UNIVERSITY OF SOUTHAMPTON

FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES

School of Psychology

Acceptance and Commitment Therapy (ACT) Process and Outcome: A Systematic Evaluation of ACT for Treatment Resistant Patients

by

Jessica Kingston

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ABSTRACT

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Although traditional Cognitive Behaviour Therapy (CBT) has achieved many clinical successes, approximately 30-50% of patients are resistant to this form of treatment. This trans-diagnostic group of *treatment resistant* patients typically have chronic, co-morbid, and/or personality disordered symptoms and often engage in a range of maladaptive behaviours (e.g., substance abuse, deliberate self-harm). Acceptance and Commitment Therapy (ACT) is a relatively modern psychological treatment which proposes that the formally dissimilar symptoms this group present result from a common cause; namely, excessive entanglement with, and a need to escape from or avoid, unwanted private events such as thoughts, feelings, and memories (*experiential avoidance*). Preliminary evidence from clinical trials suggest that ACT may prove efficacious with treatment resistant patients.

In this thesis, four studies were designed to examine the theoretical underpinnings and clinical utility of ACT. Studies 1 and 2 tested the ACT-derived prediction that diverse maladaptive behaviours serve a common experiential avoidance function. In support of this hypothesis, structural equation modelling showed that experiential avoidance predicted significant maladaptive behaviour covariance. Moreover, using the same method, a cross-sectional design showed that experiential avoidance partially mediated the effect of Negative Affect Intensity and Childhood Trauma on the tendency to engage in maladaptive behaviours. Studies 3 and 4 extended these theoretically-based investigations into the applied domain, pilot testing ACT for a sample of patients whose symptoms had been resistant to, or relapsed following, standard care. Study 3, a pre-post uncontrolled trial, revealed significant reductions in psychological distress with gains maintained at 6 and 12-month follow-up. Study 4, a randomised control trial comparing ACT to a CBT treatment as usual (CBT-TAU) condition, showed that ACT achieved more enduring effects than CBT-TAU. Furthermore, exploratory analyses suggested that, for the ACT group alone, reductions in experiential avoidance during treatment predicted follow-up outcomes. These findings support the use of ACT for treatment resistant patients.

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DECLARATION OF AUTHORSHIP

I, **Jessica Kingston**, declare that the thesis entitled

Acceptance and Commitment Therapy (ACT) Process and Outcome: A Systematic Evaluation of ACT for Treatment Resistant Patients

and the work presented in the thesis are both my own, and have been generated by me as the result of my own original research.

I confirm that:

- this work was done wholly or mainly while in candidature for a research degree at this University;
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- where I have consulted the published work of others, this is always clearly attributed;
- where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;
- I have acknowledged all main sources of help;
- where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;
- none of this work has been published before submission.

Signed: ……..

Date: …….....

ACKNOWLEDGEMENTS

This thesis is dedicated to the loving memory of my sister and my father

Natalie Elizabeth Kingston

(05.11.1979 - 11.05.2008)

Richard John Kingston

(05.06.1934 - 13.04.2001)

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CHAPTER I

A Short History of Behaviour Therapy

For many decades, experimental psychologists have worked to discover the basic mechanisms underlying human action. Often these endeavours have involved analogue research either investigating animal behaviour in laboratory conditions, or human behaviour in carefully controlled and artificial test situations. One aim of these efforts has always been to generate knowledge that can be applied to human behaviour in real world settings and, in particular, to the understanding and treatment of human clinical disorders. The purpose of this chapter is to describe how this goal has been realised through the development of psychological therapies, which have largely focused on addressing the needs of certain clusters of psychological symptoms (e.g., symptoms of depression or of anxiety). The underlying position from which this chapter is written is *radical behaviourism*, the theoretical approach that informs empirical sections of this thesis.

1.1 First Wave Behaviour Therapies: Behaviour Analysis and Intervention

Early behaviourists used animal analogue research to study learning principles. This resulted in the detailed knowledge of two fundamental learning processes: classical conditioning (Pavlov, 1927) and operant conditioning (Skinner, 1938). In both instances, conditioning described the acquisition of distinct behaviour patterns whose occurrence was dependent on their relationship with environmental stimuli (Catania, 1979).

1.1.1 *Respondent Conditioning and Behaviour Therapy*

Early behaviour therapy had firm scientific foundations in work pioneered by Russian physiologists at the turn of the 20th Century. This work was committed to scientifically assessing the effect of the central nervous system on behaviour (e.g., Pavlov, Sechenov, & Bechterev; cited in Kazdin, 1978). The most famous of these endeavours was Pavlov's work on conditioned reflexes in dogs, which was specifically concerned with conditioning salivation. Pavlov (1927) showed that pairing a stimulus that elicited a

reflex (an unconditioned stimulus; UCS) with a neutral stimulus (a conditioned stimulus; CS) established a *conditioned reflex* (CR); the previously neutral stimulus acquired the capacity to elicit the reflexive behaviour. For example, by repeatedly pairing a tone (CS) with food (UCS), the tone acquired the capacity to elicit salivation (CR) even in the absence of food (Pavlov, 1927). This process came to be known as respondent conditioning[1].

Pavlov's work was taken up by American psychologists as a possible mechanism for explaining human behaviour, informing an approach to psychology called *methodological behaviourism*. This approach upheld Pavlov's scientific principles, banning introspection and insisting that overt and quantifiable behaviour was the only admissible form of data. Respondent conditioning provided a central theoretical and methodological model for a wealth of research on *experimental neuroses*; the production of behavioural disorders through laboratory-based procedures. John B. Watson's research was particularly influential, demonstrating how phobias (or *conditioned emotional reactions*) could be created using respondent conditioning methods in humans. For example, his famous "Little Albert" experiment (Watson & Rayner, 1920) showed that pairing the presentation of a white rat (CS) with an unexpected loud noise (UCS) resulted in experimentally induced fear (CR) on later exposures to that same rat. After seven CS and UCS pairings, the child who initially played freely with the rat was now reported to fall over, cry and crawl away when the rat was brought towards him. Although experimentally induced, Albert's fear was similar to fears seen in clinical practice. For example, fear generalised to previously neutral stimuli and was durable over a 4-month period, despite the absence of further conditioning trials.

Based on the understanding that clinical disorders were learnt reflexive behaviours, respondent conditioning became a theoretical wellspring for early behaviour therapies. These therapies shared the common principle that clinical disorders could be modified or eliminated using further conditioning. For example, laboratory evidence of counter-conditioning and reciprocal inhibition (Wolpe, 1958, first documented in Watson's

[1] Pavlov initially used the phrase "conditioning" which later became known more specifically as "classical conditioning" for occasions where the behaviour was not instrumental. Skinner (1938), however, renamed classical conditioning "respondent" conditioning as a contrast term for operant or instrumental conditioning. For consistency, respondent conditioning will be used from this point forward.

laboratory in 1924; see Harris, 1979) revealed that if a response incompatible with the CR could be made to occur in the presence the CS that elicited it, the previously established CR would weaken or be eliminated. Based on this observation, Wolpe (1961) designed systematic desensitisation as a treatment for anxiety-based disorders. This involved pairing relaxation (induced, for example, by using anxiolytic drugs or hypnosis) with graded exposure to anxiety eliciting stimuli. This technique proved particularly successful for the treatment of anxiety-based disorders and lasting effects were often reported (see Bandura, 1969; Eysenck & Rachman, 1965). Although systematic desensitisation traditionally relied on exposure to physical stimuli, over time its application extended to include exposure to *imagined* hierarchies of anxiety provoking events. This broadened its range of applicability greatly, enabling the treatment of anxieties concerning CS that were not readily accessible to the clinical setting (e.g., the fear of flying).

Systematic desensitisation was one of many first wave behaviour therapies, with others including (detailed in, and cited from, Eysenck & Rachman, 1965), for example, aversion therapy (Franks, 1958), conditioned inhibition (Walton, 1961), vocal inhibition (thoughts stopping; Wolpe, 1958), conditioned avoidance (Hilgard & Marquis, 1940), negative practice (Walton & Black, 1960) and implosion therapy (Page & Hall, 1953).

1.1.2 *Operant Conditioning and Behaviour Modification*

Although respondent conditioning provided the initial foundations for early behaviour therapy, it was somewhat limited by its focus on reflexive behaviours. Watson and colleagues regarded all learning as instances of respondent conditioning, but failed to provide a convincing account of more complex, non-reflexive patterns of behaviour, or of clinical disorders that had no obvious learning-based etiology (Rachman, 1977). Furthermore, incongruence between laboratory-based and clinical-based investigations began to emerge. For example, respondent conditioning failed to explain why experimentally induced CRs could be readily extinguished, whereas those seen in clinical practice were more resistant to change (Rachman, 1977).

The work of B. F. Skinner, a *radical behaviourist*, broadened the field of behaviour therapy considerably. Skinner distinguished a different kind of behaviour—*Operant Behaviour*—behaviour that was a function of its consequences (Skinner, 1938). Unlike

respondent behaviour, which was "involuntarily" *elicited* by certain environmental stimuli, operant behaviour was said to be "voluntarily" *emitted* by the organism, and maintained by its effects on the environment (Catania, 1979)[2]. Operant behaviour was described in terms of two hierarchically nested contingencies. The elements of this contingency included (a) a discriminative stimulus (S^D), (b) an operant behaviour (or the response; R), and (c) a reinforcing stimulus (Rf). These were nested in the following way; the response to reinforcement contingency was first established (R →Rf) and any context reliably present during this pairing, over time, came to function as an S^D (S^D (R →Rf)). The S^D was said to have *stimulus control* over responding because it set the occasion for certain behaviours to be emitted. Skinner also emphasised that no behaviour is ever precisely replicated, and that the same consequence can be achieved by topographically dissimilar behaviour patterns. Skinner thus defined operant behaviour in terms of *response classes*. Responses belonged to an operant class if they shared the property required to obtain reinforcement (see Kazdin, 1978).

Although Skinner's research was almost exclusively analogue, restricted to the behaviour of non-human organisms, it had a significant impact on behaviour therapy. Understanding behaviour in terms of response classes, rather than isolated reflexive behaviours, greatly broadened the range of behaviours that could be investigated and captured the complexity of human action more completely. Operant principles enriched models of clinical disorders by suggesting that behavioural problems could be established and maintained by reinforcement contingencies. This resulted in new theoretical models, most of which were hybrids of respondent and operant conditioning (Eysenck, 1960; Eysenck & Rachman, 1965; Mowrer, 1960). Mowrer, for example, developed a two-stage account of avoidance in which respondent conditioning was the process responsible for *establishing* a fear, whereas operant conditioning maintained avoidance behaviour as a form of escape from conditioned fear. In this account, the CR was maintained because an operant class of escape behaviours prevented the natural extinction of fear through exposure to the CS alone.

In clinical practice, Skinner's theorising enhanced existing techniques and informed new ones. Treatments based on Skinner's ideas were broadly described as *behaviour modification* techniques, which shared the common principle that disorders could be

[2] Although the terms of voluntary and involuntary are useful heuristics for understanding the distinction between these two types of conditioning, they did not survive as defining characteristic.

treated by manipulating the consequences of behaviour. This could be done either by modifying the contingencies that maintained then or by introducing new contingencies to shape adaptive operant behaviour. For example, contingency management established behaviour change using selective reinforcement procedures with successive approximation; that is, positively reinforcing adaptive behaviour (or approximations to it) and extinguishing problem behaviour (e.g., Stitzer, Bigelow, & Liebson, 1979). Other techniques informed by Skinner's theorising included (non-exhaustively) extinction (e.g., Nawas & Braun, 1970), shaping (e.g., Wolff & Perkins, 1970), and the token economy (Ayllon & Azrin, 1968).

