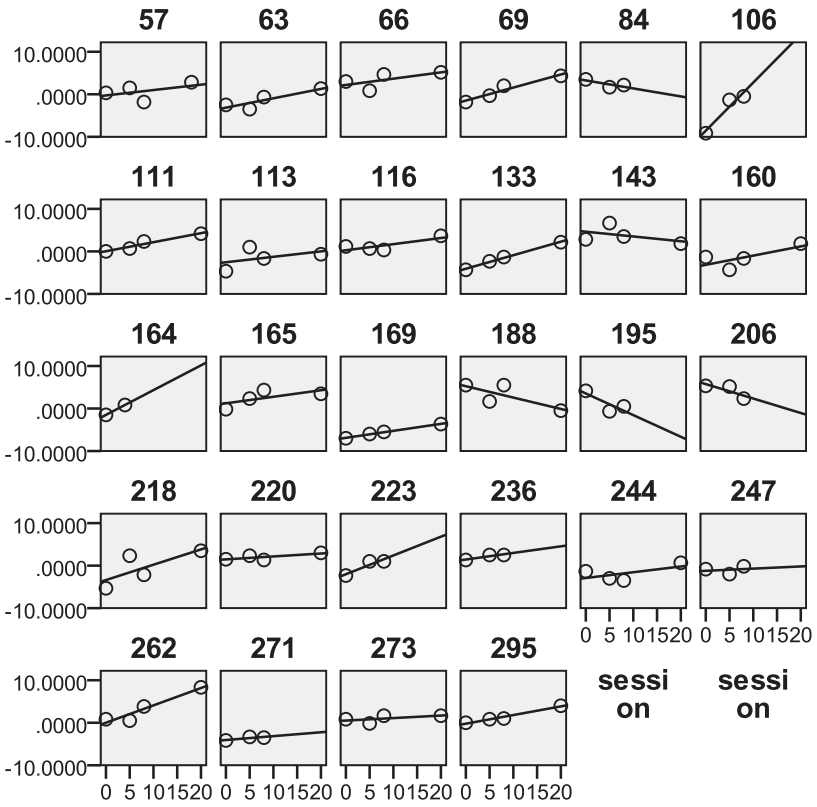

Figure 9.4: OLS trajectories of attributional style across four time points in therapy

Figure 9.3 demonstrates that on average clients attributional style tends to improve within therapy. Upon visual inspection of the individual client trajectories in Figure 9.4 one can see that a majority (82%) of clients' attributional style improves across therapy. However, it is clear that there were individual differences in both the initial ASQ scores at intake and the rate of change across therapy between clients. These results taken together indicate that there is adequate variation both within clients across therapy and between clients within the sample to warrant multi-level investigation.

Homework – HRS-II

Homework was measured by the HRS-II at the beginning of each therapy session (from session two onwards). Raw HRS-II (client version) total scores across time for each individual client are represented in Figure 9.5. The data do not appear to display any clear linear trends overtime. Scores appear to fluctuate rather than stay constant across time for individual clients. This indicat that there is variance in between-client and within-client trajectories across time.

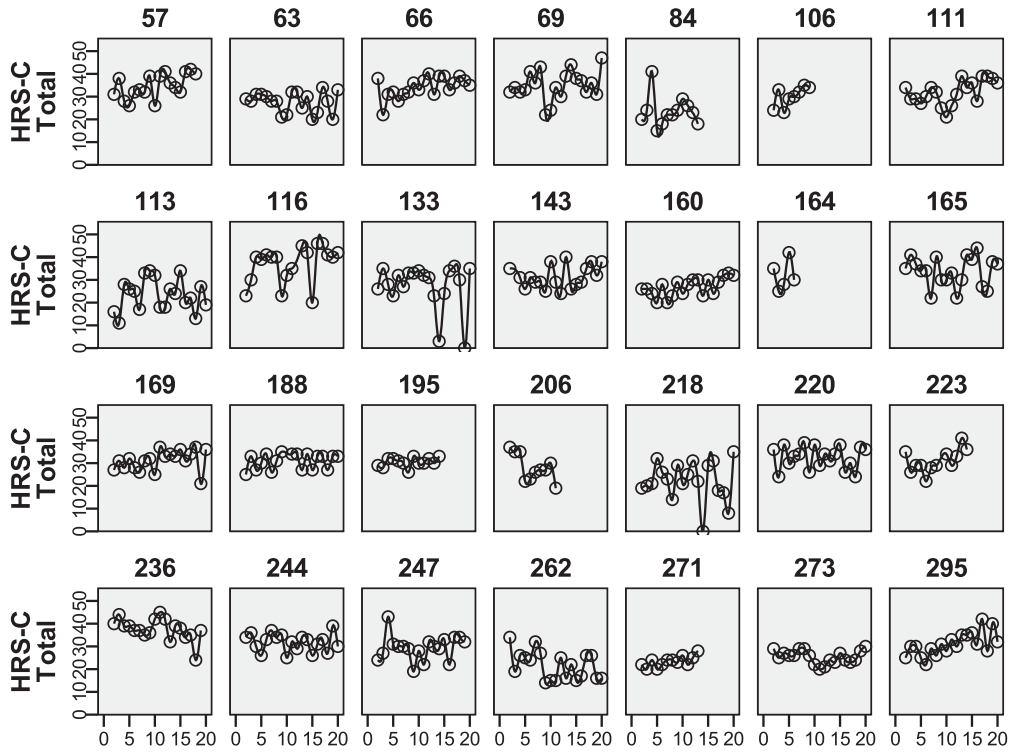

Figure 9.5: Raw score individual client trajectories of the total HRS-II (client version) scores across 18 time points in therapy (session two to session twenty)

Correlation analyses

The preceding analyses confirmed that a significant amount of variance existed within the data gathered from the primary variables used within the current study. Because there is a significant amount of variability within the data set it suggests that the introduction of potential predictors, at both *Level 1* and *Level 2* is justified. Preliminary correlation analyses have been carried out to help determine which parameters to include in the later multi-level model(s). Regression analysis which uses OLS-estimates of intercepts and rates of change was used to test whether there was any significant interaction between the predictor variables and the BDI-II. By using the estimated slopes and intercepts for each variable, bivariate correlations were carried out. The results in Table 9.2 highlights the relationships that existed between time-variant predictors used in the study. Results in Table 9.5 highlight the relationships that existed between some of the variables at the 90% level of significance.

Table 9.2

Intercept/Slope correlations between the main dependent time-variant variables used in the current study

	BDI-II α	BDI-II β	ASQ α	ASQ β	HWF3 α	HWF3 B	HWF2 α	HWF2 β
BDI-II α	-	**-.405* .032**	**- .600** .001**	.271 .163	**.331~ .086**	.117 .552	-.078 .693	.065 .743
BDI-II β		-	**.525** .004**	**- .560** .002**	-.230 .239	.077 .696	.142 .470	**-.336~ .081**
ASQ α			-	**- .740** .000**	.131 .506	-.153 .437	.209 .287	-.118 .549
ASQ β				-	-.012 .952	.060 .761	-.228 .243	.314 .104
HWF3 α					-	**.469* .012**	**.382* .045**	.060 .763
HWF3 β						-	-.115 .562	-.032 .873
HWF2 α							-	**.706** * .000**
HWF2 β								-

~ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$ significance level (2 tailed Pearson correlation). Significant correlations are in bold; α represents 'initial status', β represents 'rate of change'.

Table 9.2 demonstrates that several significant relationships existed between the predictor variables and the BDI-II. A significant negative relationship was found between the initial status of the BDI-II and the initial status of the ASQ ($p < .01$), suggesting that those clients who present more depressed at intake typically had a more depressogenic (or negative) attributional style. Furthermore, a significant positive relationship was observed between initial attributional style status and the rate of change in BDI-II ($p < .01$), indicating that a clients that present with more or less depressogenic attributional styles experience significantly different rates of recovery in BDI-II across therapy. A significant negative relationship was found between the rate of change in BDI-II and the rate of change in ASQ across the course of therapy ($p < .01$). This indicated that those clients who experienced a faster

rate of change in BDI-II (i.e., reduction in depression severity) also demonstrated a faster rate of change in attributional style (i.e., less depressogenic). The relationship demonstrated between the BDI-II initial status and rate of change were less significant when exploring their relationship with the homework variables. However, there was a significant positive relationship ($p<.10$) between BDI-II initial status and 'homework beliefs' (homework factor three) initial status. Furthermore, a significant negative relationship ($p<.10$) was found between BDI-II rate of change and 'homework progress' (homework factor two) rate of change. This indicates that those clients who experienced a faster rate of change in BDI-II also experienced a faster rate of change in 'homework progress' (i.e., they believed that they were increasing their progress in homework) across the course of therapy. Additionally, there was a significant positive relationship ($p<.05$) between the initial status of 'homework beliefs' and 'homework progress', indicating that those who have more positive beliefs about homework at the start of therapy are likely to concurrently have more positive beliefs about how they are progressing in homework.

Preliminary visual analysis for level-2 predictor variables

Visual inspection of difference in trajectories between those who experience sudden gains and those who do not

Preliminary correlational analysis indicated that attributional style interacted with depression severity significantly. Furthermore, there was some relationship demonstrated between depression severity and 'homework beliefs' and 'homework progress'. The next step was to explore whether these time variant predictors had a significant relationship with the *Level 2* predictor of sudden gains as this was the focal point of the current study.

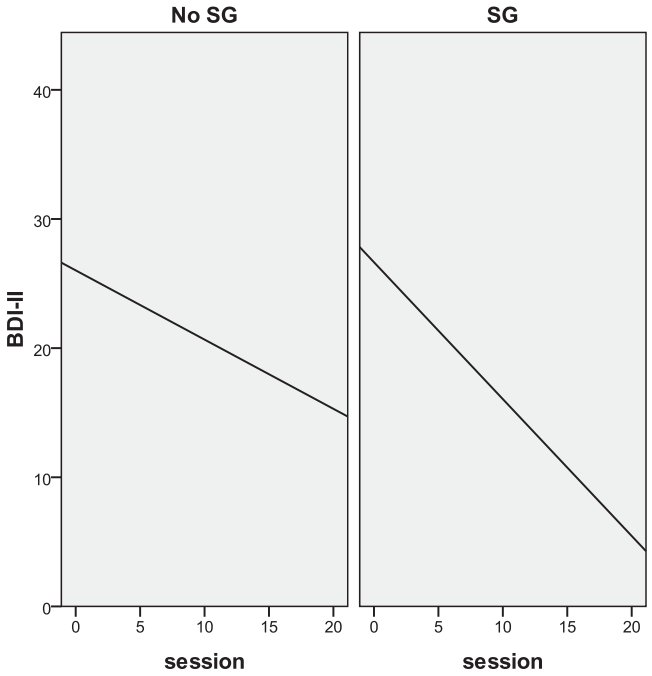

Figure 9.6: Differences in average BDI-II trajectories for clients who experience sudden gains and those who do not experience sudden gains[4]

Visual inspection of Figure 9.6 indicates that there is minimal difference in initial status between the group of clients who experience sudden gains and those who do not. However, these figures appear to demonstrate a difference between fitted rates of change across therapy sessions, with those who experience a sudden gain on average more likely to have a more rapid recovery curve and lower overall depression severity at the end of treatment compared to those who do not experience sudden gains.

[4] *NB:* Average is calculated across all clients presenting for therapy at the particular session

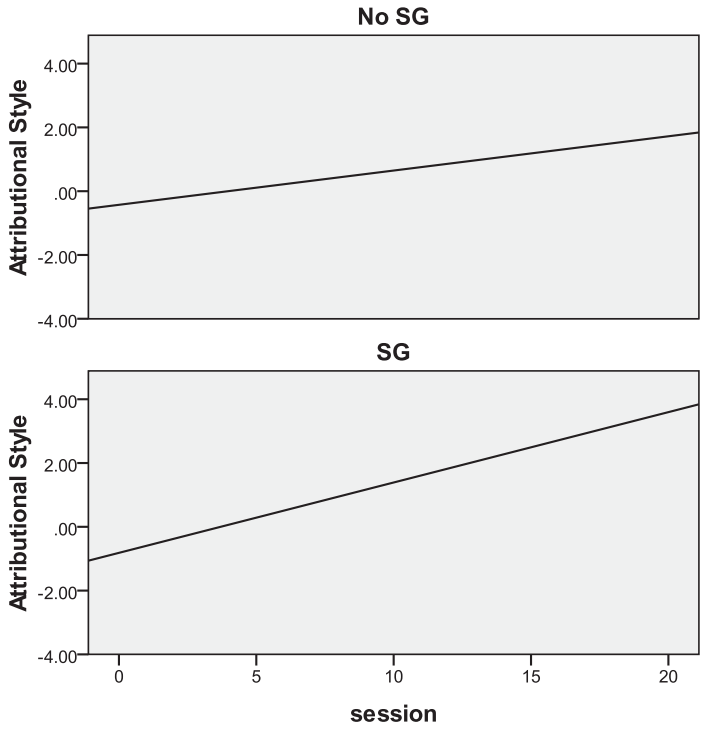

Figure 9.7: Differences in average ASQ trajectories for clients who experience sudden gains and those who do not experience sudden gains[5]

Visual inspection of Figure 9.7 indicates that there appears to be a difference in ASQ change trajectories between those clients who experience sudden gains and those who do not. There appears to be little difference in initial status of ASQ between those clients who experience a sudden gain and those who do not. However, there does appear to be a clear difference in the rates of change between the *Level 2* subgroups. With those clients who experienced a sudden gain on average more likely to have a more rapid improvement in ASQ across the course of therapy compared to those who did not experience a sudden gain. It also appears that at the end of therapy clients who experienced sudden gains were more likely to terminate with a less depressogenic attributional style than those who did not experience a sudden gain.

[5] NB: 'SG' represents 'sudden gains'

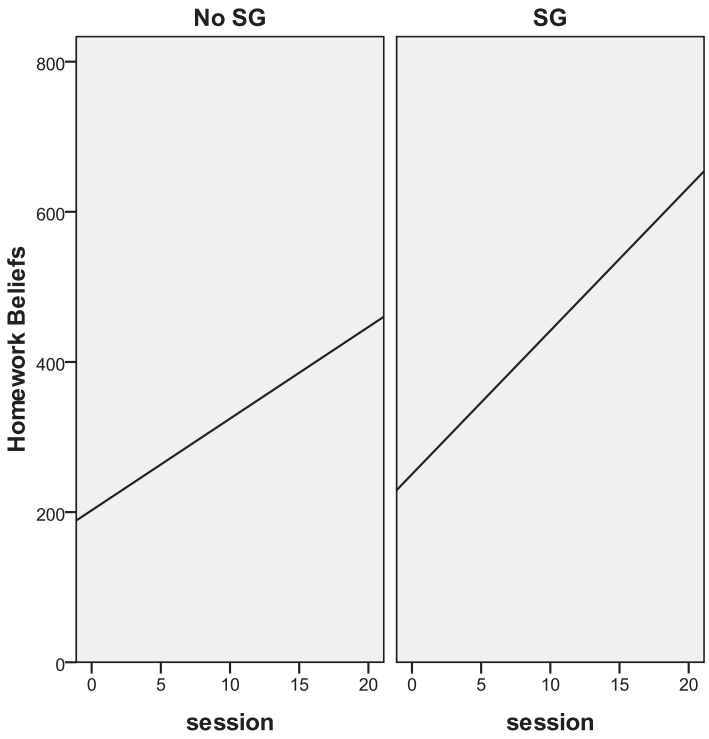

Figure 9.8: Difference in average trajectories of 'homework beliefs' (homework factor three) for clients who experience sudden gains and those who do not experience sudden gains.

Visual trajectories of 'homework beliefs' across therapy show that for both *Level 2* subgroups homework beliefs tend to increase across therapy. Whilst the 'homework beliefs' intercept did not appear to differ significantly between the subgroups, clients on average who experienced sudden gains appeared to demonstrate a higher rate of change and more improvement in 'homework beliefs' across therapy. Additionally, those clients who experienced sudden gains appeared to 'believe' in the process of homework more at termination of therapy than those clients who did not experience a sudden gain.

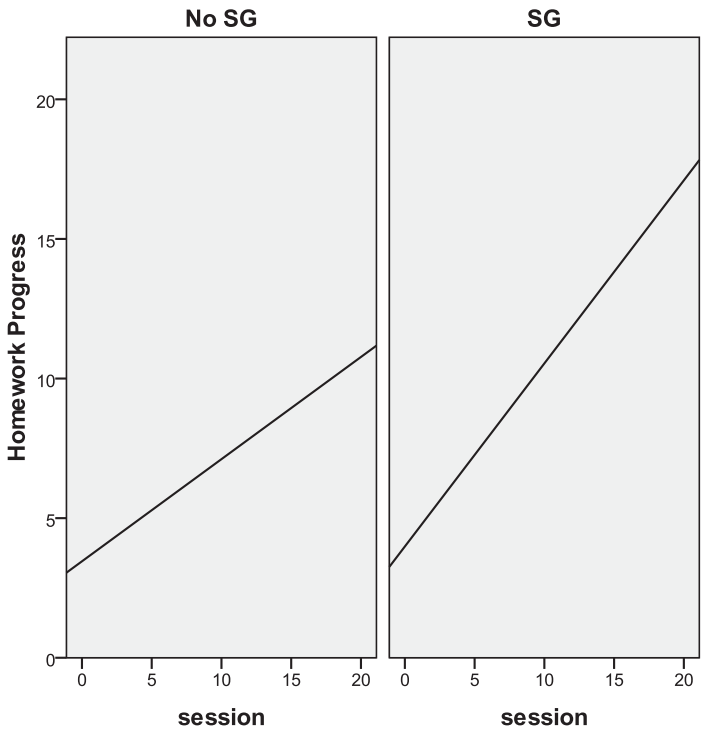

Figure 9.9: Differences in average 'homework progress' (beliefs about progress in homework) for client who experience sudden gains and those who do not

Visual trajectories of 'homework progress' across therapy show that for both *Level 2* subgroups clients' beliefs about their progress with homework tends to increase across therapy. Whilst the 'homework progress' intercept did not appear to differ significantly between the subgroups, clients on average who experienced sudden gains appeared to demonstrate a higher rate of change and more improvement in 'homework progress' across therapy. Additionally, those clients who experienced sudden gains appeared to believe in their progress with homework more at termination of therapy than those clients who did not experience a sudden gain.

Visual inspection of differences in trajectories between clients who have co-morbid psychiatric diagnoses at intake and those who do not

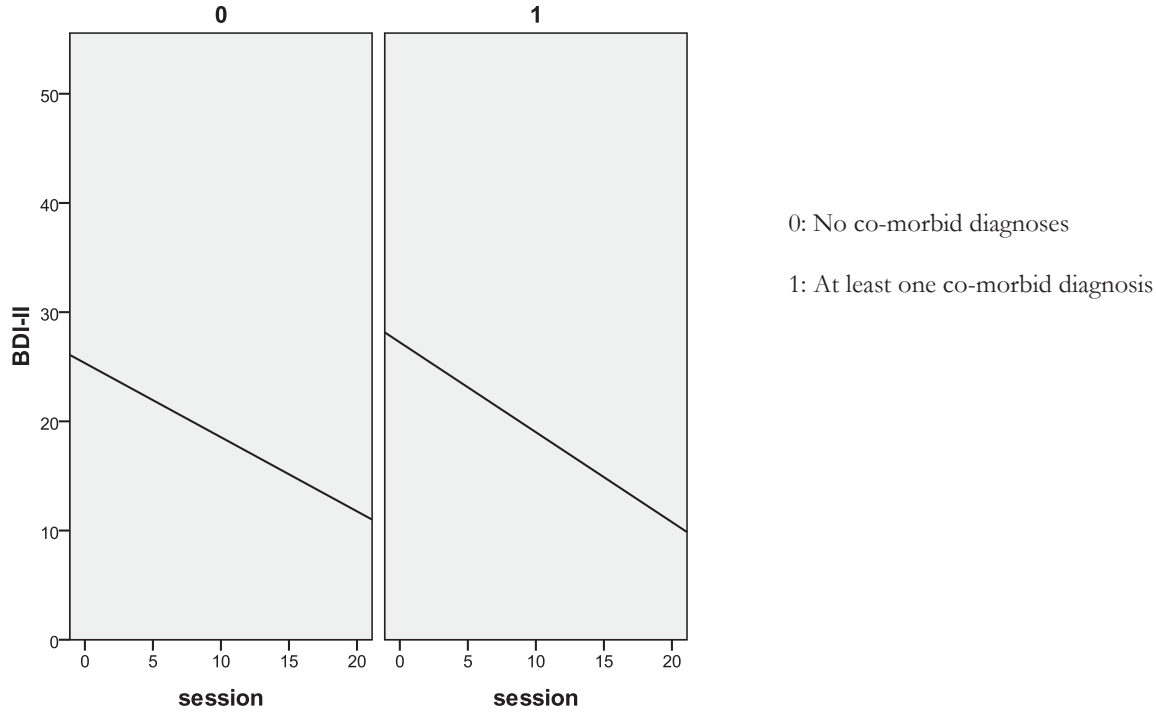

Figure 9.10: Differences in average BDI-II trajectories for clients with co-morbid diagnoses and those with no co-morbid diagnoses

Figure 9.10 indicates little difference between both the initial status and rate of change in BDI-II (depression severity) for this *Level 2* subgroup. This indicates that there is likely to be little variance indicated within the multi-level model and therefore may not be a significant predictor of variance in initial status or rates of change for those with and without co-morbid diagnoses.

Visual inspection of the empirical growth plots for the *Level 2* subgroup 'sudden gains' showed that across all four time-variant dependent variables (i.e., BDI-II) and predictors (i.e., attributional style, 'homework beliefs', 'homework progress') appeared to demonstrate that there was variance in the data between clients who experienced sudden gains and those who did not. Furthermore, these preliminary analyses indicated that on average the clients who experienced sudden gains appeared to have greater rates of recovery and better outcomes at the end of treatment. This taken with other preliminary data indicated the importance of further analysing the relationships between these factors.

Multi-level model building

'Multilevel Model 1: Sudden gains and client factors' and *'Multilevel Model 2: Sudden gains and within therapy factors'* are presented separately in this study as they are answering two separate questions. However, these models share the same initial steps in model building as they are both concerned with depression severity and sudden gains. Therefore, Model A, Model B and Model C within Multilevel Model One and Multilevel Model Two will be identical. Model A, B and C will be described initially, and then Model D onwards will be described separately for Model 1 and Model 2.

Models A & B

Models A and B represent the *unconditional means model* and the *unconditional growth model* and they are presented in Table 9.3 below.

Table 9.3

Unconditional means model and unconditional growth model for 'Model One' and 'Model Two'

	Parameter		Model A	Model B
Initial status, $\pi 0i$	Intercept	$\gamma 00$	18.94*** (1.71)	26.98*** (2.13)
Rate of Change, $\pi 1i$	Intercept	$\gamma 10$		-.935*** (.111)
Level 1	Within person	$\sigma^2\varepsilon$	59.73*** (1.71)	23.03*** (1.51)
Level 2	Initial status	$\sigma^2 0$	78.78*** (21.87)	122.58*** (33.90)
	Rate of change	$\sigma^2 1$		.288** (.094)
	Covariance	$\sigma 01$		-2.76* (1.34)
	$R^2\varepsilon$			0.614
	Deviance		3734.2	3321.8
	AIC		3740.2	3333.8
	BIC		3753.0	3359.4

~ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$ significance level (2 tailed Pearson correlation). Significant correlations are in bold; α represents 'initial status', β represents 'rate of change'.