1.1.3 *Limitations of First Wave Behaviour Therapies*

Behaviour therapy techniques varied in their effectiveness when applied to the treatment of several disorders. These included phobias (Wolpe, 1953), hysteria (Brady & Lind, 1961), enuresis (Mowrer, 1938), tics (Yates, 1958), substance abuse disorders (Azrin, 1976) and childhood disorders (Rachman, 1962; see Eysenck & Rachman, 1965; Kazdin, 1978, for reviews). Despite several successes, however, these approaches were at their most effective when applied to individuals with developmental disorders and severe adult mental health problems (e.g., Lovaas, Koegel, Simmons, & Long, 1973; Lovaas, Schreibman, & Koegel, 1974; Ward, 1978). This unevenness of application stemmed from the fact that the behavioural therapies encountered several complications when dealing with less disturbed patients. Often it seemed as though treatments were only behavioural by virtue of analogy. For example, systematic desensitisation often employed techniques that involved exposure to imagined cues rather than to actual cues (e.g., Marks, 1976; see also behaviour therapy for depression, Lazarus, 1968). This was problematic for methodological behaviourists, who could not easily account for the effect of these non-observable events.

Additionally, patients often failed to respond to direct contingencies, responding instead to their verbal account of those contingencies (e.g., see Kazdin, 1978, p. 299). For example, clinical observations showed that patients tended to behave in accordance

with their perceptions or cognitions [3] (i.e., expectations, beliefs, attitudes) regarding reinforcement contingencies, rather than with the actual contingencies (e.g., see Bandura, 1974; Kazdin, 1978). Alluding to these observations, the influential theorist Bandura (1974) wrote "Contrary to the mechanistic metaphors, outcomes change behaviour in humans through the intervening influence of thought... Our choices of action are largely under anticipatory control. The widely accepted dictum that man is ruled by response consequence… fares better for anticipated than for actual consequences…When belief differs from actuality, which is not uncommon, behaviour is weakly controlled by its actual consequence…" (p. 859-860).

These ideas were reflected by many during the '70s. Theorists argued that arranging reinforcement schedules to shape adaptive responding was not sufficient to promote behaviour change. Rather, they proposed concepts such as 'self-efficacy', 'schemas' and more generally 'cognitions' as mediators of contingency relations (Bandura, 1969, 1974; Beck, 1970, described below). During this theoretical debate, human operant laboratory-based research on behaviour emerged. This suggested that, unlike animal behaviour, human behaviour did not come under the direct control of operant schedules (e.g., Brewer, 1974) and thus, that the manipulation of behavioural consequences did not automatically affect behaviour (Dulany, 1968). These findings were interpreted by more cognitively minded theorists as supporting theories of cognitive mediation, contributing to the growing view that behavioural theorising was too simplistic an account of human action.

It is worth noting that during this time, some behaviourists had already begun to formulate theories addressing the effect of cognition (verbal behaviour; VB) on action. For example, Skinner had spent over 10 years extending his ideas into the field of VB, which he considered to be a form of operant behaviour. Unlike cognitive psychologists, who looked to cognition as an explanation of action (i.e., cognitions as independent variables), Skinner suggested that cognitions were instances of VB that were in need of explanation (i.e., cognitions as dependent variables). Although his theorising (Skinner, 1957, 1969) could have informed the problems that behaviour therapy was experiencing in the clinic, (e.g., rule-governed behaviour, see section 1.2.2 & section 2.1.1) it failed

[3] Consistent with cognitive theorists (e.g., Beck, 1970), the term "cognition" is used to refer to cognitive products such as thoughts, feelings, and attitudes. For Skinner, these were forms of verbal behaviour. Both phrases are purposefully used so as to mark the distinction between the two theoretical orientations.

to engage non-behavioural audiences and thus had limited clinical impact (Hunt, 1984; Scandura, 1984). Instead, the rise of cognitive psychology in the late ’50s and early ’60s fuelled a new approach to the treatment of clinical disorders.

1.2 Second Wave Behaviour Therapies: Cognitive Experimental Psychology and Cognitive Interventions

Despite the successes of early behaviour therapies, and emerging accounts of how language affected operant conditioning, behavioural approaches were rapidly superseded by a less constrained and a theoretically richer approach: c*ognitive experimental psychology*.

1.2.1 C*ognitive Experimental Psychology and Cognitive Behaviour Therapy*

Profiting from novel methodologies, cognitive experimental psychology rapidly came to dominate experimental research, investigating higher order human activities such as memory (e.g., Hitch & Baddeley, 1976) and perception (e.g., Bruce & Young, 1986). Although theoretical accounts of these phenomena developed in a piecemeal fashion, the computer analogy was a common feature. The human mind was conceptualised as a limited-capacity information processing system that operated on inputs (i.e., stimuli) to produce outputs (i.e., behaviour). The logic of this approach, known as *cognitivism*, was to reverse engineer the mind, studying patterns of inputs and outputs and hypothesising cognitive mechanisms that might account for regularities observed. To avoid the problem of tautology, convergent evidence was a necessary criterion for selecting amongst a range of possible causal mechanisms.

The success of cognitivism in experimental psychology had an unexpected impact on behaviour therapy, in part because of the limited success that behaviour therapy had had with mature adult patients. The fact that patients responded to the world that they reported rather than the world as it was, was deemed to reflect the action of their *cognitive system*. Thus, cognitive experimental psychology provided an opportunity to introduce cognitive concepts into the clinical setting. One of the most influential theorists of this movement was Aaron Beck. Beck’s work began with the clinical observation that depressed patients showed selective attention to negative information,

and negativity bias when processing and interpreting that information (see Leahy & Dowd, 2002). Within the broad cognitivism movement, Beck interpreted these observations as the actions of cognitive schema: "a cognitive structure for screening, coding and evaluating the stimuli that impinge on the organism" (Beck, 1967, p. 283). Schemas, thought to develop through experience, were seen as self-perpetuating cognitive maps that prioritised attention to specific types of stimuli for encoding and processing.

Because cognitive schemas were not directly accessible, Beck relied on abstraction and inference rather than direct observation (Leahy & Dowd, 2002). Inferences regarding the nature of cognitive schemas were made based on patients' self-statements or cognitions, referred to broadly as "inferred psychological states… thoughts, attitudes and the like" (Beck, 1970, p. 193). Because these were thought to provide a window of insight into internal cognitive structures, the "patient's spontaneous experience and self-reported thoughts" (Beck, 1970, p. 187), rather than their behaviour, became the object of clinical interest. According to Beck and fellow cognitivists of that time, cognitions caused behaviour; people behaved in certain ways because of the intervening role of thought. As such, cognitive interventions were based on the logic that modifying the patient's cognitions would effect a change in behaviour (e.g., Beck, 1970). This approach to the treatment of clinical disorders came to be known as *Cognitive* Behaviour Therapy (CBT)[4].

Techniques devised by Beck, such as cognitive restructuring and reality hypothesis testing, aimed to correct erroneous thinking styles by "substituting irrational thoughts with rational ones" (Hofmann & Asmundson, 2008, p. 4). CBT taught (and teaches) patients to identify automatic idiosyncratic misconceptions about themselves, their world, and their future, and trained them to evaluate and challenge the validity of those cognitions. For example, in reality hypothesis testing, the therapist and patient collaboratively devise experiments designed to test out the correspondence between anticipated and real outcomes. Such experiments are used to collect evidence for and against the validity of the patient's core beliefs, with the intention of bringing those beliefs back into line with reality. The common principle to cognitive techniques,

[4] Beck initially named his treatment Cognitive Therapy (Beck, 1970). As his techniques expanded their range of application to disorders other than depression, however, this more generic term was adopted within the literature. The broader term is used throughout.

therefore, was that correcting faulty cognitions was necessary for, and causal in the production of, behaviour change.

Early research on cognitive techniques obtained noticeable success for the treatment of depression, a disorder not well treated by behavioural therapies. For example, in a randomised control trial (RCT; see chapter 3) Blackburn, Eunson, and Bishop (1986) showed that 24-months following treatment, depressed patients who had received CBT were 50% less likely to relapse than patients who had received medication (see also Rush, Beck, Kovacs, & Hollon, 1977; Rush, Hollon, Beck, & Kovacs, 1978). Moreover, although CBT was initially a treatment for depression, a range of disorder-specific CBT manuals followed suit. This included, for example, the application of CBT to patients with Anxiety (e.g., Beck, Emery, & Greenberg, 1985), Alcoholism (e.g., Marlatt, 1979) and Anorexia Nervosa (e.g., Garner & Bemis, 1982). These extensions also had many successes. Indeed, a large scale review of meta-analyses published between 1967 and 2004 showed that, compared to no-treatment, CBT typically obtained medium to large effect sizes for many acute disorders (Butler, Chapman, Forman, & Beck, 2006). These included: uni-polar and bi-polar depression, obsessive compulsive disorder (OCD), anxiety, psychosis, panic disorder, and bulimia.

But to what extent did the success of CBT reflect a more general success for cognitivism? Perhaps less than the common name implied. This is because the link between experimental cognitive psychology (cognitivism) and CBT was in some ways a tenuous one. Cognitivism, based on the computer analogy, had limited time for introspection. For experimental cognitive psychologists, the behaviour of participants reflected processes deemed inaccessible to conscious awareness. Thus, in keeping with the behavioural tradition, cognitivists banned introspection in favour of objective measures such as reaction times. Convergent evidence from multiple measures was necessary for inferring cognitive processes. In contrast, CBT relied heavily on introspection, with therapeutic procedures predominantly concerned with testing the validity of patients' self-reported thoughts and feelings. Indeed, CBT techniques did not actually target processes deemed critical to clinical disorders, but dealt almost exclusively with modifying self-statements (Morris, 1987). Beck's early ideas were thus tautological; patients were depressed because they had negative cognitions/schemas, but they had negative cognitions/schemas because they were depressed. Furthermore, at odds with the scientific objectivism of cognitivism, early CBT lacked a firm theoretical

and empirical base and failed even to define what techniques did and did not fall within the CBT framework (e.g., Wilson, 1978, cited in Clarke & Reinecke, 2003).

Although cognitive techniques obtain some noticeable effects in clinical outcome trials, early cognitive models of clinical disorders were crude and general by comparison to their laboratory-based cousins (Gaudiano, 2008). Indeed, despite their strong criticism of the behavioural approach, early cognitive treatments were criticised for doing little more than adding cognitions to the list of behaviours to be modified (Hayes, 2004; Morris, 1987). In his early theoretical papers, Beck highlighted a need that had been long recognised within behavioural psychology; the need to explicitly accommodate the effect of language on behaviour.

1.2.2 *Radical Behaviourism and "Cognition"*

Radical behaviourist accounts of cognition, including private events and self-talk, were not well understood by second wave cognitive therapists (Hunt, 1984). For example, Skinner's writing on private events (Skinner, 1945) and rule-governed behaviour (RGB; Skinner, 1969), although not without criticism (e.g., Chomsky, 1959), has substantial power in terms of understanding human psychological problems (e.g., see Lowe, 1983; Zettle & Hayes, 1982). Skinner proposed that the verbal actions of a person could be understood as behaviours, established and maintained through operant conditioning. VB was simply defined as behaviour reinforced through the mediation of another person (Skinner, 1957). In a complementary fashion, Skinner (1969) argued that a speaker could provide verbal cues (rules) that had functional control of a listener's subsequent behaviour. Moreover, in Skinner's view, the speaker and the listener could be the same person ("inside the same skin"), so it was reasonable to think that individuals may talk to themselves (e.g., by self-instructing, or self-criticising).

Skinner's ideas can be illustrated by considering how colour names are acquired. For example a child may learn say "green" in the presence of a variety of green objects if prompted and reinforced for doing so by a 'teacher'. Subsequently, the probability of saying "green" when novel green stimuli are encountered will increase. According to Skinner (1945), the process of learning to name could still occur even when the stimuli impinging on the speaker are private events, occurring within the putative speaker's skin, and thus unobservable to the 'teacher' (e.g., feelings of anxiety or depression). For

example, verbal responses to private events may be established and maintained through reinforcement contingent on related public stimuli or responses. Thus, a child may be taught to describe a private event as "a stabbing pain" if the 'teacher' sees it has been caused by a sharp object. This way of talking may subsequently generalise to similar pains with no external referents. Similarly, a child may be taught to name an internal event as "anxiety" by a 'teacher' who has observed her withdrawing from certain situations, speaking tremulously, or shaking involuntarily. According to Skinner, because speakers are conditioned to report private events by the verbal community, they have good reason to "turn this verbal behaviour upon themselves" (Skinner, 1957, p. 192). Skinner suggested that individuals become aware of their own behaviour because the verbal community reinforces "verbal responses with respect to (their) behaviour as the source of discriminative stimuli" (Skinner, 1945, p. 379).

A second key aspect of Skinner's theorising was his distinction between *contingency-shaped* and *rule-governed* behaviour. Skinner argued that although much human behaviour is contingency-shaped by direct contact with environmental contingencies, an important subset is under the control of contingency-specifying stimuli. These words, signs, or signals provided by others describe the relationship between behaviour and its consequences. Actions under the control of such stimuli were said to be rule-governed. New forms of behaviour could thus be established based on specifying contingencies (e.g., "If you exercise regularly, your health will improve"), rather than direct experience (Skinner, 1969). The tendency to follow rules was described as a generalised operant brought about as a result of the reinforcement of a number of specific instances of rule-following. For example, a generalised tendency to follow parental instructions could be established if a child's compliance with many requests such as "do your homework" or "tidy your room" was consistently reinforced (Zettle & Hayes, 1982, see section 2.1). Skinner's conceptualisation suggested that, to the extent that rule-following is a well-developed generalised response class, it may be insensitive to the consequences experienced when a specific rule is followed. For example, an obedient child asked to "eat his vegetables" may do so despite the disagreeable taste sensations (which Zettle & Hayes called the *collateral consequences*) produced by complying with this particular request.

Recall that experiments by early cognitive researchers (e.g., Brewer, 1974; Dulany, 1968, see section 1.1.3) had shown that humans responded differently from animals in

simple learning tasks. These data led them to argue against the generalisability of operant principles to human subjects. From a Skinnerian perspective, however, the data were not out of line with the principles of RGB. Ironically, radical behaviourists would have cited Skinner in agreeing that human behaviour differed from animal behaviour, adding that the differences resulted from the human capacity for VB. In fact, many behavioural experiments based on this interpretation have provided further evidence that using rules to guide action insulates those actions from direct contingencies (see section 2.1.1; Catania, Shimoff, & Matthews, 1989; Kaufman, Baron, & Kopp, 1966; Matthews, Shimoff, Catania, & Sagvolden, 1977; Shimoff, Matthews, & Catania, 1986). Thus, for example, Harzem, Lowe, and Bagshaw (1978) showed that, regardless of whether explicit instructions about how to perform simple laboratory operant button-pressing tasks were provided, participants generated *self-rules.* These findings were summarised in the "Language Hypothesis" (Hayes, Barnes-Holmes, & Roche, 2001). In essence, inaccurate self-rules produce rigid behaviour patterns that do not naturally change through contact with the reinforcement contingencies arranged by an experimenter (see section 2.1.1). This experimental evidence that verbal control overrides sensitivity to behavioural consequences bears notable similarity to the clinical observations of cognitive therapists. That is, patients responded to their perceptions and expectancies rather than direct experience.

Although Skinner's accounts of VB and RGB have been both criticised and developed by subsequent generations of behavioural researchers (see chapter 2), three key aspects of his theorising remain important. Firstly, according to Skinner, cognitions (e.g., attitudes, expectations, beliefs, thoughts, plans and so on) are best seen as verbal codifications of past experiences that approximate the actual contingencies experienced, at best, only crudely. Secondly, Skinner argued that VB should not be seen as the cause of action; rather, as behaviour that was itself in need of explanation. From this perspective, CBT's argument that behaviour problems resulted from faulty cognitions was simply incomplete as an analysis of a causal chain. The third enduring feature of Skinner's work is the possibility that RGB can provide a framework for understanding the development and maintenance of psychological disorders. Alluding to issues in relation to CBT theorising, Zettle and Hayes (1982) proposed that cognitive distortions, depressive thinking styles, and faulty belief systems could alternatively be understood as disorders in rule formulation and rule following.

Hayes and his colleagues' have spent many years developing Skinner's early work into the understanding and treatment of clinical disorders. These developments are the focus of chapter 2. For now, however, it is important to consider whether new theoretical models and treatment approaches were (and are) necessary, given CBT's many clinical successes. With this question in mind, the following section describes two main criticisms of CBT. The first concerns the observation that the efficacy of CBT is limited when applied to patients with complex and entrenched disorders. The second concerns the observation that, although research into CBT's mechanisms of change is fairly sparse (relative to the number of outcome trials), results are often not in keeping with predictions of the cognitive model.

1.2.3 *Limitations of Cognitive Interventions*

Despite many successes, the last 30 years of research have begun to define some of the limitations of CBT. It is interesting to note that "some of the most pointed criticisms against it have emerged from within the CBT community" (Gaudiano, 2008, p. 6). For example, the two limitations that are considered below were raised by Clarke and Reinecke (2003) as two of several "unresolved issues" (p. 519) that will determine the integrity and range of applicability of CBT.

1.2.3.1 *CBT for Complex Cases.* The first limitation is that, as with behaviour therapy, CBT is not effective for all adult mental health patients. Approximately 30-50% of patients, often referred to as *treatment resistant* (e.g., Amsterdam, Hornig, & Nierenberg, 2001; Kenny & Williams, 2007), fail to improve, or relapse, following exposure to CBT techniques (e.g., DeRubeis et al., 2005; Westbrook & Kirk, 2004; Wilson, Fairburn, & Agras, 1997). This literature has identified several prognostic factors for poor outcomes. The most prominent of these include: (1) personality disorder symptomatology (PD; described below), (2) high levels of initial symptom severity, and (3) co-morbid mental health problems (the simultaneous co-occurrence of two or more clinical disorders). Patients with these characteristics are highly representative of patients seen in clinical practice (Persons & Silberschatz, 1998). However, because this type of patient tends to have been excluded from RCTs in favour of high internal validity (see section 3.1.1), this problem has often been concealed. For example,

Westen, Novotny, and Thompson-Brenner (2004) reviewed published RCTs on CBT and reported that between 40% and 70% of standard care patients are rejected from these clinical trials. This most often occurred because the patient presented with co-morbid disorders and/or met diagnostic criteria for a PD (see also Zarin, Young, & West, 2005).

Several studies have indicated that meeting diagnostic criteria for a PD is associated with poor treatment outcomes following CBT. PDs are characterised by long-standing (at least 5 years and originating in adolescence), deeply ingrained patterns of social behaviour that are detrimental to those who display them and/or to others (Diagnostic and Statistical Manual of Mental Health Disorders, Fourth ed., Text Revision (DSM-IV-TR), 2000). Turkat and Maisto (1985, cited in Bateman & Fonagy, 2000) conducted one of the first trials on CBT for a group of patients meeting PD diagnosis (N = 35). Of the 16 cases for whom outcome data were available (mainly due to high attrition), they found that only 4 patients showed improvements. More recently, Tyrer et al. (1988) have completed a 12-year follow-up RCT (N = 210) to compare CBT versus a self-help treatment programme versus pharmacology. At 2-year follow-up, they reported a differential effect of PD status on treatment outcome; participants meeting criteria for a PD showed less improvement following CBT and self-help treatment as compared to medication (Tyrer, Seivewright, Ferguson, Murphy, & Johnson, 1993). Similar findings were also reported at 5- and 12-year follow-up, indicating that initial PD status and baseline symptom severity were both predictive of poor treatment outcomes (Tyrer, Sievewright, & Johnson, 2004; see also Burns & Nolen-Hoeksema, 1992; Greenberg, Craighead, Evans, & Craighead, 1995; Shea et al., 1990).

Although poor prognosis is common to PDs in general, it is well recognised that Borderline Personality Disorder (BPD) is particularly treatment resistant (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). BPD is characterised by pervasive and recurrent patterns of affect instability, poor impulse control, and turbulent interpersonal relationships. Research suggests that the successful treatment of BPD is a challenge for traditional forms of CBT[5]. For example, a large scale project assessing CBT+ Treatment as usual (TAU) versus TAU alone arguably failed to demonstrate any persuasive

[5] Dialectical Behavioural Therapy (DBT; Linehan, 1993), a CBT-derived treatment for BPD patients, is not discussed here. This is because its techniques share common principles with less traditional "Third Wave Behavioural Interventions". It is therefore considered with these treatments later in the chapter.

evidence for the additional value of CBT techniques (Davidson et al., 2006). Despite the CBT condition receiving greater contact hours and exposure to a structured treatment, outcomes were largely comparable across conditions. CBT obtained greater reductions in suicidal acts than TAU, but the reverse trend was found for accident and emergency admissions. Although CBT+TAU further obtained significantly better outcomes than TAU for state anxiety at follow-up testing, no differences were found for a range of other psychiatric outcomes (depression, trait anxiety, other psychiatric symptom indexes, interpersonal functioning, or on quality of life). It is worth noting that although 27 CBT sessions were offered, patients only attended an average of 16. The treatment received was thus fairly brief. Giesen-Bloo et al. (2006) have evaluated the use of schema-focused CBT for BPD patients, reporting that this intervention was significantly more beneficial than a psychodynamic approach. Nonetheless, approximately 40% of the group still failed to show meaningful change despite the use of a particularly lengthy treatment protocol (two individual treatment sessions per week for three years).

Meeting diagnostic criteria for a PD is not the only factor associated with poor outcomes. Many trials also show reduced efficacy of CBT when applied to the treatment of patients with more chronic clinical disorders (defined by the DSM-IV as "Axis I" disorders, and including mood disorders such as depression and anxiety). For example, two comprehensive RCTs (Elkin et al., 1989; Elkin et al., 1995) found that 16-weeks of CBT for major depression was less effective than antidepressant medication and no more effective than no treatment (i.e., wait-list control, WLC). In a replication and extension trial, Dimidjian et al. (2006) further reported that among severely depressed patients, CBT was less effective than both behavioural activation (discussed in section 2.3.3) and antidepressants. Although there are a few anomalies to this trend (e.g., Westbrook & Kirk, 2005), many outcome trials concur with the findings of these authors (e.g., Rude & Rehm, 1991; Sotsky et al., 1991; Thase et al., 1992).

Perhaps not surprisingly, the impact of co-morbidity (or 'dual diagnosis') on CBT efficacy tells a similar story. Although research is limited (in part because these groups tend to have been excluded from RCTs), patients who have two or more co-morbid clinical disorders use the greatest proportion of mental health services (Kessler et al., 1994) and are less likely to improve following CBT (e.g., Brown, Antony, & Barlow, 1995; Conrod & Stewart, 2005; Thase, Simons, & Reynolds, 1993). Resistance to treatment appears to be especially prominent when the patient presents with a co-morbid

PD diagnosis and/or co-morbid maladaptive behaviours (e.g., substance abuse, dysfunctional eating, deliberate self-harm). Although PD symptoms are particularly high in populations who engage in such behaviours (Grant, Stinson, Dason, Chou, Ruan, et al., 2004), thus making it difficult to understand the relative contribution of the behaviours per se, research suggests that when clinical disorders are co-morbid with maladaptive behaviours, patients are less likely to stay in treatment and less likely to improve (e.g., see Conrod & Stewart, 2005; MacEwan & Remick, 1988; Randall, Thomas, & Thevos, 2001).

1.2.3.2 *Mechanisms of Change.* The second concern regarding CBT is that, more often than not, treatment gains cannot be explained in accordance with CBT theorising (e.g., see Dimidjian & Dobson, 2003; Holt & Lee, 1989; King 1998; Longmore & Worrel, 2007). Two strains of research have informed this observation. The first has used regression analysis to test whether CBT affects a change in outcome through changing the way patients think (i.e., cognitive change). The largest of these trials was a meta-analysis conducted by Oei and Free (1995), who reviewed outcome and process studies published between 1977 and 1987. Oei and Free examined what they claimed to be three necessary criteria for causality: (a) cognitive change occurs during therapy, (b) is associated with outcome, and (c) is specific to CBT. (These criteria are in fact not sufficient, however, because one must also show that cognitive change precedes symptom change). They found that although cognitive change did occur (criterion a), and often related to outcome (criterion b), it was not CBT specific (criterion c), occurring in both active *and* inactive (i.e., control) conditions. Although a few trials (five) have provided data in keeping with the CBT model (see Hofmann & Asmundson, 2008); most of this literature has been unable to support its predictions (e.g., Burns & Spangler, 2001; Dimidjian & Dobson, 2003; Imber et al., 1990; Morgenstern & Longabaugh, 2000).