Fixed effects

Within each multilevel model are *fixed effects* which provide several key estimates. The estimate γ00 in Model A is 18.94 (p <.001) and confirms that the average BDI-II score at intake is non-zero and significant. The same estimate in Model B is 26.98 (p<.001) also confirms that the average BDI-II score is non-zero and significant when taking into account the structure of time. In Model B the fixed effects of γ00 and γ10 estimate the starting point and the slope of the population average change trajectory in depression severity as measured by the BDI-II. These parameters estimate that the true average change trajectory for depression severity has a non-zero intercept of 26.98 (p<.001) and a non-zero slope of -.935 (p<.001), indicating that on average BDI-II scores decrease over time.

Variance components

One of the major reasons for fitting a MLM is to examine the random effects (Singer & Willett, 2003). The *unconditional means model* (Model A) shows significant positive variance components at both *Level 1* and *Level 2* ($\sigma^2\varepsilon$ and $\sigma^2 0$). This indicates that there is variance to be explained both within-clients and between-clients. Both these estimates are significant at the p<.001 level. The variance components in the *unconditional growth model* (Model B) demonstrate how much variance the added variable of 'time' (sessions of therapy) has explained. Table 9.6 shows that the $\sigma^2\varepsilon$ value has dropped from Model A to Model B at *Level 1,* which indicates that time explains some of the variance between people; 61.4% of the variance between-people is explained by adding the predictor of time (see pseudo R-square value, $R^2\varepsilon$). Because there is still some variance left to be explained at *Level 1* it indicates that it could be useful to add *Level 1* predictors to the model. The $\sigma^2 0$ value shows that the *Level 2* variance has increased from Model A to Model B. This was to be expected (Singer & Willett, 2003). The $\sigma^2 0$ value taken with the $\sigma^2 1$ value in Model B summarise both the within person variance in initial status of depression severity and the rate of change in depression severity across therapy sessions. Both of these values are positive, non-zero and significant (p<.001 and p<.01). It suggests that it is also useful to utilise *Level 2* predictors to help explain the heterogeneity between initial status and rate of change. The population covariance of the *Level 2* residuals, $\sigma^2 01$, assesses the relationship between $\sigma^2 0$ and $\sigma^2 1$. Therefore, it quantifies the

population co-variances between the 'true' initial status and the 'true' rate of change. The negative and significant covariance statistic in Model B indicates that those who have a higher BDI-II score at intake experience slower rates of recovery over time.

Goodness of fit statistics

The deviance statistics in Model A and B allow us to compare models of 'nested' data. From Model A to Model B this value reduced by 412.4, therefore demonstrating that Model B is a superior fit to Model A. Furthermore, the AIC value (which takes into account the number of parameters within a model), decreased by 406.4 and the BIC value (which takes into account sample size) decreased by 393.6. These values provided further evidence that Model B is a superior fit to Model A.

Model C – Introducing Sudden Gains

Model C is shown in both Table 9.4 and Table 9.5 and introduces the uncontrolled effects of sudden gains as a *Level 2* predictor. Clients who did not experience a sudden gain were given a value of "0" and clients who did experience a sudden gain were given a value of "1".

Fixed effects

The four estimated fixed effects of Model C can be interpreted as follows. $\gamma 00$ estimates the BDI-II score at intake for clients who do not experience a sudden gain (26.57; $p<.001$). $\gamma 01$ represents the estimated differential in the estimated intake BDI-II between those clients who experience a sudden gain and those do who not. This value was indistinguishable from zero (.852; *ns*). $\gamma 10$ represents the estimated rate of change in BDI-II for clients who did not experience a sudden gain. This value was negative and non-zero (-.666; $p<.001$). The final fixed estimate $\gamma 11$, represented the differential in the rate of change of depression severity between clients who did not experience sudden gains and those who did. The value was negative and significant (-.593; $p<.01$).

Variance components

The $\sigma^2\varepsilon$ value that represents within person variance indicated that there was no real change in *Level 1* variance and it actually increased slightly. However, this was to be expected as no *Level 1* predictors were added (Singer and Willett, 2003). *Level 2,* or between-person residual variance in initial status as represented by the σ^20 value, increased slightly from Model B. However, this value was still non-zero and

significant (123.01; p<.001), indicating that there is still room for more predictors in the model to help explain between-person variance in initial depression severity besides sudden gains. *Level 2* residual variance in depression severity rate of change however did reduce significantly (.196; p<.01) and represented a 31.9% reduction from Model B. This indicates that whether a client experiences a sudden gain or not explains 31.9% of the variance in the rate of change in depression severity in this study. From the results of Model C it could be concluded that the effects of other predictors should be explored as a significant amount of both *Level 1* and *Level 2* variance remains within the model.

Goodness of fit statistics

There was a small reduction in the deviance statistic (10.7) and the AIC (6.7) indicating that Model C is a slightly superior fit to Model B.

Multilevel Model One: Sudden gains and client factors

Table 9.4

Results of fitting a multi-level model to BDI-II data that accounts for sudden gains, initial attributional style and co-morbid presentation: 'Model One: *sudden gains and client factors'*

	Parameter		Model A	Model B	Model C	Model D	Model E
Initial status, $\pi 0i$	Intercept	$\gamma 00$	18.94***	26.98***	26.57***	26.33***	26.66***
			(1.71)	(2.13)	(2.82)	(2.46)	(2.95)
	Sudden gain	$\gamma 01$	-	-	.852	-.678	-.729
			-	-	(4.31)	(3.80)	(3.79)
	Attributional	$\gamma 02$	-	-	-	-1.625**	-1.647**
	Style		-	-	-	(.538)	(.544)
	Co-morbid	$\gamma 03$	-	-	-	-	-.771
	Presentation		-	-	-	-	(3.78)
			-	-	-	-	-
			-	-	-	-	-
	Intercept	$\gamma 10$	-	-.935***	-.666***	-.648***	-.754***
			-	(.111)	(1.23)	(.098)	(.117)
Rate of Change, $\pi 1i$	Sudden Gain	$\gamma 11$	-	-	-.593**	-.514**	-.524**
			-	-	(.189)	(.152)	(.182)
	Attributional	$\gamma 12$	-	-	-	.096***	.103***
	style		-	-	-	(.023)	(.023)
		$\gamma 13$	-	-	-	-	.228
	Co-morbid		-	-	-	-	(.148)
	Presentation		-	-	-	-	-
			-	-	-	-	-
Level 1	Within	$\sigma^2\varepsilon$	59.73***	23.03***	23.07***	23.01***	22.95***
	person		(1.71)	(1.51)	(1.51)	(1.50)	(1.50)
Level 2		$\sigma^2 0$	78.78***	122.58***	123.01***	92.51***	91.84**
	Initial status		(21.87)	(33.90)	(34.02)	(25.91)	(25.71)
		$\sigma^2 1$	-	.288**	.196**	.108**	.100**
	Rate of		-	(.094)	(.065)	(.041)	(.038)
	change	$\sigma 01$	-	-2.76*	-2.70*	-.993	-.883
			-	(1.34)	(1.17)	(.762)	(.738)
	Covariance						
	$R^2\varepsilon$		-	0.614	0.614	0.615	.616
	$R^2 0$		-	-	-.000	.245	.251
	$R^2 1$		-	-	.319	.625	.653
	Deviance		3734.2	3321.8	3311.1	3296.0	3293.6
	AIC		3740.2	3333.8	3327.1	3316.0	3317.6
	BIC		3753.0	3359.4	3361.2	3358.7	3368.7

~ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$ significance level (2 tailed Pearson correlation). Significant correlations are in bold; α represents 'initial status', β represents 'rate of change'.

Model D

Table 9.4 represents the results from fitting a multi-level model to explore client factors that are associated with sudden-gains and recovery from depression. Model D represents the controlled effects of a clients ASQ score (attributional style) at intake. Model D evaluated the effects of whether a client experiences a sudden gain or not on BDI-II initial status and rate of change, whilst controlling for the effects of attributional style score at intake as a *Level 2* predictor.

Fixed effects

The estimated fixed effects of Model D can be interpreted as follows. γ00 indicates the average BDI-II score at intake when clients are scoring '0' on both sudden gains and the ASQ (26.33; $p<.001$). γ01 represents the estimated differential in the estimated intake BDI-II between those clients who experience a sudden gain and those do who not. This value remained indistinguishable from zero (-.678; *ns*). γ02 showed a negative non-zero relationship (-1.625, $p<.01$) this demonstrates that after controlling for sudden gains, initial BDI-II scores are negatively related to attributional style at intake (i.e., if BDI-II scores are higher at intake, attributional style will be more depressogenic). γ10 represents the estimated rate of change in BDI-II for clients who did not experience a sudden gain and who scored '0' on the ASQ construct at intake. This value was negative and non-zero (-.648; $p<.001$) indicating a negative relationship with BDI-II scores. γ11 represented the differential in the rate of change of depression severity between clients who did not experience sudden gains and those who did whilst controlling for the effects of attributional style. γ11 decreased from -.593 in Model C to -.514 in Model D ($p<.01$). This indicates that at least some of the significant differential that was found between clients who obtain sudden gains and those who do not in Model C may be partially explained by their attributional style at intake. In other words, Model D provides evidence to suggest that attributional style at intake moderates the relationship between whether a client experiences a sudden gain and their improvement in depression severity across the course of treatment. γ12 indicates that after controlling for sudden gains, attributional style is positively associated with the

rate of change in depression severity (.096, $p<.001$). Therefore people with higher ASQ scores at intake are likely to experience a faster rate of change.

Variance components

At *Level 2* the inclusion of attributional style to the model led to a significant non-zero decrease in the between-person variance for initial status (92.51; $p<.001$). Therefore attributional style at intake explains 24.5% of the variance in BDI-II scores at intake as measured by the $R^2 0$ value. Additionally, at *Level 2* the inclusion of attributional style led to a significant non-zero decrease in the between-person variance in the rate of change of the BDI-II across therapy (108; $p<.01$). Therefore, Model D accounted for 62.5% of the variance in change trajectories between-persons as measured by the $R^2 1$ value.

Goodness of fit statistics

Overall the deviance statistics reduced from Model C to Model D, which indicates when clients' attributional style at intake is controlled for a greater level of variance was explained and improved the model fit.

Model E

Results from Model E are described in Table 9.4. Model E evaluated the effects of whether a client experiences a sudden gain or not on BDI-II initial status and rate of change, whilst controlling for the effects of a co-morbid presentation as a *Level 2* predictor at intake.

Fixed effects

The estimated fixed effects of Model E can be interpreted as follows. γ00 indicates the average BDI-II score at intake when clients are scoring '0' on sudden gains, the ASQ and '0' for co-morbid presentation (i.e., no co-morbid presentation at intake) (26.66; $p<.001$). γ01 represents the estimated differential in the estimated intake BDI-II between those clients who experience a sudden gain and those do who not. This value remained indistinguishable from zero (-.729; *ns*). γ02 showed a negative non-zero relationship (-1.647, $p<.01$). This demonstrates that after controlling for sudden gains and co-morbidity, initial BDI-II scores are negatively related to attributional style at intake (i.e., if BDI-II scores are higher at intake attributional

style will be more depressogenic). γ03 represents the estimated differential in the estimated intake BDI-II between those clients who have a co-morbid presentation at intake and those who do not. This value was indistinguishable from zero (-.771, *ns*).

γ10 represents the estimated rate of change in BDI-II for clients who did not experience a sudden gain, those who scored '0' on the ASQ construct at intake and those who presented with a non co-morbid presentation at intake. This value was negative and non-zero (-.754; $p<.001$), indicating a negative relationship with BDI-II scores. γ11 represented the differential in the rate of change of depression severity between clients who did not experience sudden gains and those who did whilst controlling for the effects of attributional style and co-morbidity. γ11 increased from -.514 in Model D to -.524 in Model E ($p<.01$). γ12 indicates that after controlling for sudden gains, and co-morbidity, attributional style is positively associated with the rate of change in depression severity (.103, $p<.001$). Therefore people with higher ASQ scores at intake are likely to experience a faster rate of change. γ13 was indistinguishable from non-zero and non-significant (.228, *ns*).

Variance components

At *Level 2* the inclusion of co-morbidity to the model led to a small yet significant decrease in the between-person variance for initial status (91.84; $p<.01$). Therefore co-morbidity at intake explains 25.1% of the variance in BDI-II scores at intake as measured by the $R^2 0$ value. Additionally, at *Level 2* the inclusion of co-morbidity led to a small yet significant decrease in the between-person variance in the rate of change of the BDI-II across therapy (.100; $p<.01$). Therefore, Model E accounted for 65.3% of the variance in change trajectories between-persons as measured by the $R^2 1$ value.

Goodness of fit statistics

Overall the deviance statistic reduced from Model D to Model E. However, the AIC and BIC statistics increased slightly indicating that Model E is not a superior fit to Model D.

Overall, Model E contributed little to the overall model. It had a non-significant controlled impact on the fixed effects in terms of initial intake in BDI-II and rate of

change in BDI-II and sudden gains. Therefore, this indicates that co-morbidity status at intake does not moderate the relationship between sudden-gains and recovery in depression.

Multilevel Model Two: sudden gains and within therapy factors

Model D

Table 9.5 represents the results from fitting a multi-level model to explore the within-therapy factors that are associated with sudden-gains and recovery from depression. Model D represents the controlled effects of a client's ASQ score (attributional style) across the course of therapy. Model D evaluated the effects of whether a client experienced a sudden gain or not on BDI-II initial status and rate of change, whilst controlling for the effects of attributional style as a *Level 1* (within-person) predictor.

Table 9.5
Results of fitting a multi-level model to BDI-II data that accounts for sudden gains, attributional style, 'homework beliefs' and 'homework progress': 'Multilevel Model Two: *sudden gains and within therapy factors'*

Parameter			Model A	Model B	Model C	Model D	Model E	Model F
				Fixed Effects				
Initial status, π_{0i}	Intercept	γ_{00}	18.94***	26.98***	26.57***	26.64***	25.52***	25.89***
			(1.71)	(2.13)	(2.82)	(2.37)	(2.69)	(2.67)
	Sudden gain	γ_{01}	-	-	.852	.953	-.529	-.469
			-	-	(4.31)	(3.62)	(4.02)	(3.92)
	Attributional Style	γ_{02}	-	-	-	-1.60***	-1.29***	-1.28***
			-	-	-	(.262)	(.340)	(.339)
	HWF3	γ_{03}	-	-	-	-	.000	.001
			-	-	-	-	(.002)	(.002)
	HWF2	γ_{04}	-	-	-	-	-	-.108
			-	-	-	-	-	(.067)
Rate of Change, π_{1i}	Intercept	γ_{10}	-	-.935***	-.666***	-.659***	-.461**	-.475***
			-	(.111)	(1.23)	(.128)	(.134)	(.135)
	Sudden Gain	γ_{11}	-	-	-.593**	-.649**	-.468*	-.468*
			-	-	(.189)	(.191)	(.180)	(.182)
	Attributional style	γ_{12}	-	-	-	.096***	.074**	.083**
			-	-	-	(.023)	(.027)	(.027)
	HWF3	γ_{13}	-	-	-	-	-.0002	-.0001
			-	-	-	-	(.00017)	(.0001)
	HWF2	γ_{14}	-	-	-	-	-	-.0002
			-	-	-	-	-	(.005)
				Variance Components				
Level 1	Within person	$\sigma^2\varepsilon$	59.73***	23.03***	23.07***	22.20***	21.52***	20.65***
			(1.71)	(1.51)	(1.51)	(1.55)	(1.60)	(1.54)
Level 2	Initial status	σ^20	78.78***	122.58***	123.01***	85.07***	103.418**	101.09**
			(21.87)	(33.90)	(34.02)	(24.21)	(31.08)	(30.40)
	Rate of change	σ^21	-	.288**	.196**	.163*	.123**	.126*
			-	(.094)	(.065)	(.072)	(.056)	(.058)
	Covariance	$\sigma01$	-	-2.76*	-2.70*	-1.16	-1.90~	-1.81~
			-	(1.34)	(1.17)	(.888)	(1.09)	(1.07)
				Pseudo R^2 Statistics and Goodness-of-fit				
	$R^2\varepsilon$		-	0.614	0.614	0.628	.640	.654
	R^20		-	-	-.000	.306	.156	.175
	R^21		-	-	.319	.434	.573	.562
	Deviance		3734.2	3321.8	3311.1	2971.9	2588.9	2562.2
	AIC		3740.2	3333.8	3327.1	2991.9	2612.9	2590.2
	BIC		3753.0	3359.4	3361.2	3033.5	2611.3	2646.6

~ $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$ significance level (2 tailed Pearson correlation). Significant correlations are in bold; α represents 'initial status', β represents 'rate of change'.

Fixed effects

Essentially Model D provides 'controlled' answers to the research question regarding sudden gains and depression. The results from Model D are shown in Table 9.5 above. The fixed effects are described as follows. γ00 indicates the average BDI-II score at intake when clients score '0' in terms of sudden gains (do not experience sudden gains) and '0' on the ASQ. This score was non-zero and significant (26.64, $p<.001$). As in Model C γ01 the estimated differential in initial BDI-II between those clients who experienced sudden gains and those who did not remained insignificant and indistinguishable from zero (.953, *ns*). The significant and non-zero γ02 value (-1.60, $p<.001$) shows that after controlling for sudden gains, initial BDI-II scores are negatively related to attributional style. Therefore, indicating that the less depressogenic a client's attributional style is, the lower their depression severity score will be at intake.

The fixed effect parameter γ10 shows the average rate of change of BDI-II scores of clients who do not experience sudden gains and those clients who score '0' on the ASQ. This value is negative and significant (-.659, $p<.001$), indicating a negative relationship with BDI-II scores. γ11 represents the population average differential between clients who experience sudden gains and those who do not whilst controlling for attributional style change across therapy. As in Model C this value remained negative and statistically significant (-.649, $p<.01$). γ12 indicates that after controlling for sudden gains, attributional style is associated with the rate of change in depression. Therefore, people with a less depressogenic attributional style are likely to experience a faster rate of change.

Variance Components

Model D shows a slight decrease in *Level 1* within person variance from Model C, with 62.8% of the variance explained versus 61.4% of within person variance in Model C. Taken together at *Level 2* sudden gains and attributional style explain 30.6% of the variation in BDI-II initial status and 43.4% of the variation in the rate of change of BDI-II (see $R^2 1$ and $R^2 1$ values). The positive and significant values $\sigma^2 0$ and $\sigma^2 1$ values suggest that there is further unpredicted variation in both initial status and rate of change, which warrants adding further predictors to the multi-level model.

Goodness of fit statistics

The deviance statistics in Model C and D allow us to compare models of 'nested' data. From Model C to Model D this value reduced by 339.2. Furthermore, the AIC value decreased by 335.2 and the BIC value decreased by 327.7. These values provided further evidence that Model D is a superior fit to Model C.

Model E

Model E extended the analysis by incorporating *homework beliefs* as another *Level 1* time-variant predictor. Model E evaluated the effects sudden-gains on BDI-II initial status and rate of change, whilst controlling for the effects of attributional style and *homework beliefs*. Results from Model E are presented in Table 9.5 and are explained below.

Fixed effects

The estimated fixed effects of Model E are presented in Table 9.5 and can be interpreted as follows: γ00 indicates that the average BDI-II score at intake when clients are not experiencing sudden gains, score '0' on the ASQ, and '0' on the homework beliefs construct is 25.52 (p<.001). γ01 represents the estimated differential in the estimated intake BDI-II score between those clients who experienced a sudden gain and those clients who did not. This value again remains indistinguishable from zero (-.529, *ns*). γ02 showed a negative non-zero relationship (-1.29, p<.001) that shows that after controlling for sudden gains and 'homework beliefs', initial BDI-II scores are negatively related to attributional style at intake.

γ10 represents the estimated rate of change in BDI-II for clients who did not experience a sudden gain, those who scored '0' on the ASQ construct at intake and those who scored '0' on the 'homework beliefs' construct at intake. This value was negative and non-zero (-.461, p<.01). γ11 represents the differential in the rate of change of depression severity between clients who did not experience a sudden gain and those who did, whilst controlling for the effects of attributional style and 'homework beliefs'. The absolute value of the fixed effect γ11 decreased from -.649 in Model D to -.468 (p<.05) in Model E and indicates that at least some of the differential between those clients who experience sudden-gains and those who do not may be attributable to their trajectories of 'homework beliefs' over therapy (as

this is controlled for in Model E). Therefore, Model E provides evidence that suggests that 'homework beliefs' moderate the relationship between rate of change in depression, sudden gains and attributional style across therapy.

Variance components

Model E shows a slight decrease in *Level 1* within person variance from Model D. 64.0% of the variance was explained versus 62.8% of within person variance in Model D. Taken together at *Level 2,* sudden gains, attributional style and 'homework beliefs' explain 15.6% of the variation in BDI-II initial status and 57.3% of the variation in the rate of change of BDI-II (see $R^2 1$ and $R^2 1$ values). The positive and significant values $\sigma^2 0$ and $\sigma^2 1$ values suggest that there is further unpredicted variation in both initial status and rate of change, which warrants adding further predictors to the multi-level model.

Goodness of fit statistics

The superior fit of Model E over Model D was confirmed by the associated Deviance statistics. Specifically, there was a 383 point reduction in the Deviance statistic. Furthermore, both the AIC and the BIC values reduced by 379 and 422.2 respectively. This indicated that Model E is an overall superior fit to Model D.

Model F

Model F extended the analysis by incorporating *homework progress* as another *Level 1* time-variant predictor. Model F evaluated the effects sudden-gains on BDI-II initial status and rate of change, whilst controlling for the effects of attributional style, homework beliefs and *homework progress*. Results from Model F are presented in Table 9.5 and are explained below.

Fixed effects

The estimated fixed effects of Model F are presented in Table 9.5 and can be interpreted as follows: $\gamma 00$ indicates that the average BDI-II score at intake when clients are not experiencing sudden gains, score '0' on the ASQ, and '0' on both homework factors is 25.89 ($p<.001$). $\gamma 01$ represents the estimated differential in the estimated intake BDI-II score between those clients who experienced a sudden gain and those client who did not. This value again remains indistinguishable from zero (-.469, *ns*). $\gamma 02$ showed a negative non-zero relationship (-1.28, $p<.001$) which shows

that after controlling for sudden gains and both homework factors, initial BDI-II scores remained negatively related to attributional style at intake.

γ10 represents the estimated rate of change in BDI-II scores for clients who did not experience a sudden gain, those who scored '0' on the ASQ construct at intake and those who scored '0' on both homework constructs at intake. This value was negative and non-zero (-.475, $p<.01$). γ11 represents the differential in the rate of change of depression severity between clients who did not experience a sudden gain and those who did, whilst controlling for the effects of attributional style and both homework factors. The absolute value of the fixed effect γ11 remained identical at -.468 ($p<.05$). Therefore, Model F provides evidence that suggests that *homework progress* does not further moderate the relationship between rate of change in depression, sudden gains, attributional style and homework beliefs across therapy.

Variance components

Model F shows a slight increase in *Level 1* within person variance from Model E. Taken together at *Level 2,* sudden gains, attributional style and both homework factors explain 17.5% of the variation in BDI-II initial status and 56.2% of the variation in the rate of change of BDI-II (see $R^2 1$ and $R^2 1$ values). Therefore, Model F did not explain more variance in the BDI-II rate of change across therapy and indicates that Model E alone is a better fit.