The second stream of research has used dismantling studies to test whether cognitive techniques have a unique effect on treatment gains. One of the most influential of these studies was conducted by Jacobson et al. (1996) investigating the treatment of major depression. Jacobson et al. randomised 150 patients to either (a) full CBT (behavioural activation (BA), identifying and modifying automatic thoughts and core schema), (b) BA and automatic thought modification only (i.e., no work on core schema), or (c) BA

only. Interventions were matched for therapist contact and competence. Jacobson et al. found no significant between-group differences following treatment or at 2-year follow-up (Gortner, Gollan, Dobson, & Jacobson, 1998), thus suggesting that cognitive techniques had no added benefit above and beyond behavioural techniques. This challenged not only whether cognitive techniques were causal in symptom alleviation, but also implicated behavioural components as necessary and sufficient (see also Dimidjian et al., 2006; Wilson, Goldin, & Charbonneau, 1983).

The implications of Jacobson et al.'s (1996) research have been reported by others (e.g., Berman, Miller, & Massman, 1985; Miller & Berman, 1983; Shapiro & Shapiro, 1982). Additionally, several large meta-analyses have shown no difference in efficacy between behavioural and cognitive techniques (e.g., Ekers, Richards, & Gibody, 2007; Gloaguen, Cottraux, Cucherat, & Blackburn, 1998). Finally, some CBT literature has also shown that clinically meaningful reductions in depression can occur prior to the delivery of cognitive techniques ('sudden gains'; e.g., Ilardi & Craighead, 1994). These findings thus further suggest that cognitive techniques are not the main agent of change.

One possible explanation of the effectiveness of CBT, which relates quite directly to new intervention trends (described below), is offered by Teasdale and colleagues (e.g., Segal, Teasdale, & Williams, 2002). These authors suggest that CBT obtains its effects by changing the patients' relationship with their thoughts, rather than changing the thoughts directly. This process, referred to as *decentring*, describes the capacity to observe cognitions as transient events of the mind that are not permanent or characteristic of the self (Watkins, Teasdale, & Williams, 2000). It has been speculated that decentring may be indirectly achieved in CBT through the process of monitoring and disputing core cognitions. For example, Teasdale et al. (2002) reported that cultivating a decentred perspective fully mediated treatment gains in a CBT trial. This finding tentatively suggests that CBT may benefit patients by indirectly altering the way they respond to their own private VB.

To summarise: cognitive techniques were developed to addresses limitations of early behavioural treatments. The main way in which this was achieved was by making the patients' cognitions (i.e., their thoughts and feelings) the target of intervention. Compared to early behaviour therapies, cognitive treatments were a breath of fresh air to clinical practice and obtained noticeable successes for a range of clinical disorders. Nevertheless, CBT is not effective for everyone. Furthermore, of those studies

investigating mechanisms of change, only a minority have found evidence in support of the cognitive model. This presents a conundrum that is yet to be fully resolved: CBT can be effective, but the way in which it achieves its effects is often unclear. The limited success of CBT for some patients, and unexplained issues regarding mechanisms of change, provided fertile ground for the development of a new set of therapies.

1.3 Third Wave Behaviour Therapies: A Hybrid of Eastern Traditions and Radical Behaviourism?

Although clinically orientated experimental psychology has begun to move in the direction of cognitive models that are based on the standard computer analogy (e.g., Bradley, Mogg, White, Groom, & Bono, 1999), often supplanted with neuroscientific insights (see Hofmann & Aunderson, 2008), it is not yet clear how these advances will drive new treatment. What is becoming increasingly apparent, however, is that as scientific advances are made, some of the central tenets of CBT have required notable modifications (Gaudiano, 2008). In the meantime, Eastern modes of thought have contributed to the gradual emergence of a "third wave of behaviour therapies" (Hayes, 2004). These include treatments such as Mindfulness-Based Cognitive Therapy (MBCT; Segal et al., 2002), Dialectical Behaviour Therapy[6] (DBT; Linehan, 1993); and Acceptance and Commitment Therapy (ACT; Hayes, Wilson, & Stroshal, 1999). These interventions have several distinguishing features, the most important of which is that they are less concerned with *changing* the patient's private VB and more with teaching them to *accept* their self-talk without being ruled by it. Stated differently, they aim to reduce the extent to which patients use VB to regulate action, by helping them to adopt the perspective of a non-judgemental (or decentred) observer, more in contact with raw (or direct) experience (see chapter 2).

Mindfulness meditation is a key feature of third wave treatments, which appears to be one of the processes through which the treatments have their effects (Baer, 2003; Kabat-Zinn, 2005). Mindfulness has been a religious practice for many years, but only

[6] Referring to DBT and MBCT as "third wave interventions" is frequently debated because it implies a categorical distinction between them and more traditional cognitive techniques. This has been resisted by some cognitively minded theorists (e.g., Hofmann & Asmundson, 2008). The categorisation is used in this thesis because it usefully clusters interventions by virtue of functionally similar treatment process.

recently have these techniques taken a role in psychotherapy. Many definitions of mindfulness have been proposed, with most identifying two key elements: (a) purposefully directing attention to whatever is present, and (b) doing this non-judgementally, as opposed to relying on habitual judgement (see Bishop, 2002). Data already suggest that mindfulness-based techniques are clinically efficacious (e.g., Kenny & Teasdale, 2007; Linehan et al. 2006; Ma & Teasdale, 2004; Teasdale, Segal, Ridgeway, & Soulby, 2000). For example, two RCTs suggest that MBCT is an effective treatment for patients with recurrent depression who are currently in remission (Ma & Teasdale, 2004; Teasdale et al., 2000). For example, no MBCT patients, but 64% WLC patients, had relapsed 1-year after treatment (Ma & Teasdale).

Developments in clinical theorising and clinical practice have not gone unnoticed by radical behaviourists. Indeed two third wave interventions, DBT (Linehan, 1993) and ACT (Hayes et al., 1999), have evolved, to some extent, as hybrids of Skinnerian principles and Eastern traditions. DBT and ACT are particularly interesting because data suggests they may be effective for patients who have typically proven more resistant to first and second wave treatments (e.g., Hayes, Wilson, et al., 2004; Kenny & Williams, 2006; Linehan et al., 1999, 2006). Both interventions, albeit to varying degrees, aim to disrupt what are seen as the tyrannical effects of RGB by employing mindfulness and acceptance-based strategies. DBT is an intensive treatment intervention that uses contingency management to contain maladaptive behaviours emitted by BPD patients. DBT employs mindfulness to balance tensions between the need for change and self-acceptance (see Linehan, 1993).

ACT, on the other hand, is a more generic treatment, intended for application across many diverse mental health disorders. ACT is based on the idea that many topographically distinct behaviour problems can be understood as forms of excessive verbal control over behaviour and subsequent insensitivity to the consequences of action (see chapter 2; Hayes et al., 1999). Research suggests that, when applied to homogeneous samples (i.e., patients presenting with the same symptoms), ACT can be effective for several acute disorders (see Hayes, Luoma, Bond, Masuda, & Lillis, 2006). Furthermore, research tentatively suggests that it may have utility for more hard-to-treat cases (e.g., Gratz & Gunderson, 2006; Hayes, Wilson, et al., 2004). ACT forms the basis of empirical work in this thesis and for that reason, ACT and its theoretical foundations are the subject of chapter 2.

1.4 Chapter Summary

This chapter aimed to provide, from a radical behavioural perspective, a review of the historical development of behavioural therapies. To summarise, the main points will be reiterated. Firstly, despite best efforts, past correspondence between theory and therapy appears often to have been more analogous than direct. This criticism is valid for both behavioural and cognitive approaches. Secondly, although treatments continue to increase their range of applicability, a noticeable proportion of patients, often termed treatment resistant, fail to improve, or relapse, following CBT. These patients are symptomatically heterogeneous and typically present with more chronic, co-occurring and/or PD symptoms. Thirdly, although cognitive techniques can obtain noticeable effects for many patients, the mechanisms through which these effects are achieved remains to be fully elucidated. Finally, an emerging cluster of new treatments may begin to address some of these limitations and tentatively hold promise for more hard-to-treat groups.

CHAPTER II:
Theoretical Underpinnings and Clinical Application of Acceptance and Commitment Therapy

Acceptance and Commitment Therapy (ACT) is a relatively modern psychological treatment designed to undermine the tyrannical effects that may arise from excessive verbal control over behaviour. As later sections of this review will show, some promising clinical effects have been found. Before reviewing this literature, however, this chapter will first describe the key principles upon which ACT is based. To this end, Hayes and colleagues' extensions of Skinner's early work will be described. First, Zettle and Hayes' (1982) re-conceptualisation of rule-governed behaviour into a broader analysis of behaviour controlled by verbal antecedents will be presented. A detailed analysis of the functions of verbal stimuli necessitated by this reformulation will then be discussed (Hayes & Hayes, 1989). Next, a brief outline of Relational Frame Theory (RFT; Hayes et al., 2001), the product of these two earlier extensions of Skinnerian thought, will be presented. Following this, the role of verbal control in the development and maintenance of adult psychological disorders will be discussed. Finally, the principles underlying ACT and the literature on its therapeutic outcomes will be reviewed. This review will show that, although there is empirical support for the claim that ACT has obtained promising effects (Hayes et al., 2006), research is still in its infancy. Several critiques have questioned the extent to which ACT is a genuine departure from earlier cognitive approaches (e.g., Hofmann & Asmundson, 2008), disputed its theoretical coherence (e.g., Palmer, 2004), and raised questions regarding its true clinical impact (Ost, 2008).

2.1. Behaviour Governed by Verbal Antecedent Stimuli

Chapter 1 introduced Skinner's (1969) suggestion that an important subset of behaviour is controlled by verbal contingency specifying stimuli that, functioning as discriminative stimuli (S^D), occasion certain actions. Skinner called such actions instances of *rule-governed behaviour* (RGB), which he differentiated from *contingency-shaped behaviour* (CSB); that is, behaviour under the direct control of reinforcement contingencies. In 1982, Zettle and Hayes suggested that RGB may provide a useful framework for understanding clinical disorders. In re-examining RGB, however, they

identified a number of problems with Skinner's account and thus subsequently developed a more comprehensive theory of their own. Their account suggested that RGB could be distinguished from more general instances of discriminative responding because such behaviour was concurrently "in contact with two sets of contingencies" (Zettle & Hayes, 1982, p. 78). According to this conceptualisation, one set of contingencies related to the direct consequences of the particular instance of rule-following behaviour that was occasioned by the verbal antecedent. The second set related to the generalised act of following rules.

This seemingly complex account can be clarified with a simple example. Imagine a mother tells her daughter to put her shoes on. One set of contingencies would be those related directly to the putting on of the shoes, and is independent of any verbal rule (e.g., wearing shoes keep one's feet dry and protected). The second set of contingencies would be concerned with the daughter's act of following the rules that her mother supplies (e.g., praise or the removal of threat). Thus, the tendency to follow rules was conceptualised as a generalised operant, established and maintained through its consequences. In other words, the consequences of following rules in general (e.g., parental praise) increase (or decrease) the probability of future rule-following, even though the prescribed actions will vary topographically (e.g., wearing shoes, eating vegetables, or praying at bedtime).