Goodness of fit statistics

Overall the deviance and AIC statistics reduced from Model E to Model F. However, BIC statistic increased slightly indicating that Model F is not necessarily a superior fit to Model E.

Overall, Model F contributed little to the overall model. It had a non-significant controlled impact on the variance components in terms of initial intake in BDI-II and rate of change in BDI-II.

Summary of significant findings

Chapter Nine has summarised the results from the preliminary analyses and the overall multilevel models. There were several significant findings that came out of these analyses which are outlined below:

- For the average client as they progressed through therapy their BDI-II score (i.e., depression severity) reduced.
- Whether or not a client experienced a sudden gain did not moderate the relationship between BDI-II scores at intake and symptom improvement over therapy. Therefore, those clients who experienced sudden gains and those who did not did not differ significantly on their initial depression severity score at the beginning of therapy.
- Clients that did experience a sudden gain within therapy were more likely to have a faster rate of change in their BDI-II scores across therapy.
- A client's attributional style score at intake moderated the relationship between sudden gains and rates of change in BDI-II across therapy.
- Whether a client had a co-morbid diagnosis at intake did not moderate the relationship between sudden gains and rates of change in BDI-II across therapy.
- Attributional style as a *Level 1* (time variant) predictor moderated the relationship between sudden gains and the rate of change in BDI-II across therapy.
- 'Homework beliefs' as a *Level 1* (time variant) predictor moderated the relationship between sudden gains and the rate of change in BDI-II across therapy.

These findings are outlined and discussed in Chapter Ten which follows.

CHAPTER TEN

DISCUSSION

Introduction

This chapter presents and discusses the main findings of this study. The following section will discuss results as pertaining to the original hypotheses of the study as outlined in Chapter Six. This section will then outline the contributions that the current study makes to the literature. Potential limitations within this study and recommendations for further research are outlined. Finally, the practical implications the current study's findings have for clinical practice in the area of treatment for depression are demonstrated and discussed.

Summary of hypotheses within current study

1. *As a client progresses through therapy, their overall levels of depression will decrease*

   As predicted, depression severity, as measured by the *Beck Depression Inventory – Second Edition* (BDI-II), reduced over the course of therapy for all clients. Table G1 demonstrated that for 100% of clients, their depression scores decreased from the point of intake to the end of therapy (See Table G1 in Appendix G). Furthermore, collection of BDI-II scores for those clients who attended 2-month and 6-month follow up sessions showed that this relative improvement was maintained after therapy had finished. Results from the multilevel analysis presented in Chapter Nine also confirmed that significant improvements were made at a session-by-session level. By fitting the unconditional growth model to the BDI-II data in the current sample showed that depression severity scores reduced over time. These findings taken in combination lead to rejection of the null hypothesis. These results support existing empirical research which endorses the efficacy of CBT for the treatment of depression.

2. *Those clients who experience an early sudden gain are more likely to have better outcomes at the end of therapy than those clients who do not experience an early sudden gain*

   Based on previous research it was predicted that those clients who experienced early sudden gains would have better outcomes at the end of therapy than those clients who did not. Clients did experience early sudden gains within the current sample; 12

out of 28 clients (42.9%) experienced a sudden gain in the current study. This percentage was within the typical range found across research in this area (Tang & DeRubeis, 1999; Tang et al., 2005). The multilevel analysis presented in Chapter Nine demonstrated that there was a difference in the rate of change in depression severity across treatment between those clients who experienced a sudden gain and those who did not. Overall findings suggested that those clients who experienced a sudden gain early within therapy had a significantly greater rate of improvement in depression severity than those who did not. Interestingly, there was no significant between group differences in initial depressive symptom severity between clients who experienced a sudden gain and those who did not. This finding suggests that symptom severity at intake does not help to predict whether a client will experience a sudden gain in therapy. Overall these findings supplement the body of sudden gains research that confirms that sudden gains do occur within CBT for depression, they are significant, and are associated with better outcomes within therapy.

3. *Those clients who experience an early sudden gain are more likely to have a less depressogenic attributional style at the start of therapy than those clients who do not experience an early sudden gain.*

   Findings from this study suggested that attributional style at intake has a moderating effect on the relationship between the experience or non-experience of early sudden gains and improvement in therapy. The relationship between attributional style at intake and depression was confirmed, where ASQ scores at intake had a significant relationship with BDI-II scores over time. The multilevel analysis in MLM1 (see Table 9.4 in Chapter Nine, p. 104) demonstrated a negative relationship between clients' initial scores on the BDI-II and their initial scores on the ASQ, suggesting that those with a more severe rating of depression at intake were likely to have a more depressogenic attributional style at intake. Furthermore, multilevel analysis demonstrated a significant positive relationship between attributional style at intake and rate of change in BDI-II across therapy whilst controlling for the presence of early sudden gains. This finding suggests that those clients with a less depressogenic attributional style at intake were more likely to experience a faster rate of change in depression. Therefore, these findings suggest that a client's attributional style before they begin therapy will have an impact on how they improve in therapy. These findings also suggest that clients with less depressogenic attributional styles at intake

are more likely to experience early sudden gains and therefore have better outcomes within therapy.

4. *Those clients who experience an early sudden gain are less likely to have co-morbid diagnoses at the beginning of therapy than those who do not experience an early sudden gain*

   It was initially predicted that the more complex a client's presenting difficulties were in terms of co-morbid diagnoses would affect the presence of early sudden gains. The multilevel analysis carried out in this study found that co-morbid status at intake did not significantly moderate the relationship between early sudden gains and improvement in depression across therapy. The occurrences of co-morbid diagnoses along with major depressive episode were determined using the *Composite International Diagnostic Interview* (CIDI; Kessler & Ustun, 2004). Although the CIDI is a standardised measure, there were several limitations to the methods employed in this study to fully capturing the relationship between diagnostic complexity and the presence of early sudden gains. First of all there were many exclusion criteria within the sample (e.g., presence of borderline personality disorder, current use of antidepressant medication, exclusion of clients with co-morbid axis one alcohol and substance use disorders). Therefore this may have limited the heterogeneity of the sample and the sample may have been less likely to represent a 'typical' clinical population of depressed clients with complex clinical presentations. It could be hypothesised that if the exclusion criteria were less stringent that the presence of co-morbid clinical psychiatric difficulties may have had a moderating effect on the relationship between early sudden gains and depression. Furthermore, the status of co-morbid psychiatric disorders within this study was determined by the clients' answers on the CIDI rather than based on a clinical interview. Therefore, it could be hypothesised that if additional co-morbid diagnoses were identified by a clinician they may have had a moderating effect on the relationship between early sudden gains and depression.

5. *Attributional style change throughout therapy will moderate the relationship between sudden gains and outcome in therapy*

   In the current study it was hypothesised that attributional style change throughout therapy would have a moderating effect on the relationship between the presence of early sudden gains and change in depression. This hypothesis aimed to explore the

overall research question about *within therapy* factors that are associated with early sudden gains in CBT for depression. Results from Model D in MLM2 (see Table 9.5 Chapter Nine) supported this hypothesis. Multilevel modelling analysis allows one to test the relative involvement of different variables whilst concurrently controlling for other variables or parameters. Results from Model D in MLM2 showed that attributional style change across therapy appeared to moderate the relationship between early sudden gains and improvement in depression. Another significant finding of this study is that ASQ scores or attributional style changed across therapy, therefore along with change in depression severity across treatment it showed that cognitive change (in the form of attributional style) was simultaneously changing. These findings support research (see Chapter Four) that postulates that cognitive change plays a moderating role in improvement in CBT for depression and suggests that attributional style change in therapy moderates the relationship between early sudden gains and improvement in depression.

6. *Client beliefs about the homework process throughout therapy will moderate the relationship between sudden gains and outcome in therapy*

It was hypothesised that clients' beliefs about homework would moderate the relationship between sudden gains and improvement in depression throughout therapy. This hypothesis was supported from the results from the multilevel model analyses in Chapter Nine. Results from Model E in MLM2 (see Table 9.5 in Chapter Nine) showed that the fixed effect estimate γ11 decreased significantly from Model D to Model E. This indicated that at least some of the significant differential between those clients who experienced early sudden gains and those who did not was likely to be attributable to change in homework beliefs over the course of therapy. Therefore, Model E in MLM2 provided evidence that indicated that 'homework beliefs' moderate the relationship between rate of change in depression, sudden gains and attributional style in therapy. This finding is significant in that it suggests that how the client thinks about the process of homework throughout therapy plays a moderating role in the relationship between sudden gains and improvement in therapy. It also provides further evidence of the importance of cognitions in the form of beliefs as a moderating factor between sudden gains and improvement in therapy.

7. *Clients' beliefs about their progress in homework throughout therapy will moderate the relationship between sudden gains and outcome in therapy*

It was predicted that clients' beliefs about their progress in homework (i.e., how well they think they completed their homework in terms of their sense of progress, mastery and how much pleasure they believed they gained from the task) would moderate the relationship between sudden gains and improvement in depression whilst controlling for attributional style change throughout therapy and clients' 'homework beliefs'. However, counter to expectations 'homework progress' as a Level 1 *time variant* predictor did not add to the multilevel model (MLM2) as a moderating factor. As presented in Chapter Nine, Model F was not able to explain additional variance in the BDI-II rate of change across therapy and therefore indicated that Model E alone in MLM2 was a better fit. This finding indicated that it may be less important how well the client believes they are doing with the homework task, but rather, whether they believe that the homework task fits within her/his goals of therapy, the client understands why they are doing the homework task and the purpose of it, and the homework task is assigned in collaboration between the therapist and the client. The majority of research into the role of homework in CBT for depression and other psychiatric difficulties has primarily focussed on the amount or the quality of homework (i.e., homework compliance; see Kazantzis et al., 2000 for review) that the client completes. These findings suggest clients' beliefs about the homework process may also be important as a moderating factor that helps to explain improvement in therapy.

Contributions to the literature

Advanced methodology

One of the significant shifts in the literature that the discovery of the phenomenon of sudden gains has emphasised is the importance of moving away from pre-post methodological designs and to rather look at longitudinal analysis when investigating therapeutic processes and outcomes. The discovery of discontinuous change patterns within therapy has highlighted the importance of individual differences in change throughout the course of therapy, rather than just looking at group means. It has been argued that discontinuities in symptom rating across therapy are significant as they can mark points of transition and draw attention to segments of therapy in

which important change processes may be taking place (Hayes, et al., 2007b). One of the main disadvantages with traditional methods of assessing change in therapy (i.e., ANOVA based approaches) is that they do not take into account the fact that individuals in the treatment and control groups are not exactly the same, they are not treated exactly the same, and ultimately individuals do not respond to treatment or control conditions in the same way. In traditional approaches, such as ANOVA, individual differences are attributed to sampling or measurement error (Laurenceau et al., 2007). These approaches are unable to take account of meaningful individual variability in change patterns. Therefore, it was important to utilise longitudinal analysis techniques in this research design that accounted for individual variability in change patterns over the course of therapy. A multilevel model design for analysis of the data was able to answer questions not only about differences between individuals, but it was also able to take into account intra-individual variability. Therefore, this approach recognises that individuals do not respond to treatment in identical ways.

As outlined in Chapter Eight the multilevel model design employed in the current study has many advantages. First of all, the use of session-by-session longitudinal analysis enabled a rich data set of up to 22 time points for each individual client. This approach took full advantage of the sample size within the study. The multilevel design was able to capture the importance of individual change trajectories whilst simultaneously investigating parameters that provided answers about within-person and between-person variance. Multilevel design is an analytic approach that helps provide answers that meet the complexity that contemporary clinical psychology research needs when answering questions about the mechanisms and processes behind change in treatment.

One advantage of the current study is that it is the first to be carried out with a New Zealand population that specifically investigates the occurrence of early sudden gains within cognitive behaviour therapy for depression. This study supports general trends (e.g., that sudden gains are experienced by some clients, that sudden gains are related to outcome in therapy) in the international research on the occurrence of sudden gains within the treatment of depression.

Theoretical contribution

This study has focussed on the *moderators of change* in CBT for depression that are related to the phenomenon of early sudden gains in treatment. *Moderators* are variables that help to clarify under what conditions and for whom an intervention works (Baron & Kenny, 1986). An increased understanding of moderating variables in process research studies is valuable for a number of reasons. First of all, moderators of change can help to match patients' characteristics with treatments that have demonstrated efficacy (Laurenceau et al., 2007). Moderators also help to identify subgroups that respond positively to a mechanism of change and those subgroups whereby the same mechanism may not relate to outcome. Furthermore, by identifying moderators of change it can help to uncover potential *mediators* of change, which represent the potential mechanisms of change through which a treatment modality has its effects (Baron & Kenny, 1986; Laurenceau et al., 2007). Cognitive behavioural therapy has been demonstrated to be efficacious in the treatment of depression (Butler et al., 2006). What is less clear in the literature is the evidence concerning whether cognitive mediation is behind the efficacy within the treatment of depression, as cognitive mediation is the conceptual core of cognitive therapy (Garratt et al., 2007). Garrett et al., (2007) carried out a comprehensive review within the literature pertaining to the question of whether changes in cognition in cognitive therapy predict changes in depression. Upon review it was concluded that the results were largely affirmative with this question in mind (see DeRubeis et al., 1990; Kuyken, 2004; Kwon & Oei, 2003; Rush, Kovacs, Beck, Weissenburger & Hollon, 1981; Tang & DeRubeis, 1999; Teasdale & Fennell, 1982). The general findings of this study contribute towards the affirmative view in support of the cognitive mediation hypothesis. The relationships between the moderating effects of attributional style and homework beliefs on the relationship between early sudden gains and depression are discussed in the section below.

Attributional style and sudden gains

Identifying the possible mechanisms to explain the phenomenon of early sudden gains has been an important endeavour for the reason that research has demonstrated that those clients who experience sudden gains have better outcomes in therapy and on average a lower incidence of relapse post therapy (Busch et al., 2006; Davies, et al., 2006; Greenfield, et al., 2011; Hardy, et al., 2005; Kelly et al.,

2004; Kelly, Cyranowski & Frank, 2007; Stiles et al., 2003; Tang & DeRubeis, 1999; Tang et al., 2002; Tang, et al., 2005). Therefore, understanding the mechanisms behind this discontinuous change pattern may help to provide answers about which clients may respond best to treatment and the ingredients within treatment that may need to be focussed on and developed. The findings within the current study that attributional style moderates the relationship between early sudden gains and rate of response to depression support the view that cognitions and cognitive change play an important role within the mechanisms behind sudden gains.

The present study confirmed that clients with more depressogenic attributional styles at intake experienced a less rapid rate of improvement in therapy. Theoretically this finding makes intuitive sense as it is hypothesised that following from the hopelessness theory of depression (Abramson et al., 1989), that those with a more depressogenic attributional style are more likely to have negative expectations about the occurrence of highly valued outcomes and believe they are helpless to change these negative outcomes. This 'style' of thinking is likely to be a hindrance to improvement within depression. There has been much focus in the literature on the role explanatory style has on predicting the occurrence and severity of depression (Robins & Hayes, 1995). Furthermore, there has been a significant body of research that supports the view that attributional style is correlated with change in depressive symptoms (e.g., Barber & DeRubeis, 2001; Barber et al., 2005; DeRubeis et al., 1990; Selgiman et al., 1988). Therefore, the findings of the current study's multilevel analysis were not unexpected.

Findings from the present study also demonstrated the moderating effects of attributional style *change* throughout therapy on the relationship between sudden gains and rate of change in depressive symptoms. These results highlighted the fact that attributional style changes over time and throughout the course of therapy and that it is not a stable trait (Gotlib, et al., 1993). Previous studies (e.g., DeRubeis et al., 1990; DeRubeis & Hollon, 1995; Jarrett et al., 2007) have found that attributional style as measured by the ASQ pre-therapy and during therapy predicted subsequent depression change within therapy. The current study is unique in the fact that it uses a multilevel model approach to investigate specifically the moderating relationship of attributional style on early sudden gains and outcome in therapy. Unlike other research the analytic approach used in the current study goes beyond just looking at

pre-, mid- and post-measurements of depression severity and ASQ over the course of treatment.

One limitation of the analytic approach employed is that it cannot provide answers about a potential *meditational* relationship between cognitive constructs (e.g., attributional style) and sudden gains. It is unclear whether change in attributional style precedes and causes change in symptoms (therefore leading to sudden gains) or whether the reduction of symptoms in sudden gains leads to change in attributional style. Tang and DeRubeis (1999) found a high degree of cognitive activity in the session preceding the sudden gain compared to a control session. Other researchers have suggested a more 'incremental' pattern of cognitive change and insight preceding the sudden gain (e.g., Goodridge & Hardy, 2009). Strunk and colleagues (2010) highlighted that the occurrence of sudden gains predicted improvement in the therapeutic alliance (i.e., the therapeutic relationship), rather than the other way around. Studies like these highlight the complexity of the relationship between cognitive change and sudden symptom improvement. DeRubeis and Hollon (1995) suggest that the relationship between cognitive change and symptom change is an *integrative* and *reciprocal* one. They explain that early change in explanatory style (i.e., attributional style) may facilitate early progress and problem solving within CBT. As the clients see early improvements, they may become more optimistic about their abilities to help themselves within therapy and more willing to test and challenge unhelpful negative beliefs. It is suggested that in this way clients would be more likely to bring techniques and skills learnt in therapy into everyday life. Therefore, all these factors would contribute to a continuing ameliorative effect on depressive symptoms (DeRubeis et al., 1990). Overall, it is unclear whether cognitive change precedes or follows rapid symptom change in therapy, the findings from this study suggest a reciprocal relationship between early sudden change in therapy and cognitive change.

A possible mechanism behind the reciprocal relationship between sudden gains and change in attributional style is the mobilisation of positive expectations and hope. Hope within therapy has been seen as a key factor in many forms of healing and has had a large impact on psychotherapy thinking and research (Frank, 1968). There has been some relationship found between early rapid response and the client's experience of hope in treatment for anxiety. For example, Hayes et al. (2007a) found

that clients who experienced rapid response to exposure-based cognitive therapy for anxiety were reported as having more hope than clients who were non-rapid responders. Westra and colleagues (2007) also demonstrated a relationship between positive expectancy (i.e., beliefs about outcome) and early change in CBT for anxiety. It could be hypothesised that a similar pattern may be occurring with CBT for depression, where clients' expectancies about therapy and level of hope may increase the incidence of early gains within therapy.

Homework and sudden gains

Results from MLM2 within this study demonstrated that clients' '*beliefs about homework'* over the course of therapy moderates the relationship between early sudden gains and rate of change in depression. This finding is significant as it highlights the role of homework not just as a therapeutic skill, but as a wider part of the therapeutic process. The process of homework is a direct link to the client's belief system (Garland & Scott, 2005; Kazantzis & Daniel, 2009; Kazantzis & L'Abate, 2005). In cognitive therapy the identification and modification of beliefs help both the client and the therapist understand how the client's depression is developed and maintained. Therefore, exploring beliefs through homework is an excellent tool to access a client's beliefs both within and out of therapy (Garland & Scott, 2005). The use of the HRS-II in the current study was advantageous as it enabled a variety of domains associated with homework to be explored. A majority of research studies that investigate the relationship between homework and outcome has focussed on whether the client is compliant with homework or not (Kazantzis et al., 2000). The HRS-II was useful as it allowed dimensions such as *beliefs* to be investigated, which goes beyond whether the client simply completed the homework or to what quality that homework was carried out. The HRS-II was also administered at every session and this allowed for change over time in *homework beliefs* and *homework progress* to be analysed over time, rather than just looking at one static, retrospective and potentially biased measurement.

The positive finding that a client's beliefs about homework throughout the course of therapy moderate the relationship between early sudden gains and rate of change in depression is significant as it provides further evidence that clients' changing beliefs or cognitions over the course of therapy play a moderating role in the relationship between sudden gains and improvement within depression. This provides further

support for the cognitive mediation hypothesis/theory (see Garratt et al., 2007). These results suggests that whether or not clients believe that the homework assigned to them is relevant, useful and fits within their individual therapeutic goals is very important to their recovery from depression. It could be suggested that a change in the focus of outcome studies from simply looking at the quantity and quality of the homework completed. Homework outcome studies may benefit from measuring and investigating the beliefs that clients have around the homework that they are assigned over the course of therapy. Although the findings from the current multilevel study can only imply that homework beliefs play a moderating role in the relationship between sudden gains and outcome (rather than a meditational or causative one), it is assumed that the effect of beliefs in homework throughout therapy are likely to have a *reciprocal* relationship with symptomatic gains throughout treatment. Therefore, it is assumed that early engagement within the therapeutic process and positive beliefs about the homework being assigned is likely to lead to early and noticeable improvements in therapeutic outcome (sudden gains), which in turn is likely to strengthen their belief in the therapeutic process and lead to more engagement within this process. All these factors are likely to interact to create greater symptom improvement throughout therapy.

Limitations of the current study

There were several potential limitations within the current study. In terms of *hypothesis one* it is important to point out that this study did not employ a control group to compare those clients who received CBT and those clients who received no therapy (control condition). Factors outside of therapy could not be ruled out as potentially influencing the positive outcome of BDI-II scores across treatment sessions. One of the main limitations of the current study was that not all measures were available for all time points. Specifically, the ASQ was only administered at intake, session 5, session 8, session 20 and follow up sessions. Therefore it was not measured between session 9 and 19. There was sound rationale for the majority of ASQ measurements to be administered in the first half of therapy, as it is hypothesised that this is where a majority of significant change occurs (Illardi & Craighead, 1994; Tang & DeRubeis, 1999). Future research would benefit from more time point measurements of cognitive structures to further investigate the relationship between sudden gains and cognitive change, as this could more

accurately demonstrate the trajectory of cognitive change across treatment. However, there are practical implications in terms of time and client engagement when several measures have to be administered to the client at every session. Each client was already completing the BDI-II and the HRS-II at every therapy session. It could be argued that additional measures to fill out at every session could be time consuming and discourage clients.

Another limitation of the current study is homogeneity of the sample in terms of demographic characteristics. There were several screening criteria for the current sample in terms of age, number of depressive episodes, co-morbid diagnoses, absence of active medication, and absence of current substance dependence. There are potential limitations in terms of the generalisation of the results to a community sample and the types of clients that clinicians would typically see. However, there was a sound rationale for these screening criteria utilised as the current study wanted to control for potentially confounding variables. It is recommended that replications be carried out within New Zealand and internationally that include community samples or clinical populations.

Another consideration of the current study is that no causal relationships were found (i.e., the results from the current study show moderating relationships between the variables and not mediators of change). Therefore, there are still questions about the direction of the relationships between sudden gains, client variables/predictors and cognitive change. It would be useful for further researchers to extend their research questions to examine potential mediators of change that may explain the phenomena of sudden gains within cognitive behavioural therapy for depression.

Suggestions for further research

The current study examined factors that were quantifiable (i.e., measureable quantitatively) and took advantage of a quantitative methodological approach. The findings from this study have shown that change in attributional style and beliefs about homework across the course of therapy moderate the relationship between early sudden gains and improvement in therapy. Qualitative research may be useful in investigating clients' subjective experiences of sudden gains and to produce a detailed exploration of the content and processes. Goodridge and Hardy (2009) used theoretically driven case analysis based on the *assimilation model* (Stiles, Elliott,

Llewelyn, Firth-Cozens, Margison, Shapiro, & Hardy, 1990) to provide a detailed descriptive account of the large improvement seen in sudden gains in therapy for depression. Their findings suggested that insight was developed gradually over the sudden gain. Qualitative findings such as these are complimentary to the quantitative multi-level approach employed in the current study to further investigate the mechanisms behind early sudden gains in therapy for depression.