2.1.1 *Rule-governed Behaviour (RGB) and Contingency Sensitivity*

Zettle and Hayes (1982) further suggested that the two sets of contingencies involved in RGB can be in competition as, for example, when a boy is told to come in and do his homework on a sunny evening. In such circumstances, the strength of one set of contingencies will determine sensitivity to the other. More specifically, and consistent with the *contingency insensitivity* literature (described below; see also section 1.2.2), if an action is predominantly governed by the contingency that includes the verbal antecedent, it will subsequently be less governed by the consequences of carrying out a particular prescribed act (i.e., its *collateral consequences*). Milgram's (1963) seminal paper on compliance is helpful in clarifying these ideas. In Milgram's study, participants (acting as "experimenters") were requested to press a button to punish an unseen "subject" positioned in another room (in fact, Milgram's confederate), every

time he appeared to make an error in a memory task. As far as participants were aware, their actions delivered electric shocks to the subject, and this was made salient by participants' hearing sounds of apparent anguish. The act of depressing the button thus participated in two sets of contingencies. One contingency had the (collateral) punishing consequence of hurting a fellow human being. The second included a verbal antecedent (the request) and produced the consequence of social pressure cessation following button pressing. Probably because a history of negative reinforcement for compliance with an authoritative person's instructions is so common, Milgram found that—despite being distressed by their actions—most participants continued to press the button. According to the dual contingency account, this happened because, for historical reasons, their behaviour was predominantly controlled by the contingency that included the verbal antecedent. They were thus insensitive to the direct consequences that would otherwise have exerted regulatory control over behaviour.

Zettle and Hayes (1982) further defined three functional units of rule-following. Each described a different type of contingency for generalised rule-following. Milgram's (1963) study illustrates *pliance*. Pliance is a form of RGB that is under the functional control of a history of speaker-mediated consequences for following the rule (the rule is known as a *ply*). Alternatively, behaviour may be governed by the "apparent correspondence between the rule and the way that the world is arranged" (Zettle & Hayes, 1982, p. 81). If so, the rule would be a *track* and following it would be called *tracking*. Tracking develops based on a history of following rules that result in reinforcing collateral consequences that are *not* mediated by the rule giver. For example, a girl may follow her father's instruction to "speak clearly" because, in the past, following her father's rules resulted in reinforcing consequences that were independent of his behaviour (e.g., "smell this flower", "taste that berry"). Finally, an *augmental* verbally modifies the effectiveness of non-verbal reinforcers. For example, a mother may say, "Gentle children are happy children" to increase the likelihood of her son's playing more gently with a younger sibling. To the extent that the reinforcing power of rough play is reduced, the boy's behaviour has come under the control of the augmental.

Rule-following can be highly adaptive. This is especially the case when natural consequences are weak or delayed (e.g., telling a child to study hard) or when rule-following prevents undesirable behaviour (e.g., warning against drug abuse). Nevertheless, a number of studies have shown that rule-following can also lead to non-

adaptive outcomes (e.g., Hayes, Brownstein, Zettle, Rosenfarb & Korn,1986; Horne & Lowe, 1993; Joyce & Chase, 1990; Kaufman et al., 1966; Kudadjie-Gyamfi & Rachlin, 2002; Lowe, Beasty, & Bentall, 1983). An early demonstration of this effect was reported by Kaufman et al. (1966), who exposed participants to a variable-interval (VI) schedule with monetary reinforcement and varied the accuracy of a rule given to explain the contingency. One of the three groups was accurately told how to maximise reinforcement on a VI schedule. The remaining two groups were given inaccurate instructions that would have been relevant to a variable-ratio (VR) or fixed-interval (FI) schedule. Participants in the latter groups persevered with ineffective responding based on the false schedule information for the duration of the 3-hour experiment, their behaviour seemingly unaffected by the actual contingencies.

More recently, research by Joyce and Chase (1990) has helped to elucidate why such effects may occur. In this study, participants were randomised to one of four groups. Group A and B received full instructions ("press the button 40 times for each point") and groups C and D received none. Furthermore, group B and D were pre-exposed to the fixed-ratio 40 (FR40) schedule until their responding met a stability criterion. Thus, before testing the effect of instruction on behaviour, stable responding was established in one instructed (B) and one uninstructed (D) group. All participants then took part in an operant task that had four unsignalled changes between an FR40 and FI10 schedule. Unlike FR schedules, the FI10 schedule did not require the high rates of responding to maximise reinforcement. Sensitivity to change was measured by calculating behavioural efficiency during the last 5 minutes of the FI10 (i.e., dividing number of responses by the total number of reinforcers available). Behaviour variability was measured by calculating the distribution of inter-response times at the end of the FR40 schedule (i.e., response variability as schedules switched). Consistent with previous research, Joyce and Chase found that instructed and trained-to-criterion groups (i.e., Group A, B and D) produced response patterns that were rigid (low in temporal variability) and insensitive to the changing contingencies. These patterns persisted despite low levels of reinforcement. In contrast, group C showed behavioural sensitivity because they engaged in more variable responding prior to the switch. This presumably occurred because—unlike group D—they had not had the opportunity to formulate self-instructions (i.e., rules) to direct their own behaviour. Put simply, when behaviour was

controlled neither by external instructions nor self-instructions, individuals could respond in a way that was flexible and sensitive to the changing environment.

Zettle and Hayes (1982) noted that characteristic features of RGB—rigid, ineffective but nonetheless persistent behaviour that is seemingly unaffected by its consequences—bore a striking similarity to patterns of behaviour seen in clinical practice. This led them to suggest that clinical disorders may be understood as the excessive or improper use of VB to regulate action. This idea played a key role in the development and practice of ACT, discussed in greater detail in section 2.2.

The Zettle and Hayes (1982) account of RGB took for granted that the language used to formulate rules was readily interpretable by listeners. Hayes and Hayes (1989), however, regarded this assumption as problematic. Skinner had not fully addressed the issue, simply asserting that a rule functioned as an S^D. This account is clearly inadequate because a S^D requires a history of differential reinforcement before it can exert stimulus control functions, whereas most humans are able to understand (and follow) novel rules heard for the first time (e.g., "If the banana ripens, open the umbrella"). Hayes and Hayes proposed that a behavioural explanation of how novel forms of verbal stimuli are interpretable by a listener was required. This is essentially a theory about the relationship between words (or other symbols) and things (or other environmental events). Two such theories have been developed. The first, Stimulus Equivalence Theory (SET), was developed by Sidman & Tailby (1982); the second, Relational Frame Theory (RFT), was proposed by Hayes & Hayes (see also Hayes et al., 2001). These theories will be discussed in turn.

2.1.2 *Stimulus Equivalence*

In everyday terms, Stimulus Equivalence (SE) describes the fact that, through learning, a variety of arbitrarily related objects can, in certain contexts, come to be treated as members of a single class of stimuli. For example, the varied faces of members of a sports team may, though learning, come to be regarded as equivalent for some purposes. Research on SE has been primarily based on the matching-to-sample (MTS) paradigm. In this paradigm, participants are trained to choose from a set of two or more comparison stimuli, the correct choice being determined by a sample stimulus. The discriminations trained are thus conditional (e.g., if A, choose X but if B, choose Y).

The key finding of the SE literature is that, after learning a limited series of conditional relations, novel untaught relations spontaneously emerge (Sidman & Tailby, 1982).

For example, Sidman and Tailby (1982) reinforced participants (a) for selecting arbitrary comparison stimulus B1 (and not B2 or B3) when shown sample stimulus A1, and (b) for selecting comparison stimulus C1 (not C2 or C3), again when shown A1 (see solid lines in Figure 2.1a). This training proved sufficient to give rise to a series of untrained relations (dotted lines in Figure 2.1a), each identified by a testing procedure in which no reinforcers were delivered. Thus, although participants had not been trained to do so, they were able spontaneously to select either comparison stimulus A1, given either B1 or C1 sample stimuli (*symmetry*) and to select B1 given C1, or vice versa (*transitivity* or *equivalence*). Furthermore, Sidman & Tailby also reported that the functional properties of all members of this equivalence class (see Figure 2.1a) may become modified if some change is made in the functionality of one class member (*the transfer of stimulus functions)*. For example, if A1 was paired with an electric shock after equivalence had emerged, aversive functions would transfer to B1 and C1. Symmetry, transitivity, equivalence, and transformation of function—the key phenomena of SE—would not be expected on the basis of simple conditioning (see Hayes et al., 2001, p. 16). Moreover, SE excited behavioural researchers because of its apparent direct relevance to language and, in particular, to naming.

To illustrate, suppose a child is taught to point to the written word S-P-I-D-E-R on hearing his mother say “spider” (Figure 2.1b). According to SE, having learnt this trained unidirectional relation, an untrained symmetrical relationship would emerge (given S-P-I-D-E-R, say “spider”). Now suppose that the mother utters “spider” and points to a real spider. Equivalence would be observed if the child spontaneously derived an untrained bidirectional relation between the actual spider and the word S-P-I-D-E-R. Finally, imagine that the child becomes fearful in the presence of an actual spider. According to Sidman & Tailby (1982), transfer of stimulus functions would be observed such that the child will subsequently show fear on seeing the written word S-P-I-D-E-R (e.g., a sign), or hearing the word “spider” spoken. According to Hayes et al. (2001), these fearful responses could be described as *verbally governed,* if the control of verbal (written or spoken) stimuli (and their derived function) predominates over the direct control of the current environment, in which no spider is present.

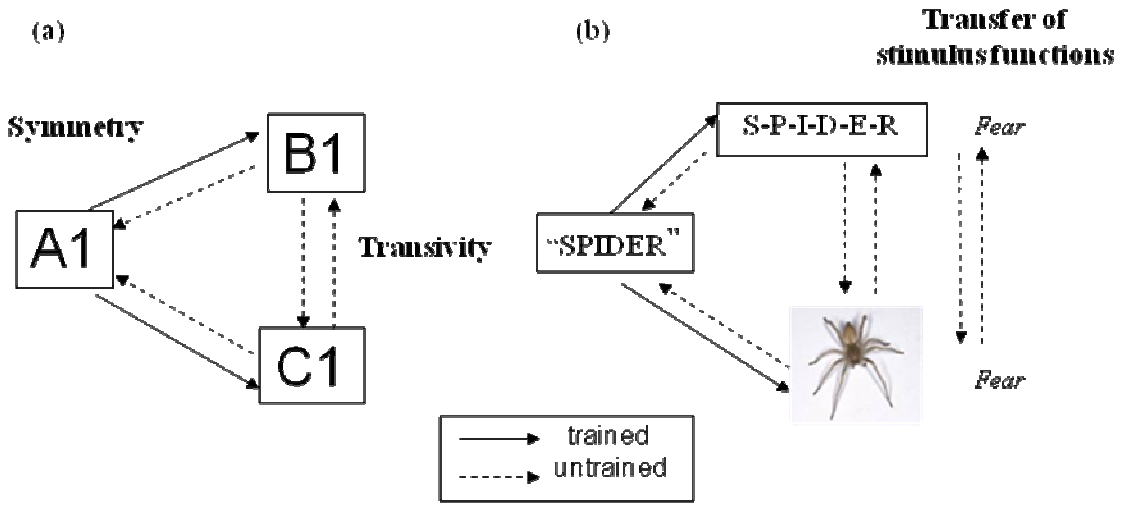

Figure 2.1 Two Visual Representations of Stimulus Equivalence based on Arbitrary Relations

Adapted from *"Acceptance and Commitment Therapy: An Experiential Approach to Behaviour Change"*, by S. Hayes, K. Stroshal, and K. Wilson, 1999, p. 37-38.

The key phenomena of SE have been extensively investigated, often using complex protocols designed simultaneously to create multiple equivalence classes each with more than three members (e.g., Hayes, Kohlenberg, & Hayes, 1991). Functions may then be conditioned to class members and transfer tested. For example, Dougher, Auguston, Markham, Greenway, and Wulfert (1994) taught participants (N = 8) two equivalence classes, each of which had four members (random shapes). Class 1 consisted of stimuli A1-D1 and class 2 of A2-D2. Phase 1 taught some critical relations and tested for the emergence of untrained relations that would indicate formation of these two equivalence classes (i.e., A1 = B1 = C1 = D1; A2 = B2 = C2 = D2). Following this, a respondent task was used to condition a member of class 1 to signal the onset of a shock (B1) and a member of class 2 to signal no shock (B2). After a resting period, all stimuli except B1 and B2 were presented during an operant task and change in skin conductance to stimulus presentation was observed. For six of the eight participants, skin conductance was elevated to class 1 but not class 2 stimuli, despite the fact that none of these stimuli had been directly conditioned. These findings suggested that aversive elicitation functions transferred to all members of an equivalence class, although most stimuli in the class have never been directly associated with a fear-eliciting event. Again, according to Hayes' theorising, behaviour predominantly under

the control of these derived functions, rather than direct environmental contingencies, would be described as verbally governed.

The transfer of stimulus functions within equivalence classes has been documented across a range of functions (e.g., skin conductance responses (Roche & Barnes, 1997), rate of responding (Barnes & Keenan, 1993) and happy and sad moods (Barnes-Holmes, Barnes-Holmes, Smeets, & Luciano, 2004)). Once established, these classes can persist for months without further training (Saunders, Wachter, & Spradlin, 1973) and can be resistant to unlearning (Wilson & Hayes, 1996). Furthermore, testing for the transfer of fear in spider phobic versus non-phobic counterparts, Smyth, Barnes-Holmes, and Forsyth (2006) found that greater levels of fear transferred in participants with pre-existing fear. This experiment thus captured the clinical observation that, once established, fear can quickly generalise to previously neutral stimuli.

What is the relevance of these findings to behaviour therapy? According to Hayes and his colleagues (2001), the transfer of stimulus functions is important because it may provide an analogue, albeit a rather elementary one, for how verbal stimuli acquire novel psychological functions. For example, the earlier 'spider' example provided a feasible account of how a verbal stimulus may elicit fearful responses even when the immediate context is not dangerous. In other words, the fear is a property of the verbal stimulus and not of the environmental context.

The observation that verbal stimuli can acquire the properties of other stimuli with which they have been associated only indirectly is also a cornerstone of Relational Frame Theory (RFT). Hayes et al. (2001) argue that the properties verbal stimuli can elicit are far more complex and dynamic than simple relations of equivalence may suggest. That is, the functions of verbal stimuli are not simply determined by equivalence relations. They may have different functions in different contexts and these may be determined by several other types of relations, such as comparison (Hayes et al., 2001, see below). At its simplest, RFT uses SET as a theoretical springboard, extending its core ideas in an attempt to capture some of the additional complexities of verbal control. The section that follows describes the elements of RFT used by Hayes to understand psychological disorders (for a book length account, see Hayes et al., 2001).

2.1.3 *Relational Frame Theory (RFT)*

The primary way in which RFT extends SE is by suggesting that equivalence (or "coordination" as it is termed in RFT) is just one of many possible dimensions along which stimuli can be related. According to Hayes et al. (2001), other stimulus relations may include, for example, relations of opposition, distinction, comparison, and hierarchy. Hayes further suggests that although the type of relation may vary (i.e., opposition, hierarchy, and so on) the fundamental, or overarching, process remains the same. This process is called *relational framing.* This term is used to convey the idea that one generic process may operate similarly, regardless of the particularities of any given relation (as a photo frame, which can fit many pictures).

Although the idea of a *relational frame* implies an object of some kind, the second main tenet of RFT is that relational framing should be understood behaviourally, as a generalised operant (perhaps better conveyed as "framing relationally"). That is, following a reinforced training history with sufficient exemplars, the process of framing events relationally is abstracted as a functional response class. Thus, like imitation, Hayes et al. (2001) suggest that any form of relational framing has a potentially infinite range of topographies.

This leads to the third main tenet of RFT, namely that regardless of the relational form (i.e., opposition, causation), a relational frame has three generic properties. As we shall see shortly, these properties correspond to the SE principles of symmetry, equivalence, and the transfer of stimulus functions. More general terms are required, however, to describe relations other than equivalence. The first property of events within a relational frame is *mutual entailment*. This means that if a relation exists between stimulus A and stimulus B in a given context, a relation between B and A will also be entailed in that context. For example, if Matthew is meaner than Sam, a relation between Sam and Matthew will be entailed (in this case that Sam is more generous than Matthew). Second, additional stimulus relations can be derived by combining two or more existing relations. For example, if A is related to B and B is related to C, then bidirectional relations between A and C will emerge (*combinatorial mutual entailment*).

So, if James is funnier than Mark, and Mark is funnier than Jon, a relation between James and Jon is derived (James is funnier)[7].

Finally, perhaps the most important aspect of this theorising is that stimulus functions are transformed based on relational properties (*transformation of stimulus functions*). For example, if stimulus A is the opposite of B (relational frame of opposition) and A is reinforcing, B will function as a punisher even if it has never been paired with an aversive event (e.g., if Steve and Jon are opposites; interactions with Steve are rewarding). In this example, functions have transferred via mutual entailment. They may also, however, transfer via relational networks; that is, through combinatorial mutual entailment. For example, continuing with the previous example, if A is opposite to B and C is less than B, it can be derived that C will function as a punisher, but a less potent one than B.

Before reviewing some of the evidence for these properties, the final aspect of RFT is that relations are *arbitrarily applied*. This simply means that the process of relating is not restricted to formal dimensions between two stimuli (e.g., bigger-than, smaller-than). Rather, stimuli can be framed according to non-formal or socially constructed features (e.g., economic value or physical attractiveness). For example, a non-human organism may be able to discriminate that a pound is formally smaller than a two pence piece, but only humans can discriminate that the pound has more economic value. According to RFT, this is an example of relational responding that has been brought under arbitrary contextual control. This is described as arbitrary because no formal features guide the discrimination.

Thus, the key tenets of RFT as described by Hayes et al. (2001) may be summarised as follows. Relational frames constitute a class of generalised operants thought to be established based on a history of multiple-exemplar training. From an early age, children are reinforced for engaging in derived relational responding which, according to Hayes et al. (2001), underpins language and drives cognition. Although an infinite number of relations can be drawn between stimuli, the defining features of relational framing are constant: mutual entailment, combinatorial mutual entailment and

[7] In some cases combinatorial mutual entailment does not allow a relationship to be derived but, for Hayes, this absence of a derivable relation is itself entailed. For example, if Ann is more beautiful than both Belle and Claire, it is mutually entailed that both Belle and Claire are less beautiful than Ann, but it is not possible to derive a relationship between Belle's and Claire's beauty.

transformation of stimulus functions. Relational framing can deal in arbitrary relationships, based on convention in a verbal community (goodness, worthiness, etc) rather than simply physical properties (height, temperature, etc.). Finally, Hayes et al. (2001) imply that, once established as a generalised operant, relational framing occurs spontaneously and without conscious intentional effort.

The procedures for testing RFT predictions stem from the SE research. First, MTS procedures are used to bring pre-learnt discriminative responding (e.g., more-than less-than) under arbitrary control. For example, participants may be reinforced for choosing smaller lines than a sample line in the presence of one nonsense letter triplet "DRT" (thus signalling pick smaller) and longer lines in the presence of another (e.g., "FTY", thus signalling pick larger). Second, these cues ("DRT" "FTY") are used to train a limited number of arbitrary relations between novel nonsense triplets (e.g., "SUO" "FIM"). These relations are 'arbitrary' because no formal characteristics can be used to guide discriminations. Third, the emergence of untaught relations is tested. Fourth, one member of the class is given functional properties through conditioning and the final stage then tests for patterns of transfer that would indicate the formation of the relational network.

A study by Dymond, Roche, Forsyth, Whelan, & Rhoden (2007) demonstrates these procedures. Phase 1 used MTS procedures to bring 'same' and 'opposite' discriminative responding under arbitrary control. Participants viewed sample and comparison stimuli that related to one another along a non-arbitrary dimension (e.g., circles differing in size). In the presence of the cue for opposite (e.g., "ABC") participants were reinforced for choosing the comparison stimulus that was opposite to the sample stimulus. Conversely, in the presence of the cue for same (e.g., "XYZ"), participants were reinforced for choosing the comparison stimulus that was the same as the sample stimulus. Phase 2 tested the participants' ability to apply these relational cues successfully to a novel stimulus set that differed along other non-arbitrary dimensions (e.g., colour). Phase 3 introduced a new stimulus set in which sample and comparison stimuli could not be reliably discriminated between based on non-arbitrary dimensions (arbitrary shapes). This phase used the relational cues for same and opposite to teach a limited set of critical relations that corresponded to a relational network that was pre-determined by the experimenter (see Figure 2.2). Phase 4 tested for the emergence of untrained relations that would indicate formation of the relational network.

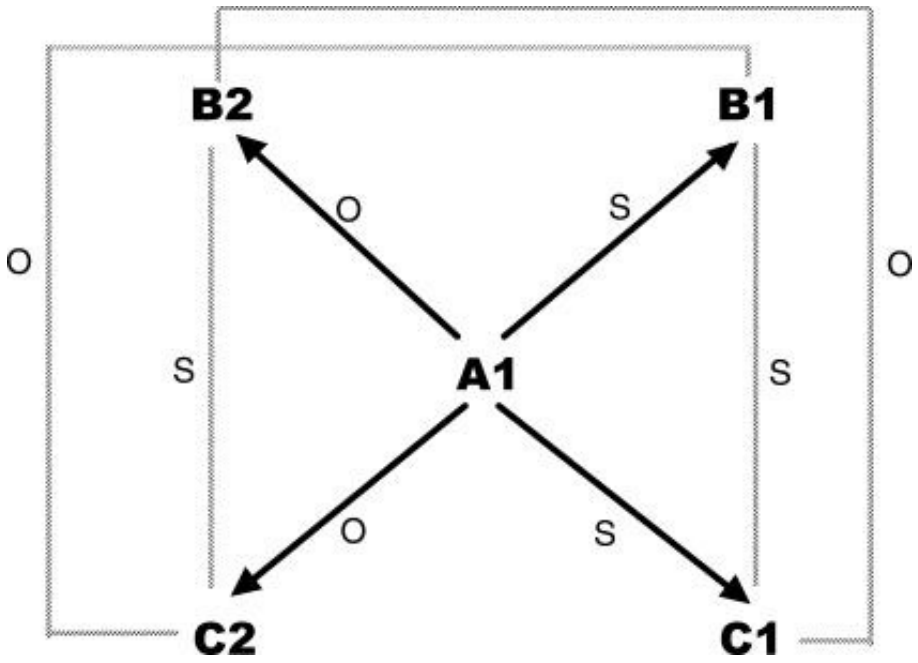

Figure 2.2 Pre-determined Relational Network of Same ('S') and Opposite ('O') Relation

From "Transformation of Avoidance Response Functions in Accordance with Same and Opposite Relational Frames," by S. Dymond, B. Roche, J. Forsyth, R. Whelan, & J. Rhoden. *Journal of Experimental Analysis of Behaviour, 88,* p. 253. Copyright 1995 by the American Psychological Association

Phase 5 involved avoidance conditioning. The presentation of stimulus B1 or B2 was quasi-randomised across the trial. Seventy five percent of B1 presentations were followed by an aversive image and an aversive sound and 75% of B2 presentations were followed by a non-aversive image (the remaining 25% were followed by a blank screen). Participants were trained to emit a simple avoidance response (press a computer space bar button) during the presentation of the B1 but not the B2 stimulus. This avoidance response replaced the stimulus with a blank screen, following which the next stimulus was presented. Conditioned avoidance was defined as: "the production of an avoidance response during each of the final 10 consecutive exposures to B1, and the absence of an avoidance response during all of the final 10 consecutive exposures to B2". In phase 6, trained and control (non-trained) participants were exposed to C1 and C2 stimuli and the transformation of avoidant conditioning was tested. That is, this trial measured the tendency for participants to avoid C1 and C2 stimuli (by pressing the space bar). Seven of the eight trained participants, but none of the control participants,

avoided C1 but not C2. This occurred despite the fact that neither C1 nor C2 had been directly conditioned. These findings, and those of others (e.g., Dymond & Barnes, 1994; Holmes, Holmes, Smeets, Strand, & Friman, 2004; Roche & Barnes, 1997), suggest that functions can transfer across arbitrarily related stimuli based on relations that are more complex than equivalence.