It is also suggested that another option for future research is to increase the time points or measurement intervals of the ASQ. Although it would be impractical to administer this lengthy measure to the client at every session, one or two additional measurement points between sessions 9 and 19 may have aided in enhanced interpolation of the data. Additionally, the use of alternative cognitive measures such as the Dysfunctional Attitudes Scale (DAS, Beck, et al., 1991) or the Automatic Thoughts Questionnaire (ATQ, Hollon & Kendall, 1980) and the like would be useful to see if similar relationships are found between cognitive change and early sudden gains in therapy.

Practical implications for clinical practice

The findings from the current study have implications that can be noted and applied to clinical practice of cognitive behavioural therapy with depressed clients.

- *Markers for identifying clients that are likely to experience an early sudden gain*

The first consideration for the clinician is to note that those clients who do not experience an early sudden gain in therapy may require a longer course of therapy than those clients who do experience early sudden gains. Clinicians may wish to identify those clients who are likely to experience these gains in terms of treatment planning. The findings from this study suggest that attributional style at intake moderates the relationship between early sudden gains and improvement in therapy. Those clients who demonstrated a less depressogenic attributional style at intake were more likely to demonstrate a faster rate of change in depressive symptoms on the BDI-II. Therefore, if clinicians notice either that their clients are not making *sudden gains* early in treatment, or display significant depressogenic attributional styles, the early stages of therapy may benefit from focussing on building positive expectancies about therapy. This may occur in the form of motivational

interviewing, building therapeutic alliance or exploring clients' beliefs about the therapeutic process. Assessing attributional style during the clinical assessment interview process may help to determine those clients who will potentially experience a sudden gain and identify those clients who may have a slower rate of improvement in depressive symptoms.

A second point for clinicians to consider is this study's finding that co-morbidity did not significantly moderate the relationship between *sudden gains* and outcome. The complexity of a client's presentation as defined by number of DSM-IV-TR diagnosis criteria met for co-morbid psychiatric diagnoses did not significantly affect rate of change in BDI-II scores or the initial intake BDI-II score. This finding highlights the importance of theory and individualised case conceptualisation over psychiatric diagnoses in making clinical decisions about treatment planning (Emmelkamp et al., 2010).

- *Clients beliefs about homework and implications for therapy*

Findings from the current study suggest that clinicians should monitor and be aware of clients' beliefs about homework as these have been found to moderate the relationship between sudden gains and improvement in therapy. This finding highlights the importance of an open and communicative alliance between the client and the therapist. Best practice guidelines around the use of homework in cognitive behavioural therapy emphasise the importance of clinicians' facilitating a collaborative therapeutic relationship, using the cognitive conceptualisation to guide the use of homework, and making sure that the client understands the rationale of how the assigned homework task aligns with their treatment goals (Kazantzis & Daniel, 2009; Kazantzis, Deane & Ronan, 2005). The use of these best practice guidelines are supported by the current study's findings. It is advised that clinicians communicate with their clients around their beliefs the homework process. The findings suggest that just because a client may not be completing their homework this does not mean that they are ignoring the principles or skills that they had learnt in therapy. Nor does it indicate that their beliefs about the process of homework were necessarily negative. Therefore, homework compliance should not be the only consideration of the practicing clinician. It is encouraged that clinicians make an effort to actively monitor and enquire about clients' beliefs about the homework process throughout therapy.

- *Be aware of session-by-session outcome change throughout therapy*

This study has demonstrated and replicated the finding that early sudden gains in CBT for depression are associated with better outcomes throughout therapy. Therefore, it is recommended that clinicians measure and are aware of session-by-session change within therapy. Administration of self-report measures such as the BDI-II at the beginning of every therapy session would be useful to identify clients' patterns of improvement throughout therapy. The clinician could potentially share this pattern of outcome change over the course of therapy with their clients. If the client sees that they are making gains s/he may be more likely to buy into and engage in the therapeutic process and increase his/her level of hope.

There are additional benefits for clinicians analysing individual data session by session. Howard, Krause and Lutz (1998) emphasised a return to the functional analysis of individual data: "in order to maximise the relevance for clinical practice, the results of treatment research should always be reported at this most disaggregated or individual change level" (p. 838). It is argued that the focus on individual time course data is important to contribute towards an understanding of how change occurs (Barkham, et al., 1993). Therefore, single subject design on part of the clinician is advocated to help answer these questions. Furthermore, tracking changes in outcome on a session-by-session basis allows for the identification of discontinuities (e.g., early sudden gains) in individual client's symptom trajectories. This can guide both researchers and clinicians to the segments of therapy that are most likely to reveal factors that mobilise and inhibit change (Hayes, et al., 2007a).

The findings from the current study suggest that critical change occurs in early sessions of therapy. Furthermore, those who experience early sudden gains have better outcomes throughout therapy. To help identify the mechanisms behind these changes, it is recommended that as scientist-practitioners, psychologists should focus on session-by-session measurement and monitoring in early sessions of therapy as findings suggest that critical changes occur within these early sessions of therapy. Additionally, the reality of clinicians offering fewer treatment sessions due to shortage of clinical resources and financial limitations highlights the value in understanding what happens in the early sessions of therapy of critical importance. Current research into early sudden gains would suggest that these early changes identified within the current study are not unique to CBT for depression, but also

occur with other psychiatric difficulties and treatment modalities (see Chapter Three, p.25, for summary). This type of assessment/measurement approach in clinical practice/or research would be justified in other treatment modalities and psychiatric diagnoses where it is desirable to identify possible moderators, mediators and mechanisms of change.

Final conclusions

The primary aims of this study were to investigate the client factors that may predict the occurrence of early sudden gains within CBT for depression and to additionally investigate the within therapy factors that are associated with early sudden gains. *Sudden gains* are defined as a discontinuous change pattern whereby a client shows a significant improvement from session-to-session. Early sudden gains within CBT for depression was the main focus of the current study for several reasons: it has been found that sudden gains are associated with better outcomes within therapy and post-therapy (Hardy et al., 2005; Kelly et al., 2004; Tang & DeRubeis, 1999; Tang et al., 2005; Tang et al., 2007); they may help to identify those clients who will respond favourably to therapy; and they may provide further clarification around change mechanisms and processes within therapy (Hayes et al., 2007a; Laurenceau et al., 2007). Research within the area of sudden gains has clearly demonstrated that sudden gains within CBT for depression are associated with positive treatment outcome. However, there remains uncertainty around the mechanisms and processes which can help to explain why these sudden and rapid improvements occur within therapy. Therefore, the focus on the mechanisms and predictors of this discontinuous change pattern was justified.

Twenty-eight adult participants were recruited to engage in up to 20 sessions of CBT for depression at Massey University, Albany, New Zealand. Two follow-up sessions at two and six month intervals were also offered to the participants. Clients were screened to ensure that they met DSM-IV-TR (APA, 2000) criteria for major depressive episode. Clients were excluded if they had experienced recurrent MDD, were taking psychoactive medication or substances, were concurrently engaging in counselling/psychotherapy, met DSM-IV-TR diagnostic criteria for psychosis of borderline personality disorder or had any imminent risk of self harm. Assessment information about depressive severity was gathered at every session. Additionally, measures of homework and attributional style were also collected throughout

therapy. Clients were seen by doctoral students who were specifically trained in CBT for depression and optimal homework implementation.

A longitudinal multi level analytic design was utilised within the current study to enable the exploration of both between-person and within-person variance over time. This type of analytic design was favourable as it recognised that individuals do not respond to treatment in identical ways and can capture the importance of individual discontinuous change patterns within therapy. Overall, clients' depression improved across the course of therapy and these gains were maintained at two- and six-month follow up sessions. 42% of clients experienced a sudden gain within the current study. Those that experienced a sudden gain within therapy were more likely to experience a faster rate of change in depressive severity than those that did not experience a sudden gain. However, clients' initial scores of depression severity at intake did not significantly moderate the relationship between sudden gains and rates of improvement in depressive severity. Additionally, whether a client was experiencing a co-morbid diagnosis at intake did not moderate the relationship between sudden gains and rate of change in depression severity across treatment. However, attributional style at intake did moderate the relationship sudden gains and rate of change in depression severity across treatment. Indicating that those clients with less depressogenic attributional styles at intake are more likely to experience a sudden gain within therapy and experience a faster rate of change in depressive symptoms. In terms of within therapy variables that are associated with sudden gains and outcome in therapy it was found that both attributional style change throughout therapy and homework beliefs throughout therapy moderated the relationship between sudden gains and rate of change in depression severity across therapy. Overall, these findings support theory that emphasises the role of cognitions and beliefs as an important change mechanism within CBT for depression.

The findings within the current study have highlighted a number of practical implications for clinical practice. First of all, clinicians are encouraged to monitor session-by-session improvement as it helps to identify significant discontinuous change patterns such as sudden gains and the factors that are associated with these change patterns. Secondly, clinicians are encouraged to monitor attributional style at the beginning of therapy. Those clients who experience a more depressogenic

attributional style may benefit from therapeutic techniques such as motivational interviewing that help to build positive expectancies about therapy and their ability to make positive changes. Furthermore, clinicians are encouraged to monitor and be aware of clients' beliefs about homework throughout therapy. The findings from this study suggested that when practitioners monitor homework with their clients they should not only focus on compliance, but monitor and explore clients' beliefs about the homework process.

Overall, this research has confirmed the significance of sudden gains within cognitive behavioural therapy for depression. It has highlighted the importance of session-by-session measurement of change (rather than simple pre- /post-measurement of change) to help answer questions about *how* and *why* change occurs in psychotherapy. The findings from this study have emphasised that discontinuous change patterns such as sudden gains are associated with better outcomes over all in therapy and that cognitive factors such as attributional style and beliefs about homework play a moderating role in the relationship between sudden gains and improvement in symptom severity over the course of CBT for depression.

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APPENDIX A

Examples of advertisements

Study to find out how to beat the blues

A team of specially trained Massey psychologists is offering free therapy to first-time depression sufferers in Auckland as part of a collaborative international study involving Harvard University and the London Institute of Psychiatry.

The University's Centre for Psychology will provide data from therapy sessions with volunteers, so that the team of international researchers can better understand the dynamics of Cognitive Behavioural Therapy (CBT), how it alleviates depression symptoms and how it equips sufferers to avoid repeat bouts of depression.

Findings from the study could offer hope for depression sufferers, many of whom do not have access to affordable, effective treatment, says Dr Nik Kazantzis, senior lecturer and practitioner who heads the team.

Depression sufferers typically experience low mood, poor appetite, lack of energy, disturbed sleep, feelings of helplessness and guilt. They may find decision-making difficult, feel miserable when they make even the smallest mistake and generally feel life has become overwhelming.

CBT teaches people how to become their own therapists by teaching them skills so they can deal better with difficult situations and the painful emotions they trigger, says Dr Kazantzis. Volunteers are being offered 20 hour-long individual sessions to learn strategies for changing problem thoughts and behaviours.

He says CBT is a widely used, mainstream therapy developed by American-born psychiatrist Dr Aaron Beck in the 1960s. Although it has been endorsed by more than 400 studies internationally as an effective, low-cost treatment for a range of disorders, including depression, little is known about why it works.

Dr Kazantzis, who trained under Dr Beck two years ago, believes the therapy is particularly suited to New Zealanders as it offers immediate, practical help in coping with the present and does not necessarily require clients to embark on in-depth analysis of their pasts to be effective.

People can volunteer for the therapy if they have not been previously diagnosed with depression and are not taking medication that affects the brain.

About 121 million people world wide suffer from depression but fewer than 25 per cent have access to effective treatment, according to the World Health Organisation. Depression is the fourth-highest contributor to the global burden of disease, and is expected to become the second highest by 2020.

For more info about participating in the study call Nicole

Source: http://www.massey.ac.nz/massey/about-us/news/article.cfm?mnarticle=study-to-find-out-how-to-beat-the-blues-21-05-2008

Request for Information

If the criteria appear to fit your circumstance, and you are interested in participating in this study please fill out the form below. Once complete fold this entire 3-sided pamphlet and return to the address provided.

Alternatively contact the researchers directly to learn more about participating in this study. Please phone Nicole at 09-4140800 extn. 41252.

First name: _____

Surname: _____

Phone number:
(____)_____

Best time to contact (between 9pm and 4pm):

Morning: _____am AND

Afternoon: _____pm

Days of the week:
_____

Send completed forms to:
Depression Study
Centre for Psychology
School of Psychology, Massey University
Level 3, North Shore Library Building
Albany, Auckland

Attach Stamp Here

Depression Study
Centre for Psychology
School of Psychology, Massey University
Level 3, North Shore Library Building
Albany, Auckland

Massey University
AUCKLAND · PALMERSTON NORTH · WELLINGTON · EXTRAMURAL

Are You Suffering from Depression?

Volunteers Needed for Depression Study

What is Depression?

Almost everyone feels sad or "depressed" at times. **Clinical Depression** (also called Major Depressive Disorder) may include symptoms such as:

- Loss of interest in usual activities
- Changes in appetite
- Changes in sleep
- Changes in sexual desire
- Difficulties in concentration
- A decrease in activities and social withdrawal
- Increased self-criticism or reproach
- Thoughts of or actual plans related to suicide

Clinical depression is distinguished from **manic-depression** or **Bipolar Disorder** in that the individual only experiences periods of depression, potentially returning to normal functioning in between times. In Bipolar Disorder, however, the individual will cycle between depression and periods of full manic problems (euphoria, high energy, lots of activity).

Quick Facts:

- Depression is widespread debilitating and costly
- 1 in 5 people experience clinical depression at some point in their life
- Women are at twice the risk of men
- People who experience depression are at high risk of repeated experiences with relapse as high as 60% 1 year after recovery

Can Psychological Treatments Help with Depression?

Cognitive Behavioural Therapy (CBT) involves the recognition of negative patterns in depression, and correcting these patterns through various practical skills. CBT also uses behavioural strategies. CBT is shown to be successful in 67% of individuals with clinical depression and can reduce risk of further episodes of depression.

Although complex, a variety of factors increase the risk of clinical depression. These include having a parent who has been clinically depressed, physical illness, the death or separation of parents, negative life events, pervasive negative thinking, physical or emotional deprivation, seasonal onset, childbirth, or having previously experienced depression.

Benefits of CBT:

- Safe alternative to drug therapy for depression
- Roughly as successful as medication
- Lower drop out rates (10% versus 25-30% on medication)
- Increased likelihood of longer term results

The Depression Study

Researchers at the Centre for Psychology, at Massey University, Albany are investigating a theory as to how we can help individuals maximize their gains in psychological therapy and reduce the risk for the future. Dr. Nikolaos Kazantzis, a Senior Lecturer in Psychology, has conducted CBT research for over a decade. Key co-investigators at Massey University are Associate Professor Paul Merrick and Professor Janet Leathem, who are both senior researchers and experienced practitioners. This team is currently recruiting volunteers to participate in a research project on depression.

You might be eligible to receive free assessment and treatment as part of volunteering for this research study. The researchers are looking for men and women between the ages of 18 and 65 who are currently experiencing a major depressive episode for the first time.

For inclusion in the study you must also be:

(a) Able to read, write, and speak in English
(b) NOT taking medications that Affect the brain (an occasional sleeping tablet and/or the oral contraceptive is ok)
(c) NOT involved in concurrent supportive counselling or psychotherapeutic treatment
(d) NOT meet diagnostic criteria for substance abuse, psychosis, borderline personality disorder
(e) NOT currently at risk to yourself or someone else

The time commitment is about two and half hours for an initial comprehensive assessment. The first half of the assessment includes completing questionnaires and the second half involves a diagnostic interview. Participants will receive feedback regarding the assessment and will then receive 20 sessions of cognitive behaviour therapy for depression (free of charge) over an 18 week period.

To learn more about participating in this study, please phone Nicole at 09-4140800 extn. 41252 or follow the instructions and post the 'Request for Information' form on the back of this pamphlet.

APPENDIX B

COGNITIVE BEHAVIOR THERAPY HOMEWORK PROJECT

PHONE SCREENING INTERVIEW PROTOCOL

Introduction

When person answers say something like:

Hello, may I speak to Jane Smith (who presumably has left a message somewhere saying she is interested in taking part).

If yes, then say, *Jane* or *Ms Smith* (depending on age) *this is Mary Brown, I'm a researcher from Massey University. You left a message on* (say when or can't remember say recently) *about possibly taking part in our research study. Is this a convenient time to talk about it?*

If yes then listen to what they spontaneously say and if they don't spontaneously talk say *the easiest way to start is by just outlining again the what the study is about and if you're still ok with it, I'll ask you some questions to help work out whether you fit within the group of people we are looking for this time.*

If no, then arrange a more convenient time to call back if they are still interested

So, basically what we are wanting to do is work out how treatment for depression, in particular Cognitive Behavioural Therapy can be improved to help people with depression. It is already a proven treatment, but we think there are ways that it could be improved. We are looking for 70 people between the age of 18-65 years from the greater Auckland area that are experiencing an episode of major depression for the first time in their lives. People can only be included in this study if they meet a certain set of criteria, therefore I will have to ask you several questions, this may take 20-30 minutes of your time.

But before going any further, you should know that every you say will be confidential. But there are two exceptions to this – and that is if I think there is any chance that you may cause harm to yourself. In that case I have break confidentiality for the sake of yours and others safety.

OK?

If yes, then proceed with, *right, then let's start with the questions*

If not ok, then listen to reservations, reassure and proceed or otherwise terminate.

Initial Questions

a) *What is your DOB? How old are you?* – needs to be between ages of 18 and 65 but since age already discussed at outset, won't be too many problems with this.

b) *Can you read, English OK? Write? Hold a conversation?*

c) *Are you currently taking any medication prescribed by your doctor?* If yes, *What is it*? This could include the contraceptive pill or sleeping medication. Google. Exclude occasional hypnotic and oral contraception.

d) *What help are you getting if any for your depression*?

e) *Is this the first time you have felt like this?*

I now have to ask you some questions about the way you have been feeling lately, the reason I am asking these questions is to assess whether you would benefit from participating in the study. We ask these questions so we are sure that the people participating in this study are likely to benefit from the treatment we provide.

DSM-IV-TR MAJOR DEPRESSION

At least 5 of the following symptoms have been present during the same two week period and represent a change from previous functioning; at least one of the symptoms is depressed mood or loss of interest.

1. Do you have a depressed mood (feel "sad", "down", "angry" or "empty") most of the day, every day. How is your mood?
   - How long have you been feeling…..
   - Do you feel that way nearly every day?
   - How much of the day does it last?
   - How bad is the feeling?
   - SUD – how would you rate this feeling on a scale of 0-100 (0 being not at all sad and 100 being the most sad you have ever been)?

   Criteria – depressed mood most of the day, nearly every day, as indicated by subjective report or observation by others

2. Have you lost interest in or do you get less pleasure from the things that you used to enjoy?
   a. What do you normally enjoy doing? (TV, reading, sports, shopping, socialising, eating, hobbies?)
   b. What do you still enjoy?
   c. What have you lost interest in?
   d. For how long have you not enjoyed these things like you used to?
   e. Is it like that nearly every day?

   Criteria – Markedly diminished interest or pleasure in all or almost all activities most of the day nearly everyday

3. Have there been any changes in your appetite for food?
   a. Increased? Decreased?
   b. How much more/less have you been eating?
   c. Is it like that nearly every day?
   d. For how long has it been this way?
   e. Have you gained or lost any weight? How much? Since when?

   Criteria - Significant weight loss/gain when not dieting (change of more than 5% in month) or a decrease or increase in appetite nearly every day.

4. How has your sleeping been?
   a. How many hours per night have you been sleeping?
   b. How does this compare to normal?
   c. Increased? Decreased?
      i. Is it a problem nearly every day?
      ii. How long have you had these sleep problems?
      iii. If decreased – do you have any problems falling asleep, staying asleep, or waking up to early in the morning?

   Criteria – insomnia or hypersomnia nearly every day.

5. Listen for slowed speech, long pauses before answering

questions or between words.

a. Agitation: Have you been feeling more fidgety lately? Are you having problems sitting still?
    i. IF YES: Do you pace back and forth?
    ii. Have others noticed your restlessness
b. Retardation: Have you felt slowed down, like you are moving in slow motion
    i. IF YES: have others noticed this?

Criteria – psychomotor agitation or retardation nearly everyday (has to be observable by others)

6. How has your energy levels been?
    a. Have you been feeling tired or worn out?
    b. IF YES: Duration? (For how long have you…) Persistence? (Do you feel like this nearly everyday?)

Criteria – Fatigue or loss of energy nearly everyday?

7. How have you been feeling about yourself?
    a. What has your self-esteem been like?
        i. IF LOW: What types of thoughts do you have about yourself?
        ii. Do you feel like you are worthless or a failure?
            1. If yes: Tell me about it
    b. Have you been blaming yourself for things?
        i. Like what?
    c. Do you feel guilty
        i. IF YES: what about?
        ii. How hard is it to get your mind off this?
        iii. Do you think about things from the past and feel guilty about them?
        iv. IF YES: Like what?
        v. Is the patient's guilt or worthlessness on the patients mind everyday?
        vi. What has your **self esteem** been like?
            1. Has there been any change?

Criteria – feelings of worthlessness or excessive or inappropriate guilt nearly everyday (not merely self-reproach or guilt about feeling sick)

8. Have you been having problems thinking or concentrating?
    a. IF YES: what does this interfere with?
    b. Are you able to watch TV? Read? Follow a conversation?
    c. How long have you noticed this happening?
    d. Does it happen nearly everyday?

Is it harder to make decisions than before?

IF YES: What kind of decisions is harder to make?

a. What about every day decisions?
b. How long have you had this problem?
c. Does it happen nearly everyday?

Criteria – Diminished ability to think or concentrate, or indecisiveness, nearly everyday.

9. **SUICIDE SCREEN** - Sometimes when a person feels down or depressed they may think about dying, this is

quite common. It is very typical and common for people to have thoughts about harming or killing themselves. Have you been having any thoughts like that?

a. IF YES: Tell me about it. Have you thought about taking your life?
   i. IF YES: Did you think of ways to do it?
   ii. Do you currently have a plan?
   iii. Do you have the means to carry out this plan?
   iv. How close have you come to doing it?
   v. IF you have not attempted, why not? What stops you from doing it? What are the protective factors?
   vi. IF NO: Do you wish you were dead? Do you have thoughts of death or dying?
   vii. When you go to sleep do you often wish that you would not wake up?

Criteria – Recurrent thoughts of death, not just of dying, recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide.

10. What difficulties in your life has the depression caused? OR: How have these difficulties affected your life?
    a. Does it bother you a lot that you feel this way?
    b. Has it caused problem in your job? Study? Relationships? Friends? Family? Social life? Doing household chores?

Criteria – The symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

11. Do you have any medical conditions?
    a. YES:
       i. When did the conditions start?
       ii. Has there been any change to this condition? Lately?
12. Have you lost a loved one in the last two months? Exclude: unless – if associated with marked functional impairment, morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms, or psychomotor retardation.
13. During this time have you been experiencing delusions or hallucinations? At the same time as depressive symptoms.
14. IF PSYCHOTIC
    a. Was there a time when you had the (hallucinations/delusions) but did not feel sad or depressed?
    b. IF YES: How long did you have [psychotic symptoms] only?
    c. When did the depression begin in relation to this?
15. Has there been a major event in your life lately that may have came before you felt like this? E.g. a death of a close relative or friend (bereavement)?