2.1.4. *Evaluation of SET and RFT*

Although the empirical data obtained though laboratory work on equivalence and relational framing is not in doubt, it is possible to question whether the research described is capable of supporting the conceptual load required of it. Recall that Hayes' aim was to create a theory that would explain how novel rules could be understood and acted upon by linguistically competent speakers. Thus, the experimental programme for RFT (which incorporated SET) was designed to elucidate the processes involved. Whether they have done so, however, is open to debate. For example, Horne and Lowe (1996) have argued convincingly that SE research does not shed light upon the processes underlying the acquisition of naming because, although equivalence emerges following conditional discrimination training, it does so only in participants who already possess naming skills. Much evidence suggests that it is difficult or impossible to demonstrate SE in animals, infants, or people with profound intellectual disabilities (e.g., Devany, Hayes, & Nelson, 1986; Dugdale & Lowe, 2000), and most attempts to suppress naming in equivalence training are unconvincing (Randell & Remington, 1999).

A similar argument can be raised with regard to laboratory demonstrations of relational framing. The experiments designed to illustrate relational responding do show the spontaneous emergence of complex derived relations, but only after prolonged training and only in linguistically competent participants. It is tempting to conclude that the derived relations obtained in such studies are the hard-won product of a laborious process involving private speech, namely verbal reasoning based on existing knowledge applied to the complex problems posed by RFT experiments (Palmer, 2004). In other words, like SE, successful demonstrations of relational responding could depend on the pre-existence of high levels of verbal skill. If so, the experiments on relational

responding cannot be considered to capture the processes underlying the acquisition of such skills.

To summarise: although RFT has been the subject of many laboratory investigations, primarily by Hayes and his colleagues, it has not escaped criticism. Critics have questioned both its fitness for purpose and its clarity (e.g., see Palmer, 2004). The debate is as yet unresolved. What is important in the present context, however, is the proposed link between relational framing, rule-governed behaviour, and psychological distress. Hayes et al. (1999) suggest that relational processes are fundamental to psychological problems. They have argued that many clinical disorders are characterised by the tendency to become excessively entangled with the functions of private verbal events (*cognitive fusion*), coupled with the motivation to attempt to escape from or avoid such unwanted private events (*experiential avoidance*). Because these processes only occur in verbally-able beings, Hayes et al. (2001) suggests that they distinguish human from animal behaviour. Thus, "a non-human shocked in a coloured box will be reminded of the pain by the coloured box, but not by self-reports of being there. If a non-human is trained to report whether or not it was shocked, it will do so without distress because such events followed the shock and thus do not contain the functions of the shock. For humans, it is different: reports of past events can themselves produce pain, because the two are mutually entailed (i.e., bi-directionally related)" (Hayes et al., 2001, p. 215).

2.1.5 *Cognitive Fusion and Experiential Avoidance*

Cognitive fusion is described as the phenomena that occurs when "stimulus functions... dominate over other sources of behaviour regulation ... making an individual less in contact with the here-and-now experience and direct contingencies" (Hayes, 2004, p.650). In more general terms, this has been described as a state of merging with the content of one's private experiences (thoughts, feelings, memories, sensations), and using those experiences as a predominant guide for action (Hayes et al., 1999). Fusion further conveys the quality of treating the content of one's private VB as literal and accurate reflections of reality, as opposed to verbal codifications of experience, having behavioural functions (e.g., "that's just a thought" or "that's just an expectation"). Because fusion is described as a natural consequence of language, Hayes et al. (1999)

suggest that it is not a characteristic of a certain person or thought, but a process that everyone is susceptible to. This process is described as problematic when an individual is *excessively* entangled with the content of private VB and when this supports narrow, rigid and ineffective behaviour patterns. This theorising can be contrasted to more traditional cognitive theorising, therefore, because the literal presence or absence of certain cognitions is not thought to be problematic per se.

Experiential avoidance describes the phenomenon that occurs when "a person is unwilling to remain in contact with particular private events and takes steps to alter their form or frequency" (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996, p. 1154). According to Hayes et al. (1999), because verbal stimuli can elicit the functions of other stimuli (such as their referents), aversive states cannot simply be avoided by avoiding certain contexts. This is because, in Hayes' (2004) view, aversive states can occur via relational processes in almost any situation. Hayes suggests that because of this, verbally-able individuals are not only motivated to avoid certain contexts, but certain private experiences also. Experiential avoidance thus describes all attempts to avoid, escape, control or alter unwanted private events even when doing so can be harmful (Hayes et al., 2006).

Experiential avoidance is similar to earlier ideas such as emotional and cognitive avoidance and suppression (e.g., Roth & Cohen, 1986). Research on these concepts suggests that avoiding private events may be a futile endeavour. For example, research on *thought suppression*—deliberate attempts not think about something—has shown that the avoidance of internal events is not only unsustainable, but it can actually increase the salience, intensity, frequency, and functional importance of that thought post-suppression (e.g., Wegner, 1994; Wegner & Gold, 1995; Dejonckheere, Braet, & Soetens, 2003). Hayes (2004) accounts for this observation by suggesting that learnt associations are extraordinarily resistant to unlearning. Furthermore, he suggests that because part of the avoidance rule will necessarily include the to-be-avoided-stimuli ("I must not think about X"), following it will ultimately cue the very functions that the rule was designed to avoid.

Unlike accounts of emotional and cognitive avoidance and suppression, which tend to consider avoidance of private events as a *contributory* factor in the development and maintenance of clinical disorders, Hayes et al. (1999) identify experiential avoidance as a far more central concept. In fact, Hayes et al. (1996) have gone as far as to suggest

that experiential avoidance may be usefully considered as a *functional diagnostic dimension* that underlies many formally dissimilar clinical disorders. From this perspective, clinical disorders (or at least many of them) are thought to constitute a functional response class whose shared property for negative reinforcement is the capacity to temporarily alleviate distress (see chapter 4). This theorising has included reference to, for example, OCD (Twohig, Hayes, & Masuda, 2006), trichotillomania (Norberg, Wetterneck, Woods, & Conelea, 2007), substance abuse (Forsyth, Parker, & Finlay, 2003; Hayes et al., 1996), DSH (Gratz, 2006; Najmi, Wegner, & Nock, 2007), panic disorder, borderline personality disorder (BPD), suicidality (Hayes et al. 1996), and disordered eating (Wilson & Roberts, 2002).

Research on experiential avoidance predominantly relies on the Acceptance and Action Questionnaire (AAQ; Hayes, Stroshal, et al., 2004), a self-report inventory that was designed specifically for its measurement. The AAQ is a broad measure, however, with items described as tapping fusion with, attempts to control and avoid, and the predominant use of private experiences to regulate action. This has led to some debate regarding the precise nature of the AAQ (see Hayes et al., 2006; see also section 8.3.3), but it is apparent that it relates to other variables in ways that are consistent with the theoretical construct of experiential avoidance. For example, cross-sectional research has found that the AAQ engages in moderate to large correlations with thought suppression, emotional escape and avoidance, and multiple indices of psychological distress (Hayes et al., 2006; Hayes, Stroshal, et al., 2004; Tull, Gratz, Salters, & Roemer, 2004).

Using the AAQ, several cross-sectional studies have also found evidence to suggest that experiential avoidance may play an important role in the development of clinical disorders. For example, the AAQ has been found to mediate the relationship between sexual abuse and psychological distress (Marx & Sloan, 2002), and between anxiety sensitivity and problem drinking (Stewart, Zvolensky, & Eifert, 2002). Although cross-sectional, these findings suggest that the risk factors of sexual abuse and anxiety sensitivity affect distress through heightened levels of experiential avoidance. This implication is consistent with research showing that thought suppression mediates the relationship between intense experiences of negative affect and BPD symptoms (Cheavens et al., 2005), even when controlling for childhood abuse (Rosenthal, Cheavens, Lejuez, & Lynch, 2005). Despite the clear rationale, research has not yet

tested whether experiential avoidance explains common variance (or covariance) across dissimilar clinical disorders. Similarly, few studies have tested for associations between the AAQ and the tendency to engage in maladaptive or risky behaviours (e.g., anorexia, substance abuse). Although thought suppression has been found to partially mediate the relationship between emotional intensity and DSH (Najmi et al., 2007), similar relations between the AAQ and DSH (Chapman et al., 2005) and substance abuse (Forsyth et al., 2003; Polusny, Rosenthal, Aban, & Follette, 2004) were not found.

Although these cross-sectional studies are unable to determine the direction of causation, their implications are consistent with longitudinal and experimental research. For example, using a longitudinal design, Plumb, Orsillo, and Luterek (2004) found that for students experiencing negative lifetime events, the AAQ was a stronger predictor of future distress than baseline distress. This suggests that experiential avoidance played a causal role in maintaining distress. Similarly, experimental designs comparing participants with high and low AAQ scores (i.e., 1SD ± group norm) have found that participants high in experiential avoidance exhibit greater emotional arousal during exposure to emotion induction procedures (e.g., Sloan, 2004), and respond more rapidly to prevent exposure to aversive pictures (Cochrane, Barnes-Holmes, Barnes-Holmes, Stewart, & Luciano, 2007). Finally, linking the AAQ to the contingency insensitivity effect (see section 2.1.1), F. Bond and colleagues (personal communication, 19th August, 2006) found that the AAQ was significantly, prospectively predictive of sensitivity to unsignalled contingency changes during an operant task. Although preliminary and unpublished, this finding is important because it supports the hypothesised link between experiential avoidance and contingency insensitivity.

To summarise: Hayes and his colleagues' account of verbally controlled behaviour has been reviewed, highlighting some of its strengths and limitations. This section has also described cognitive fusion and experiential avoidance, the two key vehicles that are used to link equivalence and relational theorising to clinical disorders. It is apparent that most conceptual extensions of relational laboratory studies to clinical phenomena are, as yet, more theoretical and analogous than empirical and direct. Nevertheless, research using the AAQ suggests that experiential avoidance may prove useful in understanding diverse clinical problems. Furthermore, it has become increasingly evident that ACT, whose core techniques are derived from this theorising, has produced some promising clinical effects. In the sections that follow, ACT will be described in terms of its aims,

therapeutic techniques, and stages of change. This is followed by a review of component analysis and outcome research. This review will show that, despite many theoretical and empirical gaps, the outcomes obtained when using ACT are impressive and worthy of continued investigation.

2.2. Acceptance and Commitment Therapy (ACT).

ACT is firmly grounded in the functional proposition that a diverse range of psychological disorders can be characterised by excessive verbal control over behaviour and subsequent insensitivity to direct contingencies. In Hayes' view, excessive verbal control leads to persistent behaviour patterns that do not naturally change with changing circumstances. Moreover, akin to many therapeutic interventions that predate it, ACT identifies experiential avoidance as a specific cause of psychological distress (Hayes, 2004). Behaviour in the service of long-term *values*—desired life qualities that have intrinsically fulfilling properties (see section 2.2.1)—become less frequent as behaviours maintained by the immediately reinforcing properties of experiential avoidance predominate in the repertoire (Hayes et al., 2006). As previously noted, in Hayes' view, the content of verbal behaviour (e.g., thoughts, feelings) is not assumed to be a problem in and of itself; rather, the tendency to take that content literally (*cognitive fusion*) and excessive attempts to escape or otherwise reduce its impact (*experiential avoidance*), is thought to be psychologically harmful.

ACT aims to disrupt verbal governance when that governance leads to behaviour problems, and to increase psychological flexibility (often used synonymously with behaviour flexibility). Psychological flexibility is the ability to adapt one's behaviour in a manner that is sensitive to the here-and-now experience and direct contingencies (Hayes et al., 2006). Furthermore, ACT aims to construct a new repertoire for behaviour that allows increasing access to positive reinforcement. This is achieved by identifying the patients' core values and shaping up behaviour patterns that are consistent with them. Disrupting excessive verbal regulation is challenging for many reasons; perhaps most specifically because therapists need to use VB in order to affect a change in verbal governance. Thus, ACT is more experiential that didactic, aiming to provide multiple contexts in which patients' experiences, rather than their verbal codification of experience, are made salient.

The following section describes how ACT aims to achieve these goals. Therapeutic techniques, stages of change, and the therapeutic stance are outlined. This is followed by a review of the empirical literature. The effects of each ACT technique are first evaluated, followed by a review of outcome research on ACT as an integrated treatment package.