16. When you started experiencing these symptoms have you ever felt this way?
    a. Have there been times lasting at least a few days

when you felt the opposite of depressed, that is when you were very cheerful or high and this felt different from your normal self?

IF YES or UNCLEAR:

1. Did you feel hyper, or like you were high on drugs, even though you had not taken anything?
2. Did anything cause your good mood?
3. How long did it last?
4. So, was this more than just feeling good?
5. When did this occur?
6. How many periods like this have you had?

IF NO:

7. What about a period lasting at least a few days when you were unusually irritable, and quick to argue or fight?

IF YES: Describe what that was like.

i. Were you using drugs or alcohol?
ii. Did you get into many arguments or fights?
iii. How long did this period last?
iv. Was there a reason you felt this way?
v. When did it occur?
vi. How many times have you felt this way?

If the clients report manic symptoms ask these questions:

- Did you find during this period you needed less sleep than usual?
- Did you notice you had racing thoughts and ideas?
- Were you easily distracted?
- Did you find you were overly occupied with pleasurable activities (or goal oriented)?
- Did you find during this period you were involved in a lot of risky and potentially self-damaging behaviour e.g. gambling, drinking, stealing, speeding?
- During this time did you feel more irritated than normal?
- During this time did you find it hard to relax?
- Did you find you felt like this constantly over a period of a week? (apply to all questions).

DRUG ABUSE/DEPENDENCE

Alcohol:

Pre-screen:

Frequency: How often do you have a drink containing alcohol?
Daily? Weekly?
How many drinks containing alcohol do you have on a typical day when drinking?
How often do you have six or more drinks in one occasion?

Now I am going to ask you some questions about your use of alcohol
What are your drinking habits like? – for example how

much alcohol do you consume a night? How much would you consume in a week?
Was there ever a time in your life when you drank too much?
IF YES: How old were you?
Has anyone in your family said that you were an excessive drinker?
Have friends, a doctor, or anyone else ever said that you drank too much?
Has alcohol ever caused problems for you?
IF YES: What kind of problems?
How old were you when you had these problems?
If all questions are answered NO unlikely to meet diagnosis of Alcohol abuse

Street drugs:

Have you ever used street/recreational drugs?
*if used less that 10 times go to prescribed medicines
Did you ever think you used drugs too much?
IF YES: how old were you?

Has anyone in your family said that you use drugs too much?
Have friends, a doctor, or anyone else said that you use drugs too much?
Have drugs ever caused a problem for you?
IF YES: What kinds of problems?
How old were you when you had these problems?

Prescribed medication:

Have you ever used sleeping pills, tranquilisers, weight loss medicines or pain killers?
IF YES:
How long did you take the drug?
Did you get hooked or addicted to it?
Did you take much more than was prescribed?
IF all the above are answered NO, diagnosis of drug dependence unlikely

A1:

Because of drinking or taking drugs how often have you
- Missed work or school?
- Have trouble at work or school?
- Got fired etc?
- Not taken care of children?
- No cook, clean house, go grocery shopping?

A2:

Do you drive while intoxicated?
How often?
Did you ever drink (take drugs) and them do something that was potentially dangerous (e.g. operating machinery)

A3:

Were you ever arrested for driving under the influence, or disorderly conduct?
Were you ever busted for selling/buying drugs?

IF YES: How many times

Recurrent drug related legal problems

A4:

Because of your drug taking/drinking did you….

- frequently have problems or arguments with friends or family?
- Spend less time with family or friends?
- Get separated or divorced?
- Get into physical fights?
- Get violent?
- IF YES TO ANY: do you still drink/take drugs despite these problems?

**Have you taken any drugs/consumed any alcohol today? Do you plan to drive anywhere? (e.g. do your children need to be picked up etc – follow up.)

Drug Abuse = A maladaptive pattern of drug use leading to clinically significant impairment or distress, as manifested by one or more of the following occurring within a 12 month period.

Screen for psychosis: *Now I will ask you some questions about how you perceive or view the world around you.*

DELUSIONS AND HALLUCINATIONS

DELUSION OF REFERENCE

When watching TV, listening to the radio, or reading the paper do you notice that they are referring to you, or that there are special message intended for you?

- What have you noticed?

Does it seem like **strangers on the street are taking special notice** of you or talking to you? Is it a feeling you have, or **are you pretty sure** that they are talking about / referring to you?

How do you know?

Does it seem like things are especially arranged for you?

In what way?

DELUSIONS OF PERSECUTION

Is anybody against you, following you, giving you a hard time, or trying to hurt you?

Do you feel like there's a plot to hurt you?

Who's involved?
Why would they want to hurt you?

THOUGHT BROADCASTING

Do you ever think of something so strongly that people could hear your thoughts?

So, people can hear what you are thinking even when you are not talking?

How do you know?

DELUSIONS OF MIND READING

Are people able to read your mind and know what you're thinking?

How can they do this?

Do they literally read your thoughts, or do they read your facial expression to know what you're thinking?

THOUGHT WITHDRAWAL

Are your thoughts ever taken out of your head?

Does someone or some force reach into your head an steal or remove your thoughts?

THOUGHT INSERTION

Are there ever thoughts in your head that have been put there from the outside?

DELUSION OF GUILT

Do you think you've done something so terrible and deserve to be punished?

I know it will be hard to talk about, but what do you feel so guilty about?

Do you blame yourself for bad things going on in the world, like wars, crime, and starvation?

DELUSION OF GRANDIOSITY

What is your self-esteem like?

Do you feel more self-confident than usual?

Do you think you have special talents, abilities, or powers?

When some people feel (HIGH, EUPHORIC, etc) they may think they're going to become famous or do great things. Did you have any thoughts like that?

DELUSION OF CONTROL

Do you ever get the feeling that you're being controlled by some force or power from the outside?

At times, does it seem like you're not in control of your body, almost like you're a puppet and something from the outside pulls the strings?

So, at times your body does certain things without your willing it?

SOMATIC DELUSION

Are you concerned that you have a serious physical illness that a doctor hasn't found, or that something is wrong with your body?

HALLUCINATIONS

VISUAL HALLUCINATIONS

Have you seen visions or other things that other people didn't see?

What did you see?
What time of the day did this occur?

How long ago did it start?
Do you see it everyday?
How often do you see it?

AUDITORY HALLUCINATIONS

Have you heard noises, or sounds, or voices that other people didn't hear?

What did you hear?

Do the voices seem to come from inside or outside your head?

IF INSIDE: But you hear it with your ears?

How many voices do you hear?

Are they male or female?

Do you recognize them?

Do you ever hear two or more voices talking to each other?

Do the voices ever talk about what you're doing or thinking?

IF YES: Do they keep a running commentary on what you are doing or thinking just like a sports commentator?

How long ago did the voices start?

Do you hear them every day?

How often during the day do you hear them?

Do they influence your behaviour?

Do they tell you to do things?

TACTILE HALLUCINATIONS

Do you ever notice strange sensations in your body or on your skin? Do you ever feel something creeping or crawling on your body, or something push or punch you but no one is there?

IF YES: Like what?

When did it happen for the first time?
How often has it happened?

OLFACTORY AND GUSTATORY HALLUCINATIONS

What about smells that other people don't notice, or strange tastes in your mouth?

IF YES: Like what?

When did it happen for the first time?

How often has it happened?

Are they associated with any other physical symptoms like an upset stomach, numbness, tingling, or brief memory loss?

Tell me about that?

Screen for Borderline PD

I am now going to ask you some questions about your relationships with other people. I will specifically ask you questions about your feelings in your relationships

- **Inclusion = at least 5 criteria**

- **Pervasive pattern of instability of interpersonal relationships, self-image, and marked impulsivity beginning at early adulthood and present in a variety of contexts:**

1) Have there been times when you've been very upset, almost to the point of being distraught, because you thought someone you loved or needed, might leave you?

a. How often has this happened?

b. What did you do to stop them from leaving?

Do you spend a lot of time thinking of ways to keep people from leaving you?

Frantic efforts to avoid real or imagined abandonment (do not include self mutilating behaviour.

2) Do your relationships with friends and lovers tend to be intense and stormy with lots of ups and downs?

a. IF YES: can you tell me about some of them

With some people do you switch from loving, respecting, and admiring them at one time, to despising them at another time?
If YES: Tell me about that

A pattern of unstable and intense interpersonal relationships characterised by alternating between extremes of idealisation and devaluation.

3) Does the way you think about yourself change so often that you don't know who you are?

a. IF YES: tell me about this
Do you ever feel like you are something else, or that you're evil, or maybe that you don't even exist? Tell me about that.

Identity disturbance: markedly and persistently unstable self-image or sense of self.

4) I am going to read you a list of behaviours that sometimes causes problems for people. How many times in the last 5 years have you:

a. Gambled more money than you could afford to lose
b. Spent money on things you could not afford
c. Been high on drugs
d. One night stands/ sexual affairs
e. Intoxicated by alcohol
f. Charged with reckless, driving, speeding ticket etc
g. Driving while intoxicated or high
h. Gone on eating binges
i. Done anything impulsive where you could have gotten hurt

Impulsivity in at least two areas that are potentially self-damaging (do not include suicidal or self-mutilating behaviour.

5) Have you ever been so upset that you told someone that you wanted to kill or hurt yourself?
a. IF YES: Tell me about it
b. How often have you done this?
Have you ever made a suicide attempt, even one that wasn't very serious?
IF YES: What did you do?
How many attempts have you made?
Have you ever been so upset or tense that you deliberately hurt yourself by cutting your skin, putting your hand through a glass window, burning yourself or anything like this?
IF YES: What have you done?
How often?

Recurrent suicidal behaviour, gestures or threats, or self-mutilating behaviour.

6) Has anyone ever told you that you are irritable *(touchy, short-tempered, cross, ill-tempered)* or that your moods seem to change lots?
a. IF YES: tell me about it
Do you often have days when your mood is constantly changing—days when you shift back and forth from feeling your usual self, to feeling angry or depressed or anxious?
IF present:
Are the mood swings mild or very intense?
How often does this happen in a typical week?
How long do the moods last?
Affective instability due to marked reactivity of mood – lasting from a few hours and only rarely a few days.

7) Do you feel empty most of the time?
a. IF YES: What percent of the time do you feel that way?
Chronic feelings of emptiness

8) How easily do you lose you temper?
How often do you lose your temper?

Do you feel angry much of the time?
What kinds of things get you really angry?
Are you sometimes angry with knowing why you feel that way?

Tell me what you are like when you are angry.
How long do you usually stay angry?

Do you ever throw or break things? Have you ever hit anyone? Do you get into physical fights? IF YES: tell me about it.

When you are angry do you ever give someone the silent treatment?
IF yes: how long can you keep it up?
Is that a common reaction for you?
Inappropriate, intense anger or difficulty controlling anger

9) When some people are under stress, they have experiences that are very hard to explain to other people. Have you ever felt like things around you were somehow strange, or changed in size or shape?

IF YES: describe what that is like.

When you've been under stress, have you ever felt your body of part of it was somehow changed or not real?

Have you ever felt you were watching yourself from outside your body?
IF YES: describe what that was like?
Do you ever have brief blackouts and forget what has happened?

When you are feeling stressed, do you ever get paranoid or suspicious of people you usually trust?
IF NO: what about being afraid that someone is spying on you or trying to hurt you?
IF YES: Does this happen even when you are stressed?

IF YES to any of above
- were you using any drugs or alcohol when these experiences happened?
- IF YES: does this only happen when you are taking drugs and alcohol?
- IF NOT: How long do the experiences last?
- DO they go away when you are not under stress?

Transient, stress related paranoid ideation or severe dissociative symptoms.

Acceptance and Referral

ACCEPTANCE

a) We would like to offer you the opportunity to be involved as a participant in this study.
b) We are optimistic about this therapy, there has been significant scientific support to demonstrate CBT as an effective

and beneficial first line treatment for first episode depression. *c) Due to limited resources in this study it is important for you to keep in mind that this study is based in western/Pakeha culture. We do not offer a culturally specific service. However, if you identify as Maori and you are concerned, we will have a kaumatua available for consultation during the therapy process – he is available to consult us on any cultural issues that may arise during the process.* - Would you prefer a culturally specific service? Yes? Refer to WDHB. Please note that if a participant identifies culturally-related concerns during the course of their participation in assessment or treatment, clinical supervisors should be consulted in the first instance. The School of Psychology kaumatua koro Turoa will also be available for consultation regarding any cultural issues that may arise in the process of this study. Turoa may be contacted in emergencies at 027-2888-135, and less urgent enquiries should be directed through Robyn Knuth, Secretary to the Head of School, Turitea campus. Set up time for interview and meeting with therapist… REFFERAL Make sure safety checks have been carried out. (Sucide screen, present use of alcohol drugs) – contacted acute services if needed. Make a referral to the best alternative mental health service. Provide contact details. Contact referral source and contact study co-ordinator.	

APPENDIX C

Information and consent forms

Massey University
COLLEGE OF HUMANITIES AND SOCIAL SCIENCES
Te Kura Pūkenga Tangata

Participant Information Sheet

Depression Study

You are invited to take part in a research study involving a brief psychological treatment for depression called Cognitive Behaviour Therapy (CBT). The purpose of the study is to examine certain processes of therapy which may increase its positive benefits. The study will involve 70 individuals between the age of 18 and 65 years, recruited within the greater Auckland area. Like yourself, these individuals will currently be experiencing a major depressive episode for the first time. Before you consent to be part of this study, please read the following. Ask as many questions as you need to be sure that you understand what taking part will involve. The decision to take part is entirely your choice.

If you provide written consent to be involved, you will receive a comprehensive psychological assessment, then a 20 session protocol of CBT for depression over a 16 week period. Treatment will be individualised based on your specific needs and goals, and provided by advanced clinical psychology trainees under close supervision. Consistent with prior research on CBT for depression, sessions will be scheduled twice a week for the first 4 weeks and then weekly for the next 12 weeks. Follow-up sessions will occur at 2 months and 6 months after treatment has ended. Participants will be asked to complete some assessment questionnaires to determine treatment gains, and also asked to provide informal feedback on the CBT they received. Your total time commitment (assessment, therapy sessions, questionnaires, and follow-up) is estimated to be about 30 hours, plus travel to and from the Centre for Psychology. Therapy will be provided by clinical psychology doctoral/masters students trained in delivering this protocol.

How will the study benefit you? It is expected that new information, which may benefit you or others, will be obtained by this study. Furthermore, it is very likely that the comprehensive psychological assessment and therapy offered as part of this study will improve your condition, although this cannot be guaranteed. These services will be provided free of charge. Due to funding limitations, you will be responsible for your own travel costs to and from the Centre for Psychology in Albany. Parking will be provided free of charge.

Who is unable to take part? Participants will need to be proficient in reading, writing, and conversing in English. They must be free from taking drugs which act on the central nervous system. They must not meet diagnostic criteria for substance abuse, psychosis, or borderline personality disorder. Lastly, they must be able to be managed safely with outpatient psychotherapy.

If you do agree to take part, you are free to withdraw from the study at any time without having to give a reason. This will in no way affect your continuing health care, as you will be referred to an appropriate provider to further assist your specific needs. Participation in this study will be stopped should any harmful effects appear or if an appropriate medical professional feels it is not in your best interest to continue. You may be taken out of the

study if you need treatment that is not allowed during this study, or if the study is cancelled. You will be asked to check with your study therapist before taking any other treatment; this includes anything from the supermarket, pharmacy or health shop.

Will my information remain confidential? Participating in this study will involve having your therapy sessions videotaped (and transferred to DVD discs) in order for the researchers to monitor the therapy protocol. All information collected about you during the study, including the recorded sessions, will be kept strictly confidential and only accessed by those researchers and clinical supervisors directly involved in the study. The only time in which confidentiality is breached is in the event that you express an intention to harm either yourself or somebody else, in which case a crisis team would become involved. No material which could personally identify you will be used in any reports on this study. All assessment information and clinical notes will be kept in individual files stored in a locked clinical records room, with files coded with anonymous identification numbers. Files will be stored in a separate location from both the identifying information and the DVD archive.

The information collected will be used for the research project and for publication in academic journals. All participants will be offered a summary of the findings at the conclusion of the study. This will include details of any publication arrangements that have been made. Please note that there is likely to be a delay between data collection and publication.

In the unlikely event of a physical injury as a result of your participation in this study, you may be covered by ACC under the Injury Prevention, Rehabilitation and Compensation Act. ACC cover is not automatic and your case will need to be assessed by ACC according to the provisions of the 2002 Injury Prevention Rehabilitation and Compensation Act. If your claim is accepted by ACC, you still might not get any compensation. This depends on a number of factors such as whether you are an earner or non-earner. ACC usually provides only partial reimbursement of costs and expenses and there may be no lump sum compensation payable. There is no cover for mental injury unless it is a result of physical injury. If you have ACC cover, generally this will affect your right to sue the investigators. If you have any questions about ACC, contact your nearest ACC office or the investigator.

If at any time you have questions or concerns about this study, you are welcome to contact: Dr. Nikolaos Kazantzis (who now has an academic office at La Trobe University), phone: Auckland (09) 8898292, or email: N.Kazantzis@latrobe.edu.au

If you have any questions about any issues pertaining to Maori in this study, regardless of your own ethnicity, you are welcome to contact Kaumatua koro Turoa, via the School of Psychology, phone Auckland (09) 414 0800 extension 2040.

If you have any queries or concerns regarding your rights as a participant in this research study, you can contact an independent Health and Disability Advocate. This is a free service provided under the Health & Disability Commissioner Act:

Telephone (NZ wide):	0800 555 050
Free Fax (NZ wide):	0800 2787 7678 (0800 2 SUPPORT)
Email:	advocacy@hdc.org.nz

This study has received ethical approval from the Northern X Regional Ethics Committee.

Massey University
COLLEGE OF HUMANITIES AND SOCIAL SCIENCES
Te Kura Pūkenga Tangata

Consent Form

Depression Study

This consent form will be held for a period of five (5) years

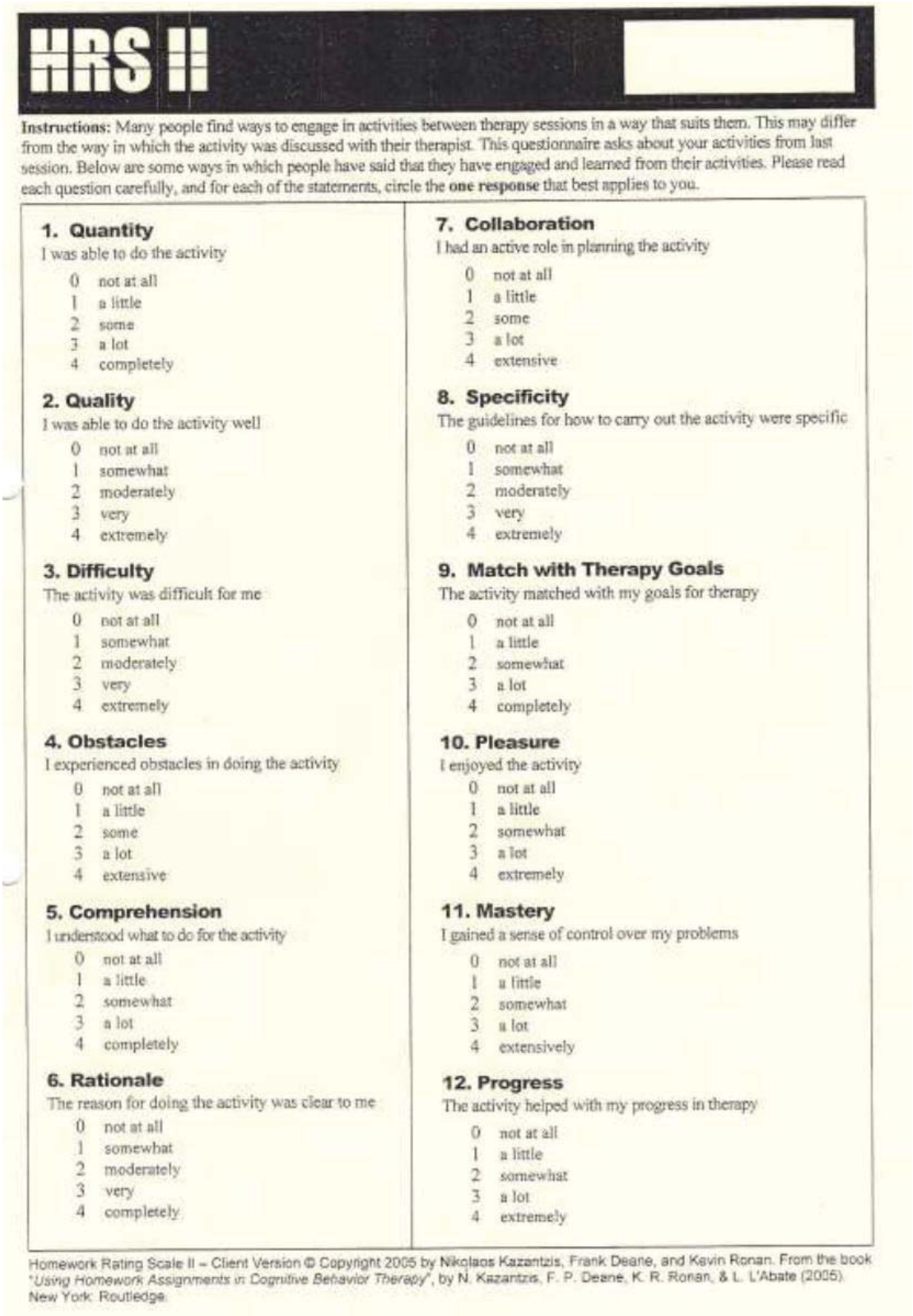
- I have read and I understand the Information Sheet dated 20 February, 2009, for volunteers taking part in the Depression Study
- I have had the details of the study explained to me.
- I have had the opportunity to use whanau support or a friend to help me ask questions and understand the study.
- My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.
- I have been given contact details to use in case I have future questions about the study.
- I understand that taking part in this study is voluntary (my choice) and that I may withdraw from the study at any time.
- I understand that my participation in this study is confidential and that no material that could identify me will be used in any reports on this study.
- I agree to my sessions in this study being videotaped.
- I understand that I will not receive any compensation for travel costs or for the time I spend as a participant in this study.
- I have had adequate time to consider whether or not to take part in this study. I agree to participate in this study under the conditions set out in the Information Sheet.

Signature: _____ **Date:** _____

Full Name - printed _____

APPENDIX D

Homework Rating Scale – II: Client & Therapist Versions



HRS II

Instructions: Many people find ways to engage in activities between therapy sessions in a way that suits them. This may differ from the way in which the activity was discussed with their therapist. This questionnaire asks about your activities from last session. Below are some ways in which people have said that they have engaged and learned from their activities. Please read each question carefully, and for each of the statements, circle the **one response** that best applies to you.

1. Quantity
I was able to do the activity
0 not at all
1 a little
2 some
3 a lot
4 completely

2. Quality
I was able to do the activity well
0 not at all
1 somewhat
2 moderately
3 very
4 extremely

3. Difficulty
The activity was difficult for me
0 not at all
1 somewhat
2 moderately
3 very
4 extremely

4. Obstacles
I experienced obstacles in doing the activity
0 not at all
1 a little
2 some
3 a lot
4 extensive

5. Comprehension
I understood what to do for the activity
0 not at all
1 a little
2 somewhat
3 a lot
4 completely

6. Rationale
The reason for doing the activity was clear to me
0 not at all
1 somewhat
2 moderately
3 very
4 completely

7. Collaboration
I had an active role in planning the activity
0 not at all
1 a little
2 some
3 a lot
4 extensive

8. Specificity
The guidelines for how to carry out the activity were specific
0 not at all
1 somewhat
2 moderately
3 very
4 extremely

9. Match with Therapy Goals
The activity matched with my goals for therapy
0 not at all
1 a little
2 somewhat
3 a lot
4 completely

10. Pleasure
I enjoyed the activity
0 not at all
1 a little
2 somewhat
3 a lot
4 extremely

11. Mastery
I gained a sense of control over my problems
0 not at all
1 a little
2 somewhat
3 a lot
4 extensively

12. Progress
The activity helped with my progress in therapy
0 not at all
1 a little
2 somewhat
3 a lot
4 extremely

HRS II

Instructions: This questionnaire consists of 12 questions regarding your client's homework completion from last session. Please read each question carefully, and circle the number of the **one response** that best describes your impression of the client's experience. If several statements apply equally well, circle the lowest number for that group. Be sure not to choose more than one response for any question.

1. Quantity
The client was able to do the activity

0 not at all
1 a little
2 some
3 a lot
4 completely

2. Quality
The client was able to do the activity well

0 not at all
1 somewhat
2 moderately
3 very
4 extremely

3. Difficulty
The activity was difficult for the client

0 not at all
1 somewhat
2 moderately
3 very
4 extremely

4. Obstacles
The client experienced obstacles in doing the activity

0 not at all
1 a little
2 some
3 a lot
4 extensive

5. Comprehension
The client understood what to do for the activity

0 not at all
1 a little
2 somewhat
3 a lot
4 completely

6. Rationale
The reason for doing the activity was clear to the client

0 not at all
1 somewhat
2 moderately
3 very
4 completely

7. Collaboration
The client had an active role in planning the activity

0 not at all
1 a little
2 some
3 a lot
4 extensive

8. Specificity
The guidelines for how to carry out the activity were specific

0 not at all
1 somewhat
2 moderately
3 very
4 extremely

9. Match with Therapy Goals
The activity matched with the client's goals for therapy

0 not at all
1 a little
2 somewhat
3 a lot
4 completely

10. Pleasure
The client enjoyed the activity

0 not at all
1 a little
2 somewhat
3 a lot
4 extremely

11. Mastery
The client gained a sense of control over their problems

0 not at all
1 a little
2 somewhat
3 a lot
4 extensively

12. Progress
The activity helped with the client's progress in therapy

0 not at all
1 a little
2 somewhat
3 a lot
4 extremely

APPENDIX E

Normality plots and graphs

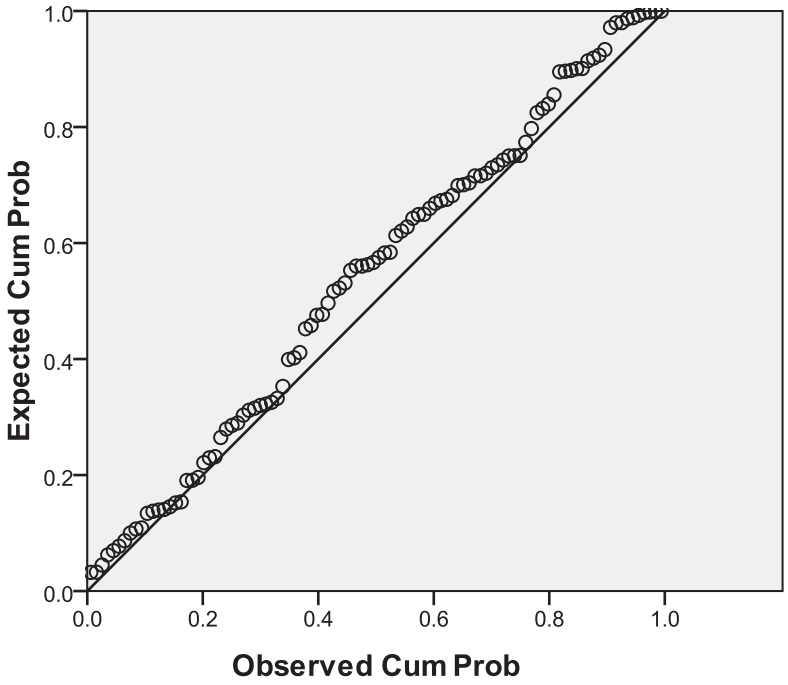


Figure E1: Normal P-P standardised residual plot for attributional style (composite score) across five time points. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

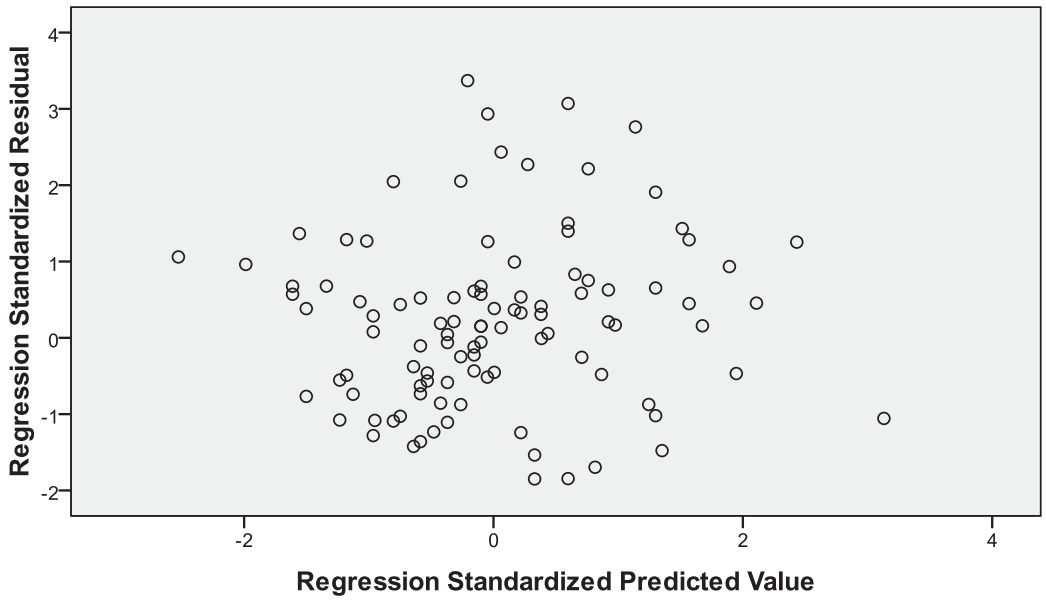


Figure E2: Standardised residual scatter plots for attributional style across five time points (composite score). *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

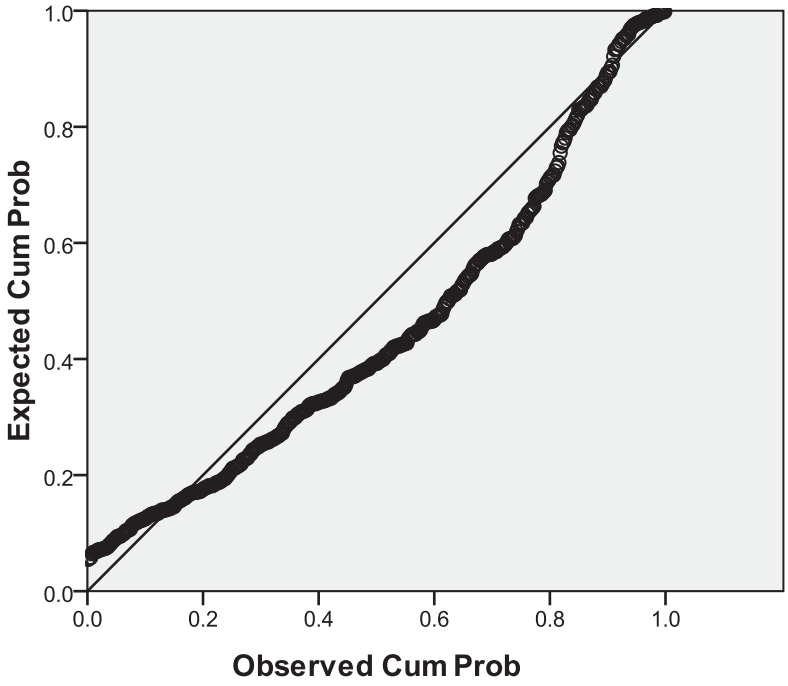


Figure E3: Normal P-P standardised residual plot for HRS-II (total score) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

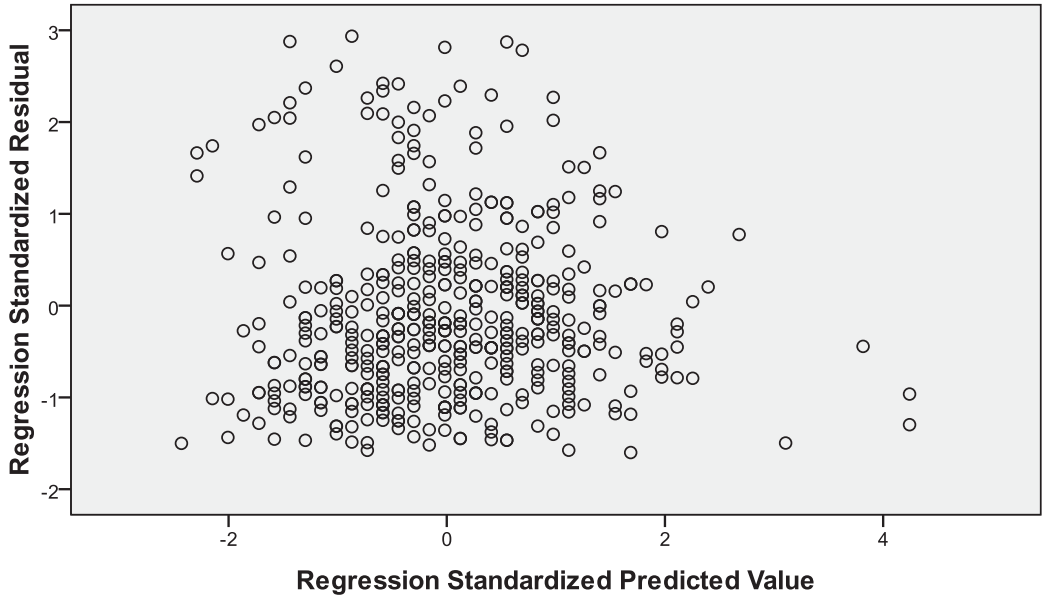


Figure E4: Standardised residual scatter plots for HRS-II client version (total score) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

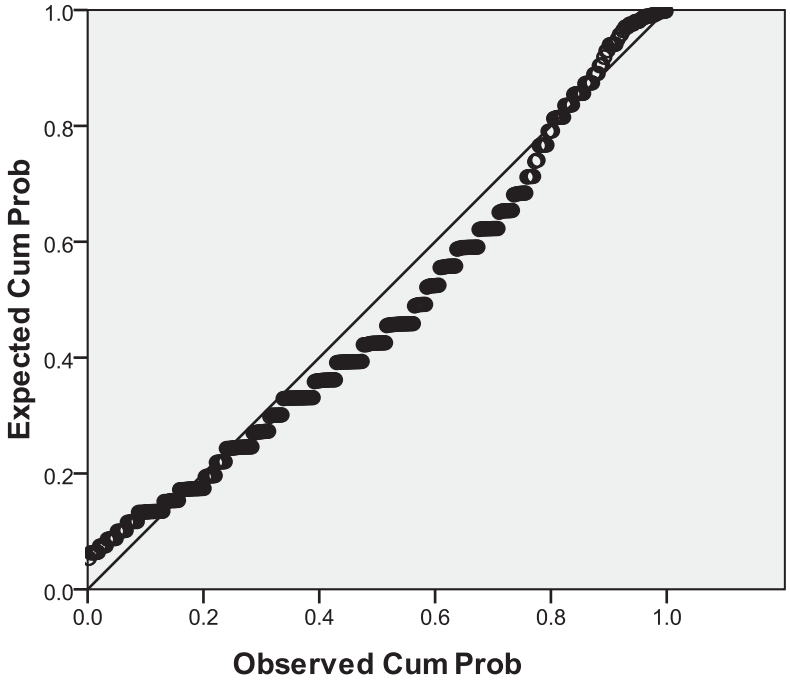


Figure E5: Normal P-P standardised residual plot for Homework Factor Three across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

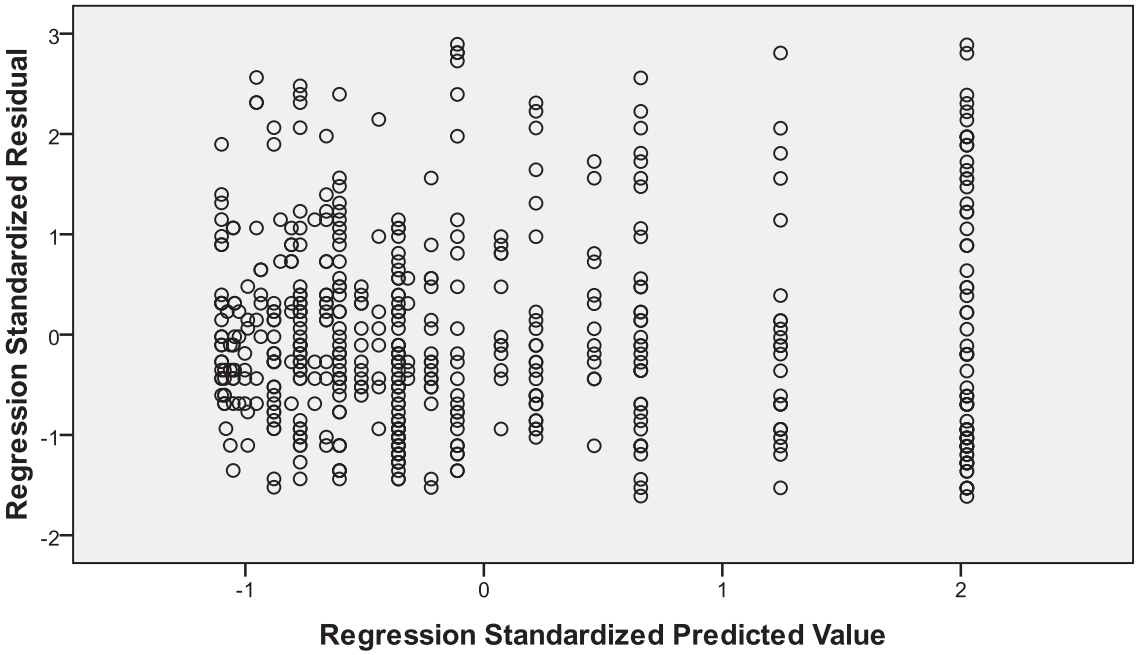


Figure E6: Normal P-P standardised residual scatter plot for Homework Factor Three across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

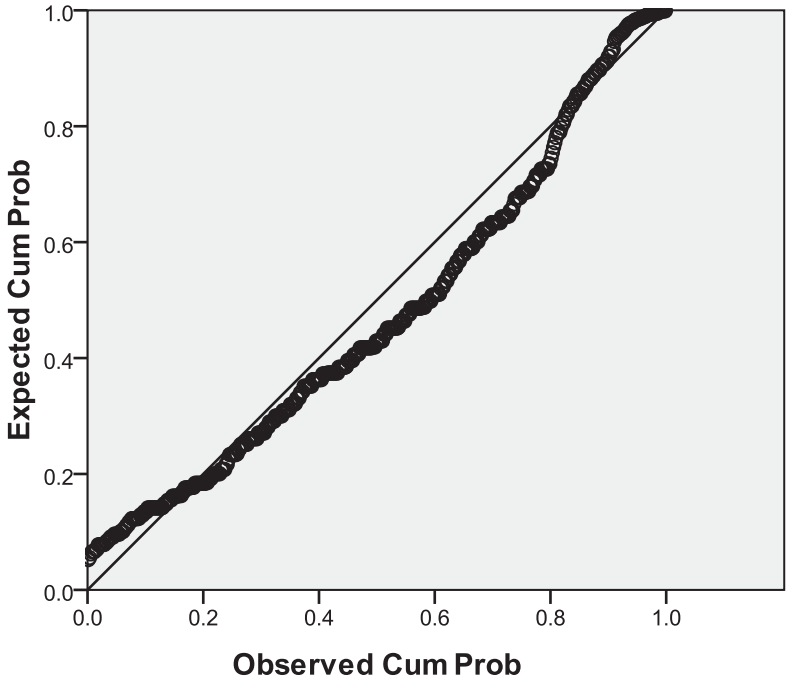


Figure E7: Normal P-P standardised residual plot for HRS-II (Item 1 – Quantity) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

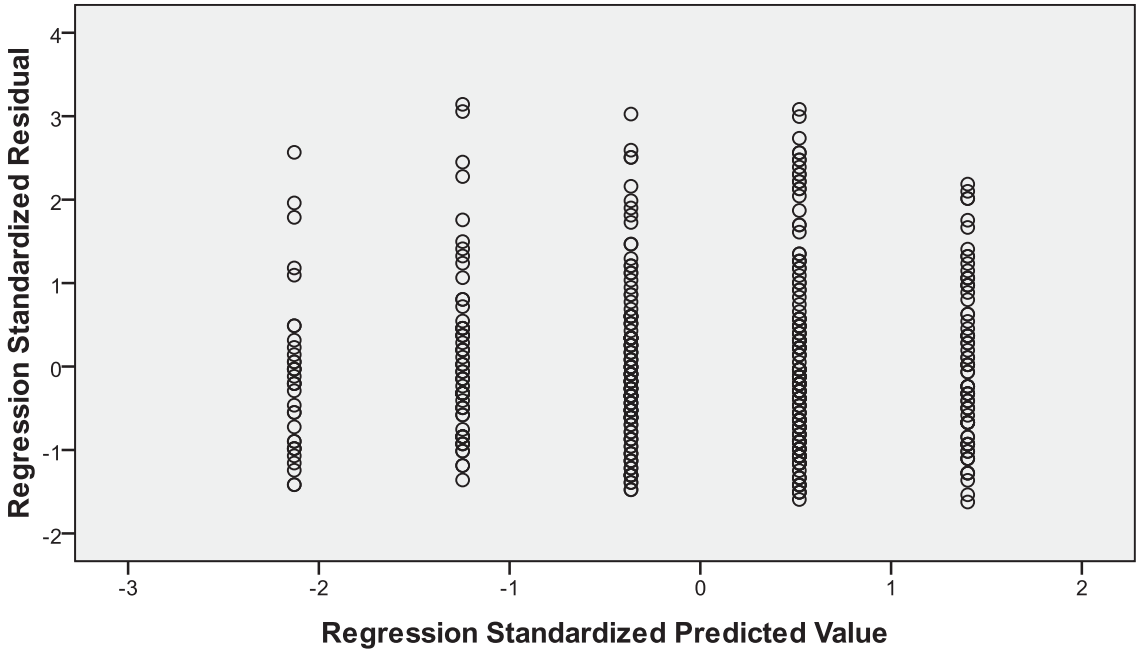


Figure E8: Normal P-P standardised residual scatter plot for HRS-II (Item 1 – Quantity) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

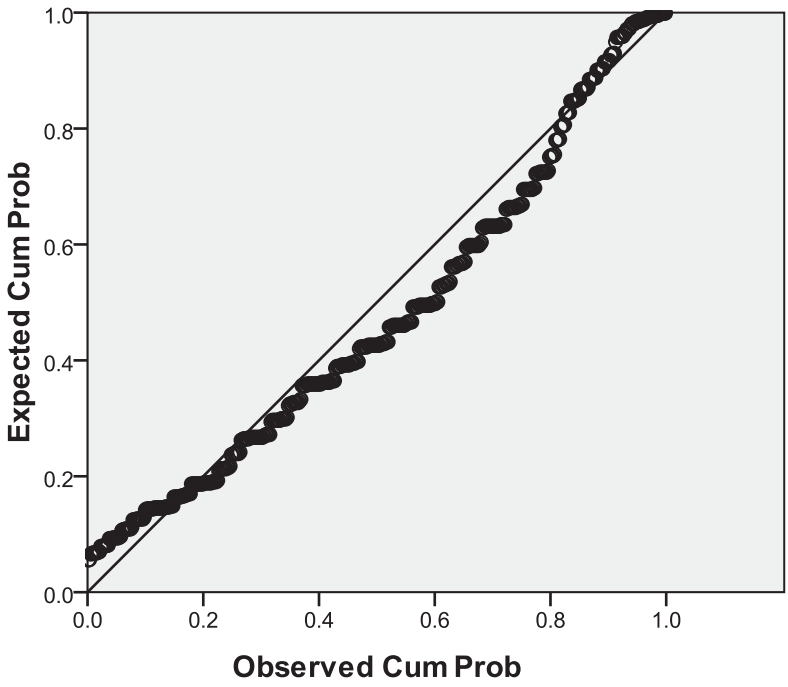


Figure E9: Normal P-P standardised residual plot for HRS-II (Item 2 – Quality) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

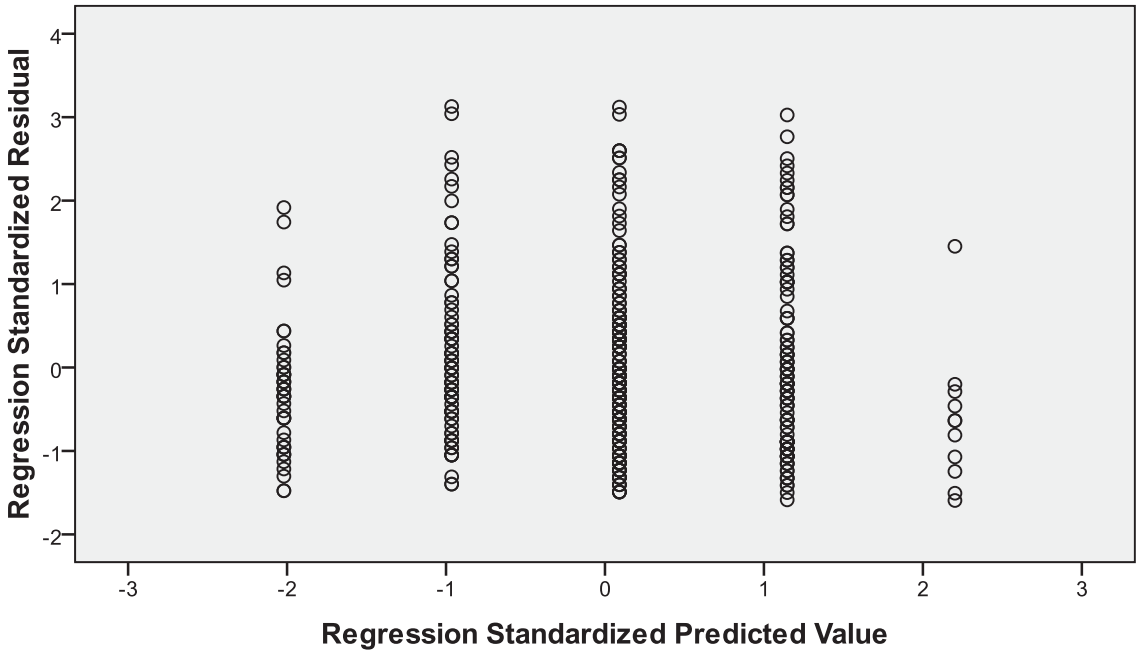


Figure E10: Normal P-P standardised residual scatter plot for HRS-II (Item 2 – Quality) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

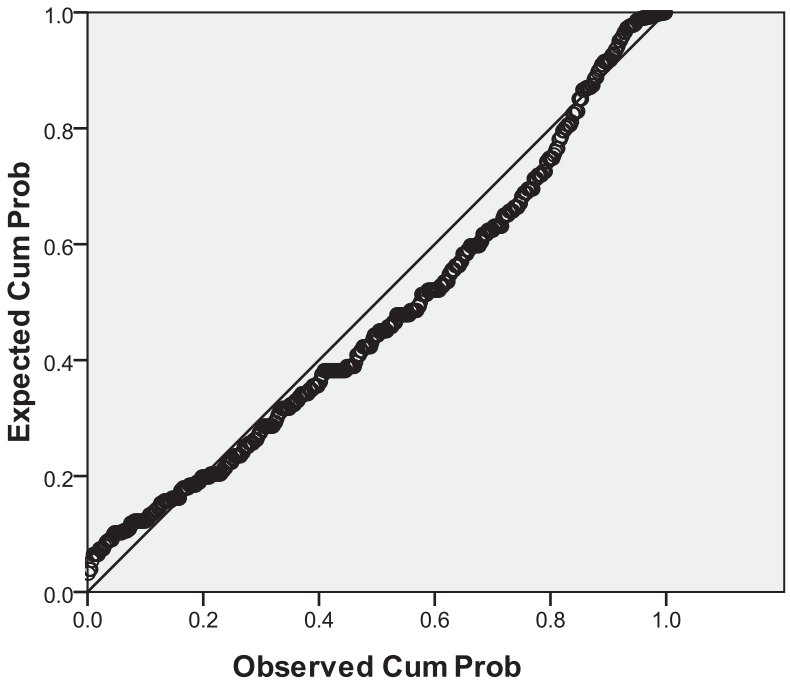


Figure E11: Normal P-P standardised residual plot for HRS-II (Item 3 – Difficulty) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

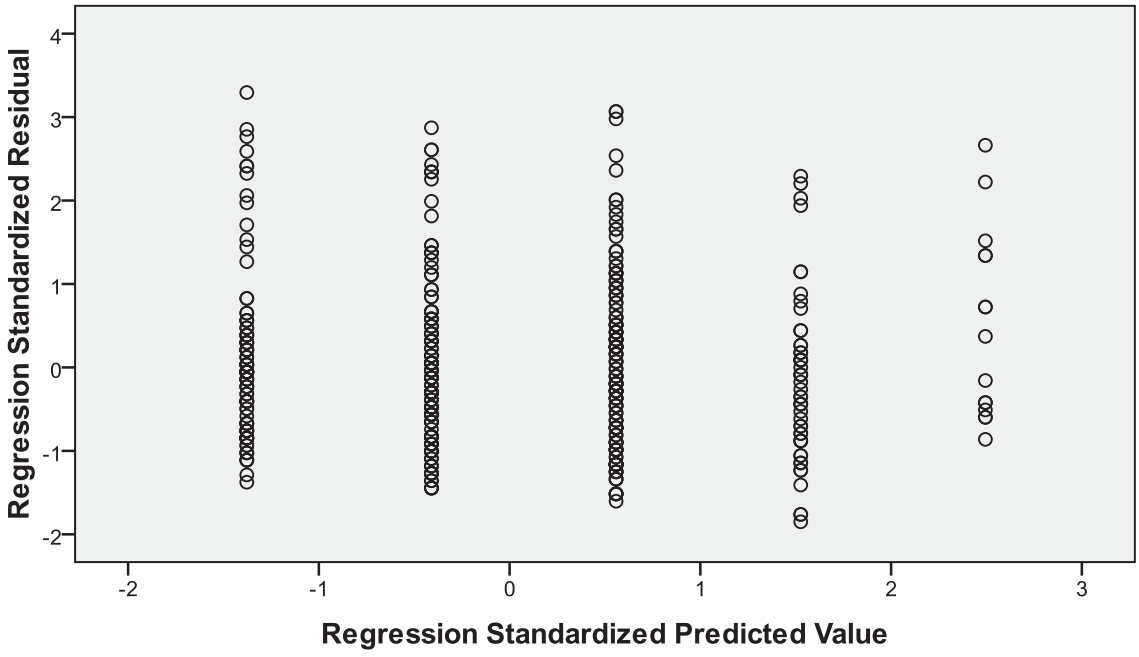


Figure E12: Normal P-P standardised residual scatter plot for HRS-II (Item 3 - Difficulty) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

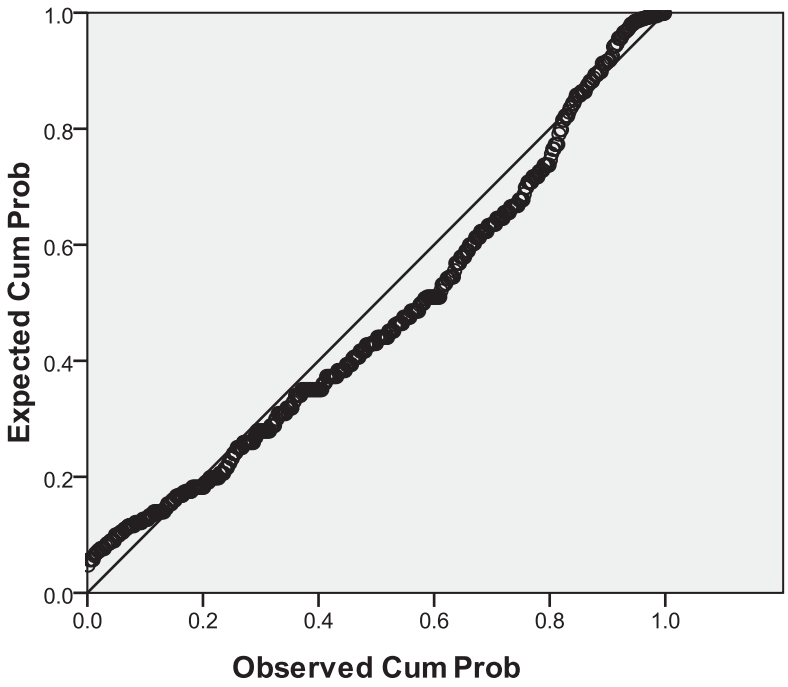


Figure E13: Normal P-P standardised residual plot for HRS-II (Item 4 – Obstacles) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

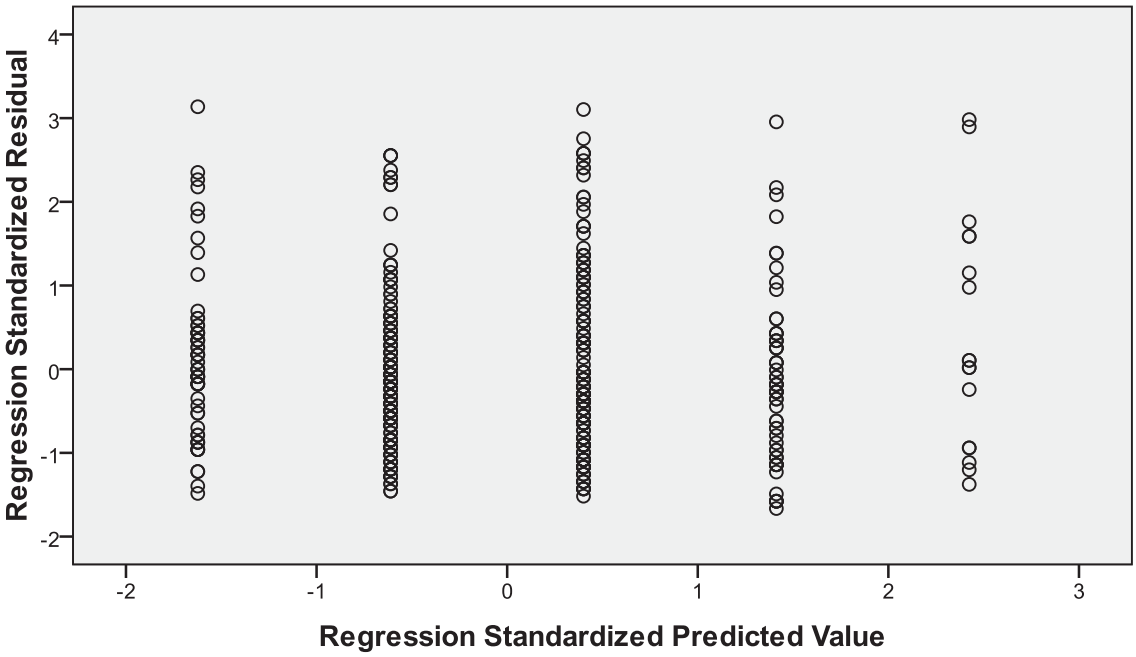


Figure E14: Normal P-P standardised residual scatter plot for HRS-II (Item 4 - Obstacles) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

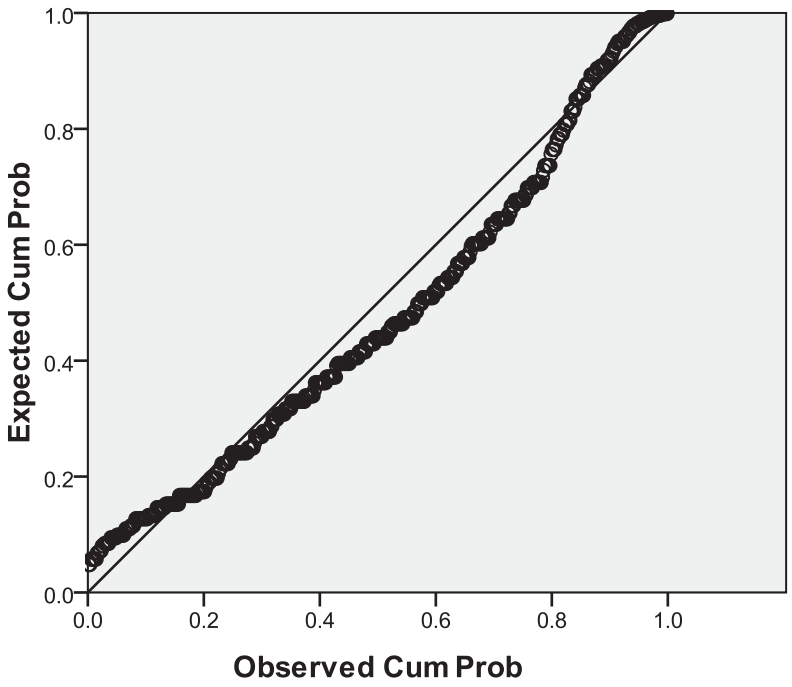


Figure E15: Normal P-P standardised residual plot for HRS-II (Item 5 – Comprehension) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

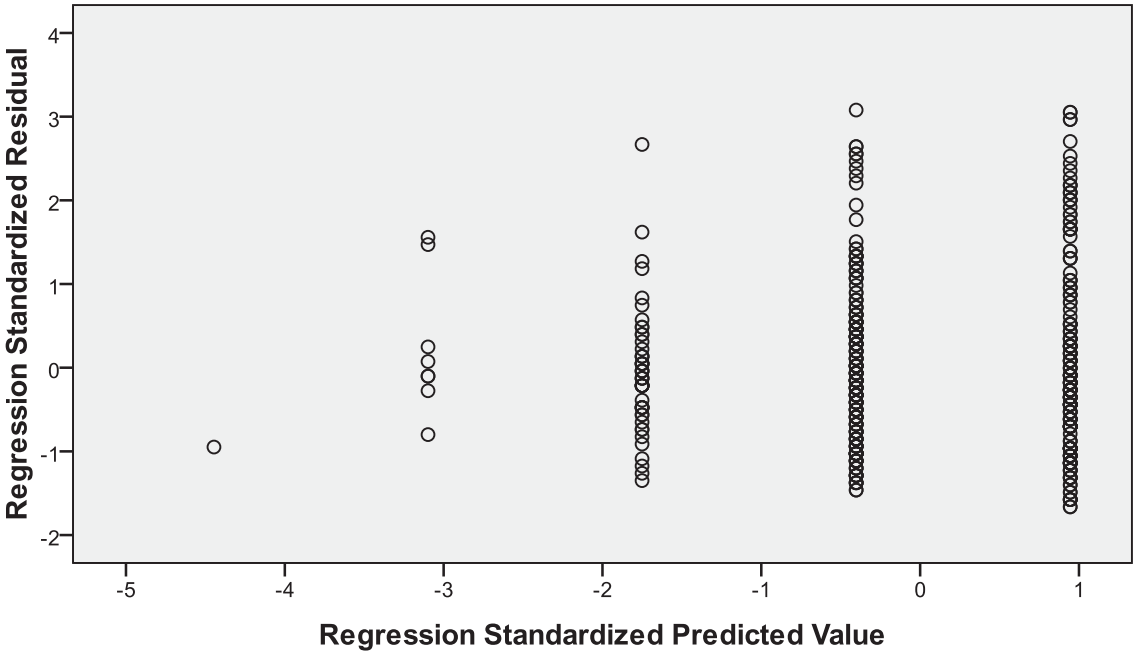


Figure E16: Normal P-P standardised residual scatter plot for HRS-II (Item 5 - Comprehension) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

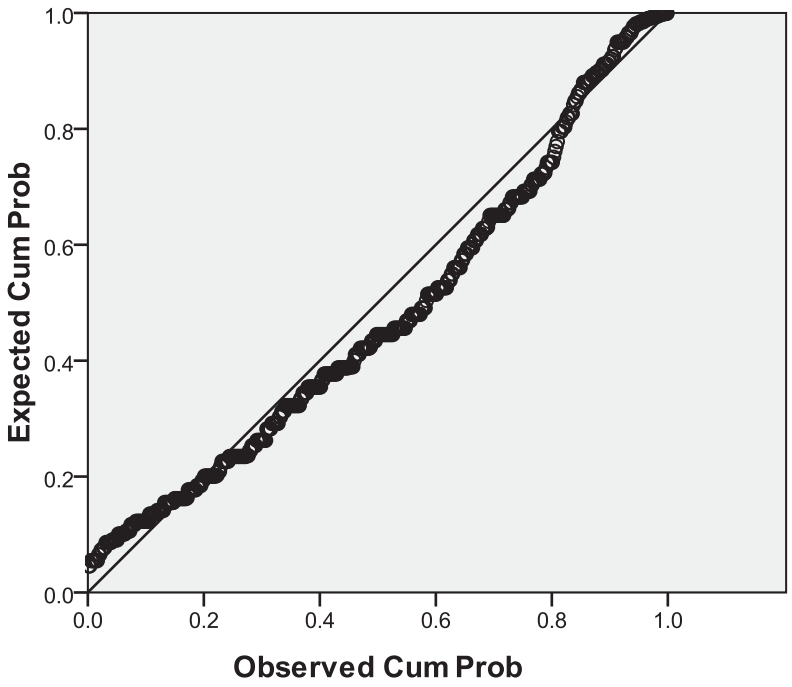


Figure E17: Normal P-P standardised residual plot for HRS-II (Item 6 - Rationale) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

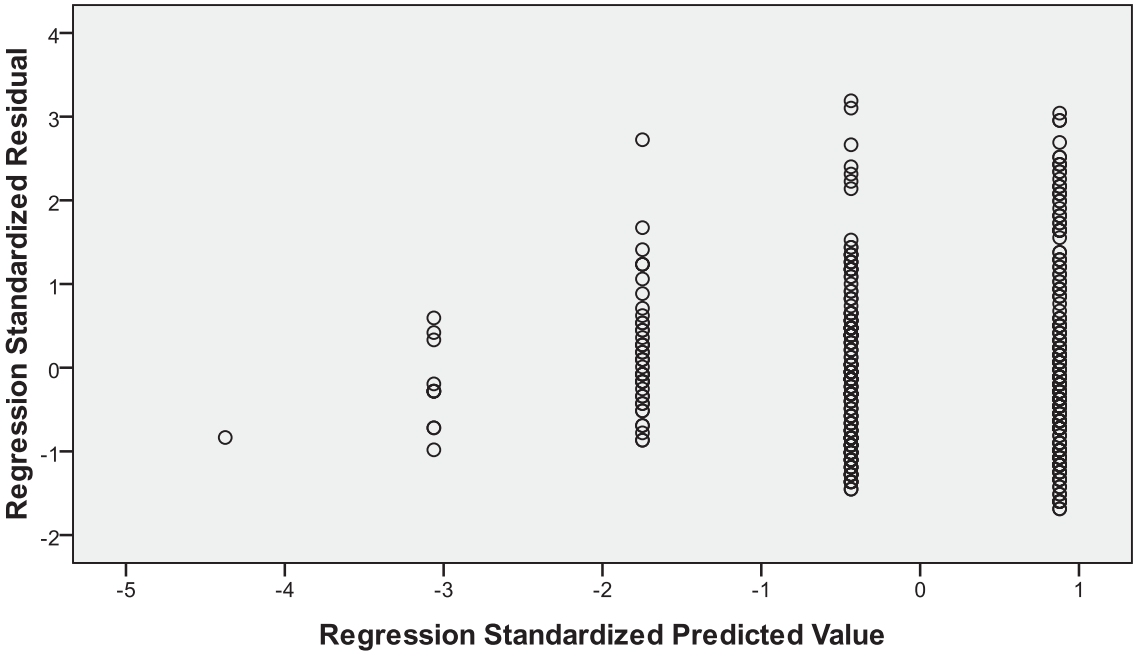


Figure E18: Normal P-P standardised residual scatter plot for HRS-II (Item 6 - Rationale) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

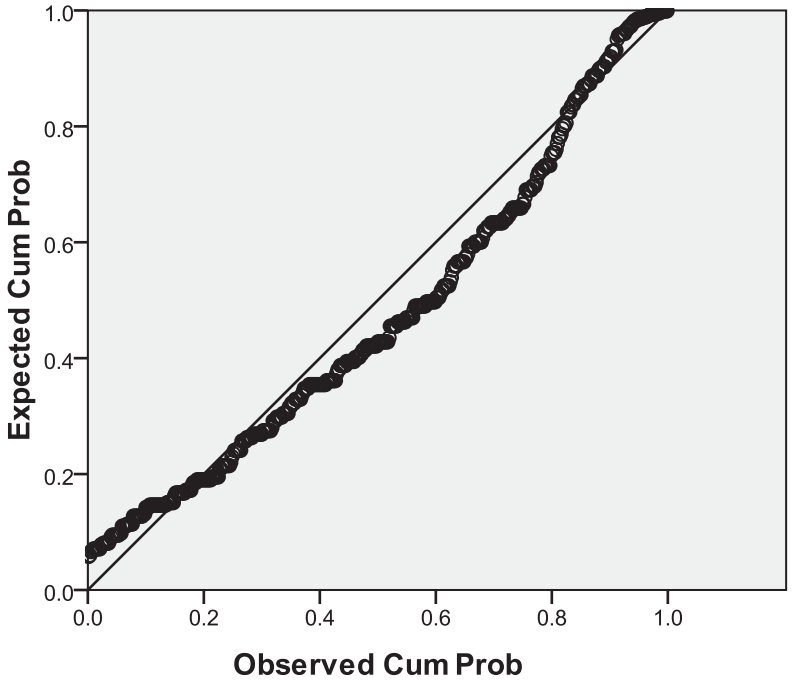


Figure E19: Normal P-P standardised residual plot for HRS-II (Item 7 - Collaboration) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

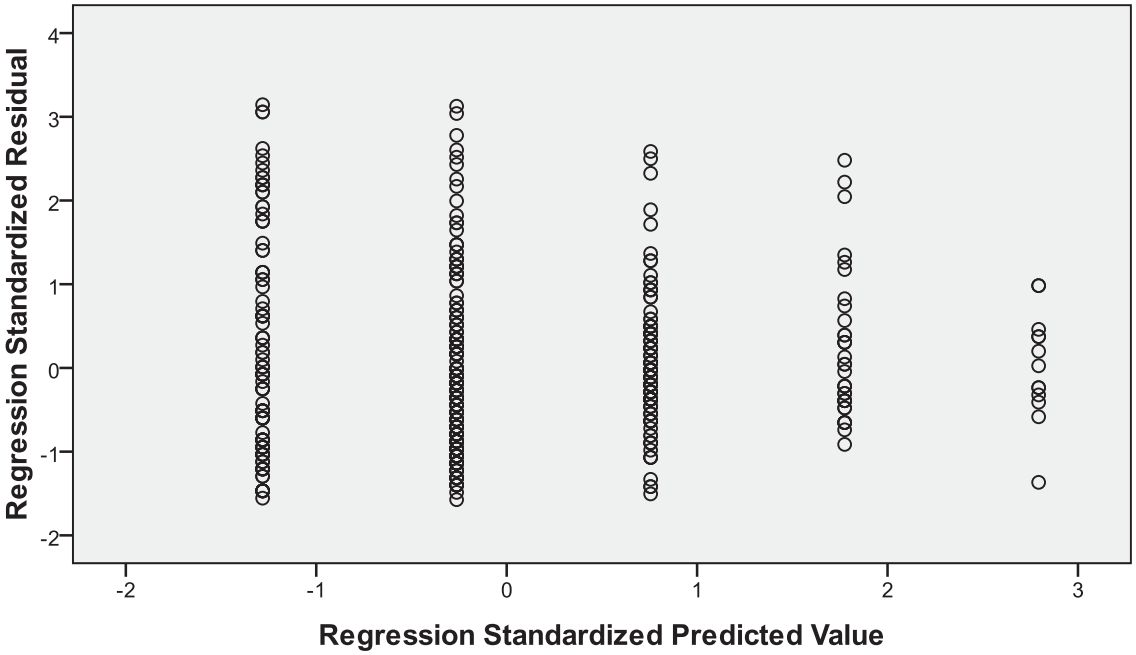


Figure E20: Normal P-P standardised residual scatter plot for HRS-II (Item 7 – Collaboration) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

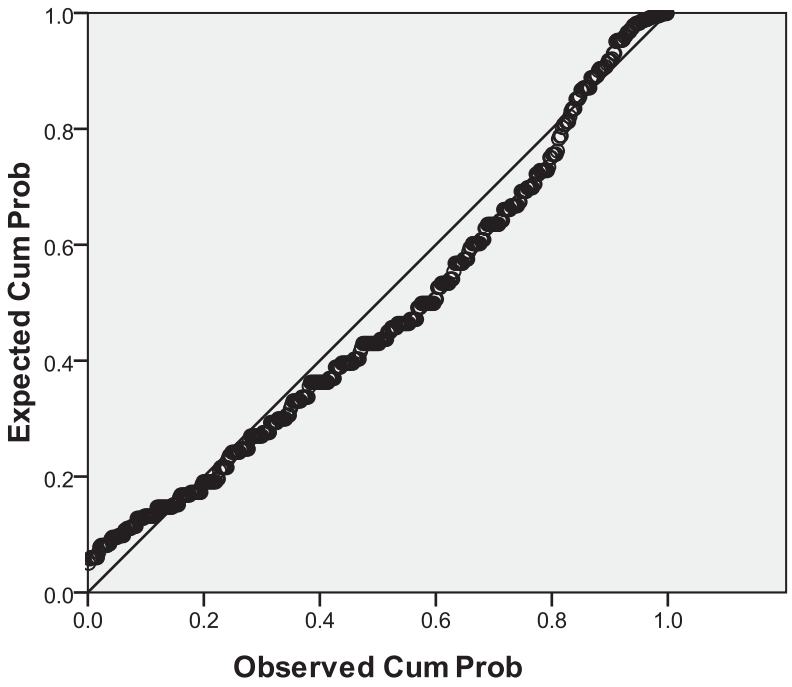


Figure E21: Normal P-P standardised residual plot for HRS-II (Item 8 - Specificity) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

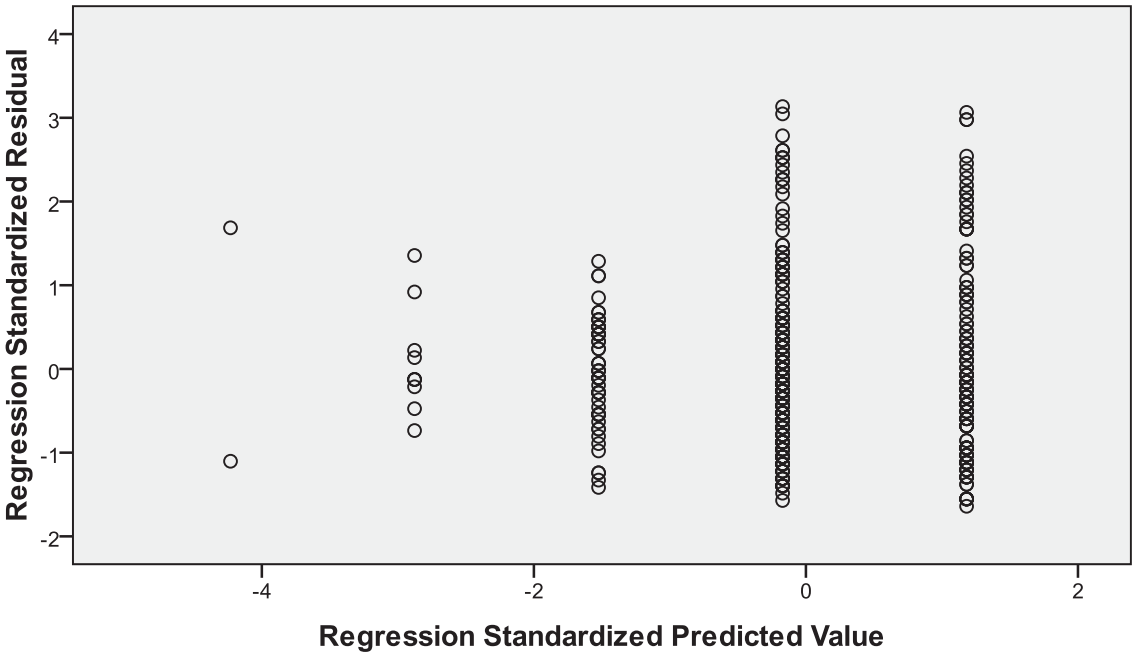


Figure E22: Normal P-P standardised residual scatter plot for HRS-II (Item 8 - Specificty) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

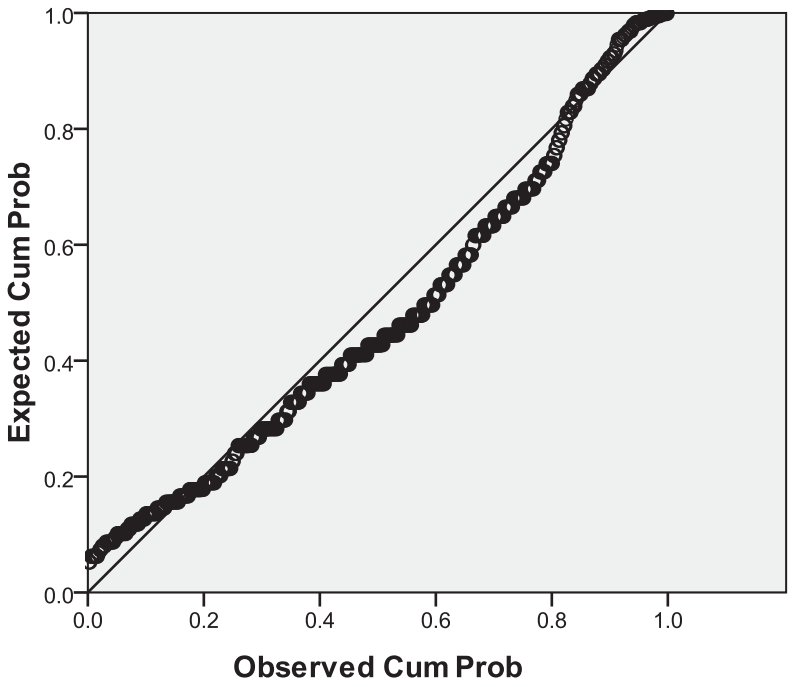


Figure E23: Normal P-P standardised residual plot for HRS-II (Item 9 – Match with therapy goals) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

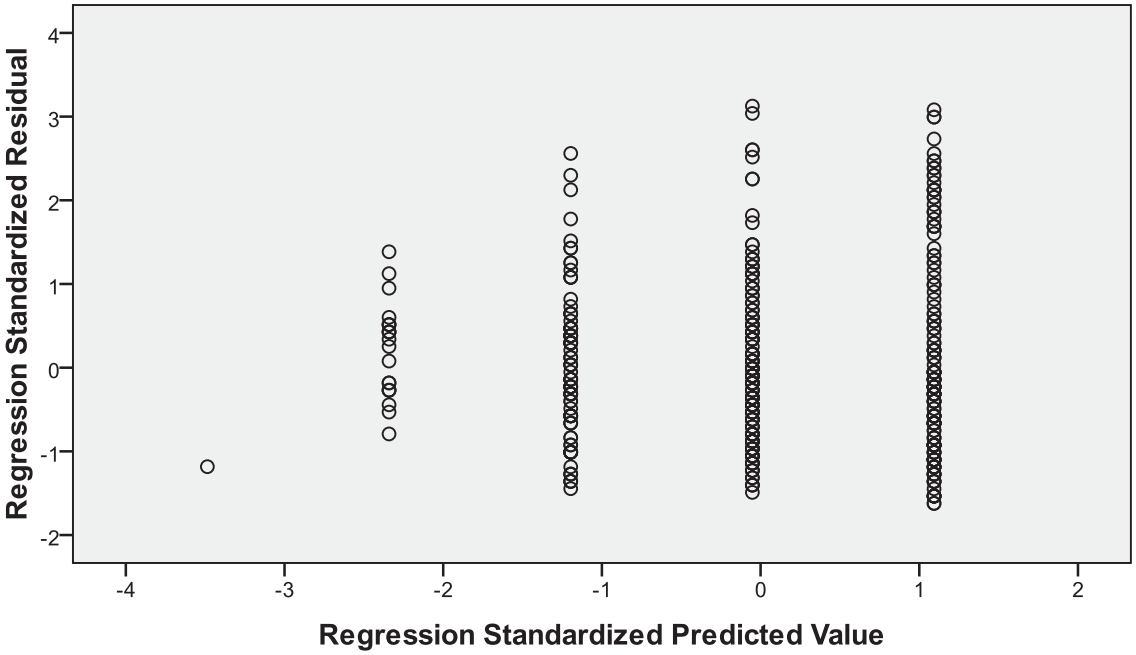


Figure E24: Normal P-P standardised residual scatter plot for HRS-II (Item 9 - Match with therapy goals) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

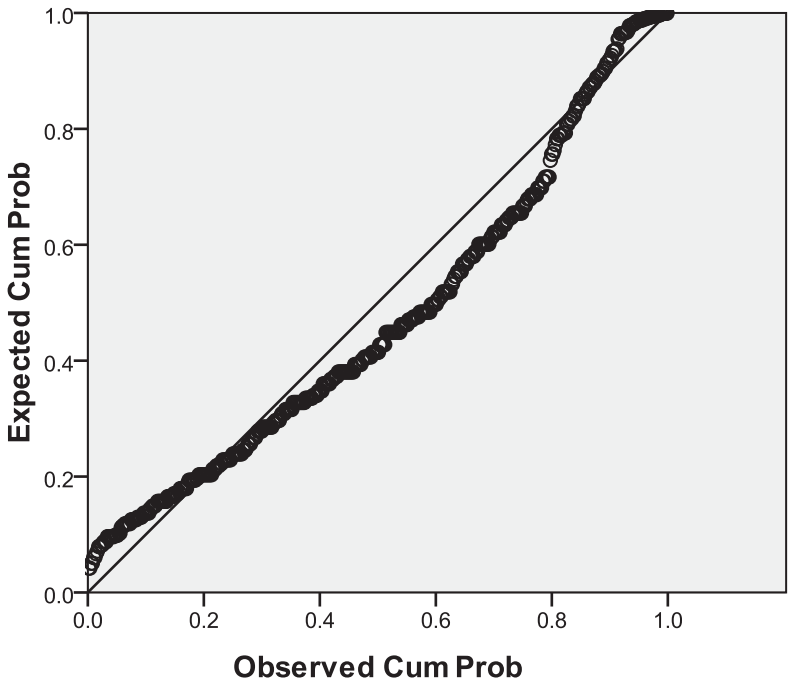


Figure E25: Normal P-P standardised residual plot for HRS-II (Item 10 - Pleasure) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

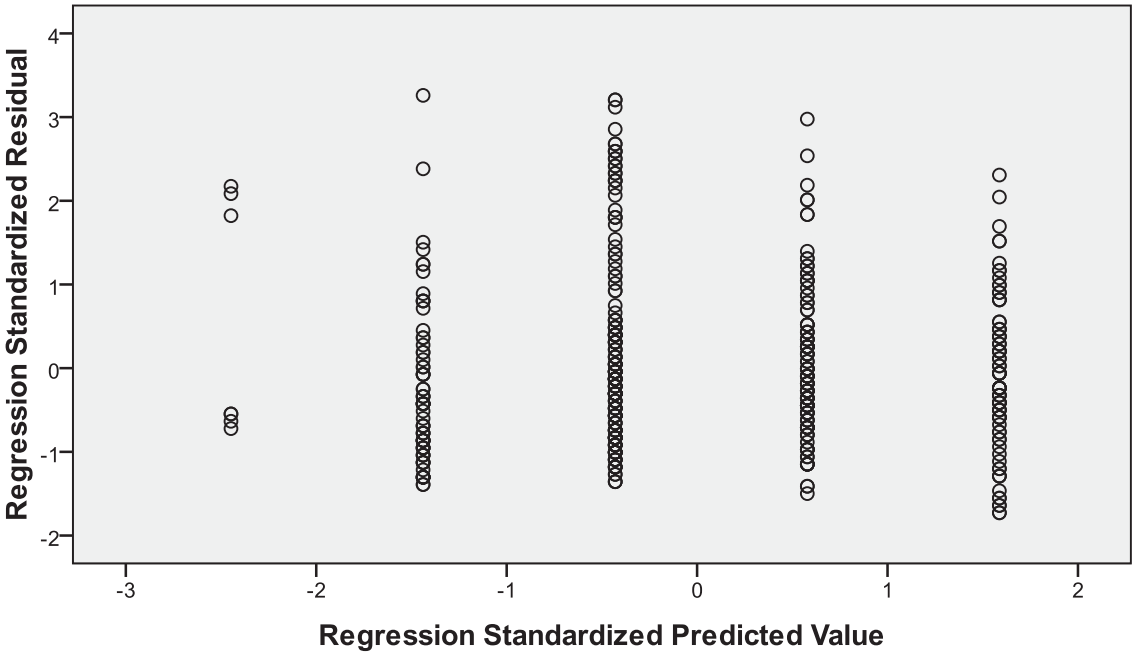


Figure E26: Normal P-P standardised residual scatter plot for HRS-II (Item 10 - Pleasure) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

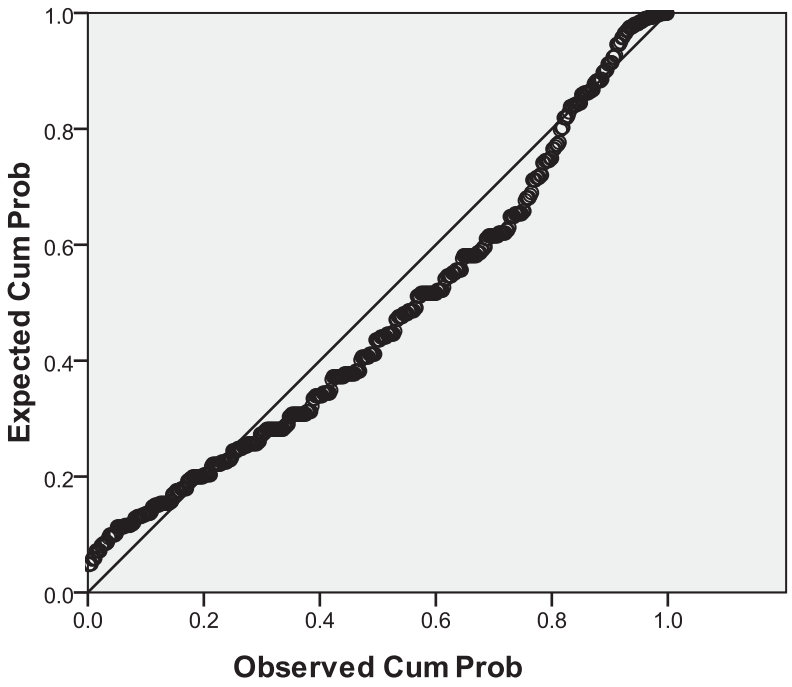


Figure E27: Normal P-P standardised residual plot for HRS-II (Item 11 - Mastery) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

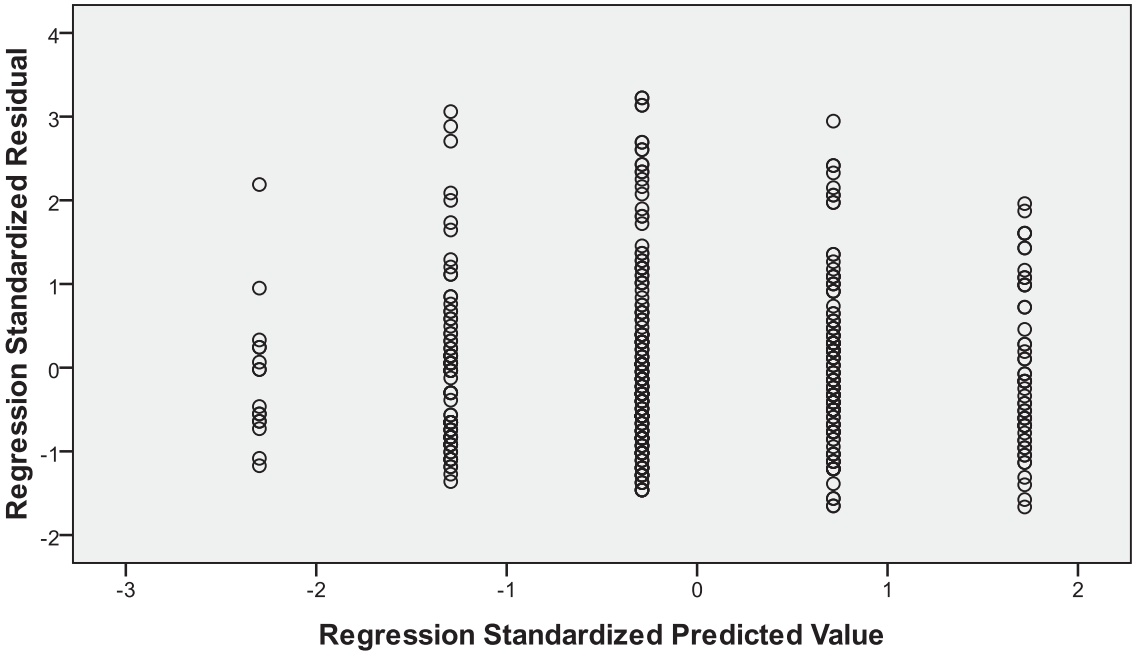


Figure E28: Normal P-P standardised residual scatter plot for HRS-II (Item 11 - Mastery) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

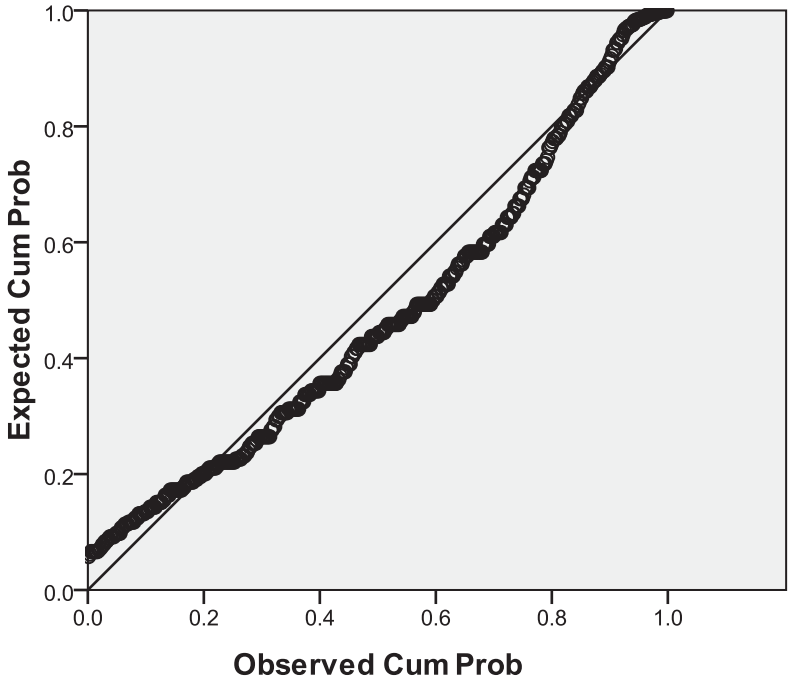


Figure E29: Normal P-P standardised residual plot for HRS-II (Item 12 - Progress) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

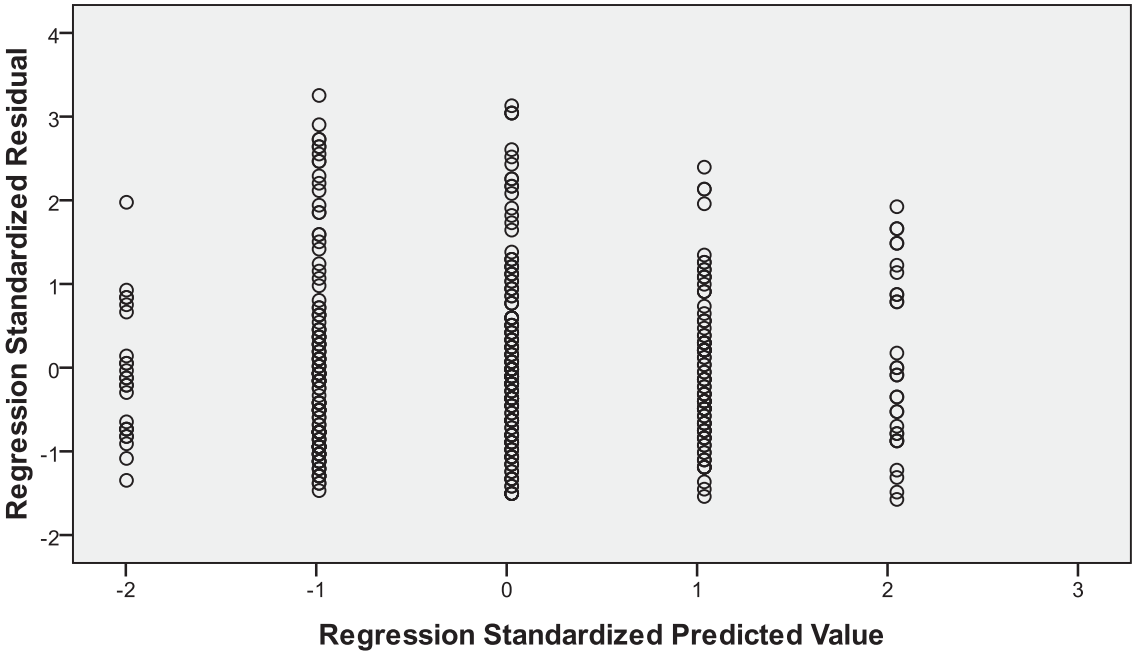


Figure E30: Normal P-P standardised residual scatter plot for HRS-II (Item 12 - Progress) across time. *N.B.* – scores are regressed against BDI-II as the primary dependent variable of the study.

APPENDIX F

Reliability analyses

Table F1

Reliability of the ASQ across four time points in therapy

Session	N	α	M	SD
		CoPos		
0	28	.64	86.93	10.77
5	28	.54	85.89	9.54
8	27	.61	88.63	8.86
20	20	.81	92.69	11.95
		CoNeg		
0	28	.85	90.42	15.06
5	28	.80	84.72	13.70
8	27	.86	85.44	13.65
20	20	.67	77.43	9.98

Table F2

Reliability of the HRS-II (Client Version) across 18 time points in therapy

Session	N	α	M	SD
2	28	.67	26.71	4.9
3	28	.73	27.29	5.7
4	28	.64	28.50	5.0
5	28	.71	27.43	5.4
6	28	.71	27.18	5.3
7	27	.61	26.81	4.7
8	27	.73	28.22	5.2
9	26	.78	25.88	6.2
10	26	.82	27.62	6.2
11	26	.82	26.81	6.4
12	24	.84	28.38	6.3
13	25	.78	28.24	6.6
14	22	.85	30.41	6.3
15	21	.78	30.05	6.6
16	21	.80	28.24	6.5
17	21	.86	30.67	6.7
18	20	.76	30.20	6.0
19	19	.93	28.68	9.6
20	18	.82	31.00	6.4

APPENDIX G

BDI-II scores across therapy

Table G1

BDI-II scores at intake, end of therapy, and follow up sessions

	BDI-II Score				*Percentage change*		
ID	**Intake**	**End of therapy**	**2 month follow-up**	**6 month follow-up**	**End of therapy**	**2 month follow-up**	**6 month follow-up**
57	21	4	-	-	81%	-	-
63	28	15	13	11	46%	54%	61%
66	33	12	-	-	64%	-	-
69	46	0	1	-	100%	98%	-
84	15	1	-	-	93%	-	-
106	32	6	-	-	81%	-	-
111	24	6	-	-	69%	33%	-
113	33	22	22	-	33%	-	-
116	50	24	-	-	52%	84%	-
133	43	11	7	-	74%	-	-
143	18	8	-	-	56%	57%	-
160	37	18	16	-	51%	-	-
164	32	14	-	-	56%	88%	81%
165	26	2	3	5	92%	84%	-
169	49	15	8	-	69%	-	-
188	13	4	-	-	69%	-	-
195	23	18	-	-	22%	-	-
206	21	6	-	-	71%	95%	-
218	42	0	2	-	100%	95%	100%
220	19	3	1	0	84%	-	-
223	28	9	-	-	68%	-	-
236	37	3	-	-	92%	41%	-
244	53	31	31	-	41%	-	-
247	23	2	-	-	87%	-	-
262	25	11	-	-	56%	-	-
271	44	15	-	-	66%	42%	8%
273	24	16	14	22	33%	93%	100%
295	30	4	2	0	87%		
Mean	*31*	*10*	*10*	*7.6*	*67.6*	*72*	*70*
SD	*11.3*	*8.0*	*9.6*	*9.2*	*21.0*	*24.6*	*38.2*