2.2.1 *Therapeutic Techniques and Stages of Change*

ACT has been described as employing six classes of therapeutic techniques (Hayes et al., 2006) and as involving six stages of change (Hayes et al., 1999). Although many authors refer to techniques and stages interchangeably, their correspondence is not exact. Therapeutic stages tend to have a temporal order (see Hayes et al., 1999), whereas techniques can be used throughout therapy (although their use is more or less probable in certain stages). Figure 2.3 suggests one way in which their correspondence may be conceptualised (parts of which are hinted to in Hayes et al., 2006). This Figure suggests that the first four stages of treatment (*creative hopelessness, control as the problem, acceptance and defusion and defining the self*; Hayes et al., 1999) predominantly involve four classes of techniques; acceptance, defusion, mindfulness and self-as-context. These techniques are described as classes, because the procedures used within them are similar by virtue of function but not form. For example, metaphors, poems, experiential exercises and meditation could all be used in any one of the four classes of techniques. Furthermore, because the aims of these techniques overlap conceptually (discussed below), they are depicted as belonging more broadly to a higher order class of 'undermining verbal governance' techniques. The last two stages, values and committed action, are differentiated from earlier stages because they deal specifically with constructing a new repertoire of effective action. However, because this stage is thought to be dependent on obtaining successful skills for undermining verbal governance, it is rarely pursued in the absence of further work on defusion, acceptance, self-as-context, self-as-process, and mindfulness[8].

[8] Recent conceptualisations of ACT tend not to depict it as linearly as in the Hayes et al. (1999) treatment manual. For example, K. Wilson (personal communication, October, 2008) suggests a more flexible and pragmatic approach that is more principle, than protocol, driven. He suggests that when a patient is psychologically inflexible, the therapist should use techniques to undermine verbal governance. Conversely, when a patient is psychologically flexible, the therapist should focus on values and committed action.

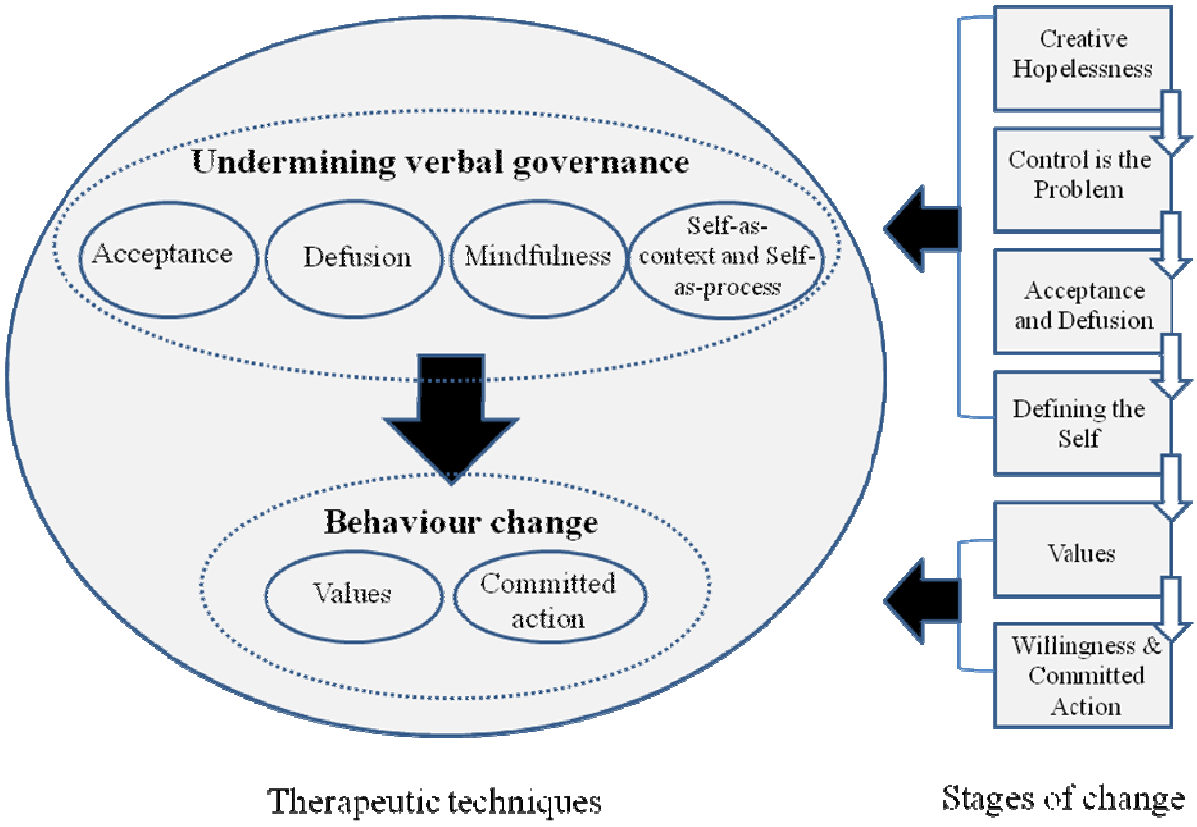

Figure 2.3 Visual Conceptualisation of the Correspondence between ACT's Therapeutic Techniques and Stages of Change

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According to Hayes et al. (1999), *undermining verbal governance* begins with creative hopelessness. ACT assumes that patients come to therapy with a surfeit of failed attempts to control, eliminate, or problem-solve their difficulties. This stage aims to explore the patient's key psychological difficulties and elicit the strategies that he/she have been using to manage and control those difficulties. This stage aims to disrupt this "control and eliminate agenda" (Hayes et al., 1999) by exposing verbally guided and logical problem-solving strategies as futile when applied to private events. The patient is invited to consider the long and short term effectiveness of control strategies, helping them to arrive at the conclusion that the act of controlling private experience may paradoxically serve to maintain them. (Exemplar techniques are described in Appendix E, which reports the ACT protocol that was used in studies 3 and 4 of this thesis).

This stage naturally extends into stage two, in which control is explicitly established as the problem. Experiential avoidance is introduced as a logical and natural consequence of language processes, but which can have substantial costs. These costs include the paradoxical amplification of unwanted experience and inhibited valued living. These ideas can be explored using thought controlling exercises and daily records to document the short and long term consequences of avoiding unwanted private experiences. Because this stage aims to undermine excessive experiential avoidance, it should end with the patient responsive to, perhaps even curious about, alternatives.

One way to disrupt the avoidance of private events is to tolerate exposure to them. Stage two thus naturally extends into stage three, in which *acceptance, defusion,* and *mindfulness* are proposed as alternatives. Hayes et al. (1999) describe acceptance as engaging with habitually avoided events non-judgementally and without attempting to change or escape them (Hayes et al., 1999). This differs from exposure in aiming to cultivate undefended contact with difficult internal events, rather than reducing the occurrence of them through habituation. *Mindfulness*/*being present*[9] also aims to cultivate non-judgemental awareness of internal and external events (Hayes et al., 2006). Mindfulness achieves this by increasing sensitivity to direct experience broadly, rather than targeting problematic forms of experiential avoidance specifically. For example, acceptance techniques may be used to help a socially anxious patient stay in contact with feelings of anxiety, rather than avoiding them. Mindfulness techniques, however, may be used to direct attention towards, and enhance awareness of, direct experience in day to day activities. Together, these techniques are thought to undermine experiential avoidance by creating a context in which the patient can begin to experience private VB for what it is (i.e., memories, thoughts, judgements); rather than being entangled with its functions (see Appendix E for treatment examples).

Acceptance is also functionally similar to *cognitive defusion.* Defusion techniques create contexts in which the patient interacts with unwanted private events in novel and less threatening ways. Defusion can thus be conceptualised as a special type of exposure to internal events. For example, the therapist could use humour to help the patient to

[9] Although Hayes uses the phrase "being present" this is, in terms of function, indistinguishable from mindfulness. Topographically, mindfulness describes a more formal and meditative approach to cultivating present moment living. Because more formal mindfulness practise was used in the current thesis, this phrase will be used throughout.

interact with private events in novel and more flexible ways, or draw on Gestalt techniques such as physicalising a psychological problem (e.g., describing it in terms of colour, size, and shape). Defusion thus differs formally from acceptance by intentionally manipulating the functions of private events. However, defusion does not simply aim to replace aversive functions with more tolerable ones. Instead, using multiple exemplar training, defusion aims to expose the process of transferring functions. That is, it aims to teach the patient that private verbal events have different functions in different contexts. The ultimate aim of defusion, therefore, is to undermine the perception that thoughts and feelings are literally true, by experiencing them as contextually determined, transient and changeable (Hayes et al., 1999). In this way, cognitive defusion is thought to create psychological distance between private VB and subsequent action.

The fourth stage of treatment is called *Defining the Self.* ACT theorising suggests that there are at least three possible senses of self. First, self-as-content describes the self as defined by the content of VB (e.g., "I am ... kind, old, anxious, tall, and so on). Smith and Hayes (2005) refer to this as an integrated verbal summary of oneself. Secondly, self-as-process describes an observing self that is routed in present moment experiences and that can observe this verbal activity (e.g., "Now I am having the thought that I am kind..."). Finally, self-as-context describes the self as a context within which this VB occurs, but that is not defined by it. This transcendental sense of self is thought to be stable and consistent over time and context. Hayes et al. (1999) suggest that individuals predominantly experience self-as-content. ACT thus aims to cultivate, or make more salient, the other two senses of self. Self-as-process is facilitated by all of the 'undermining verbal control' techniques. For example, *mindfulness* cultivates a sense of self that has sensitivity to and awareness of the present moment. An exemplar self-as-context technique, on the other hand, could guide patients to recall an event from their past and to identify consistent and continuous aspects of the self that witnessed this event and that is present now. The main aim of this stage of treatment, therefore, is to develop a sense of self that is consistent and continuous over time but flexible and adaptive to changing circumstances (see Appendix E for example techniques).

Once reliance on VB as a predominant guide for action has been disrupted, later stages of ACT aim to bring the patients' behaviour into line with their values. Values are described as desired life qualities that have intrinsically fulfilling properties. ACT draws a clear distinction between goals and values. Goals are described as verbally

specified, tangible actions (e.g., teaching my child to ride a bike). Values are also verbally specified, but they are molar and cannot be obtained in the literal sense (e.g., being a loving parent). Values are, however, verbally specified. ACT thus aims for the quality with which patients follow their values to be qualitatively different from RGB, exhibiting sensitivity to both sets of contingencies described by Zettle & Hayes (1982). For example, patients are taught to track values with sensitivity to the *present moment* and from the perspective of a non-judgemental *observing self* (e.g., self-as-process). Furthermore, they are taught to use *acceptance* and *de-fusion* skills to resist the pull towards verbally controlled behaviour patterns. The final stage, *committed action,* involves publicly committing (i.e., to the therapist and/or members of a therapeutic group) to verbally specified, value congruent goals and revisits earlier skills to overcome barriers to change. The main aim of these final two stages is to help the participant construct a new, value orientated repertoire for adaptive responding that provides access to natural reinforcers.

Finally, the *ACT therapeutic stance* has several distinguishing features. Patients typically seek therapy in order to establish new rules for behaviour regulation, and thus perceive the therapist as the rule giver. The ACT therapist thus aims to balance tensions between giving required information (e.g., socialisation to the ACT model) and fastening experiential learning using the techniques of change described above. The therapist may thus seek to establish themselves as someone who, despite knowing more in a content sense, is equally susceptible to fusion, experiential avoidance, and so on. They may also undermine mindless derivation of new rules by using statements such as "don't believe a word I am telling you, what does your experience tell you" and "I've been a clinician for years now and I still can't get it right!". Similarly, the therapist may resist explaining or justifying in-session experiences so as not to model verbally codifying or intellectualising experience. The ACT clinician may also use tangible examples from their own lives to model ACT processes or use think aloud procedures in situ (e.g., "My mind's says I'm a lousy therapist because I don't know the answer to your question").

2.2.2. *Evidence for Therapeutic Techniques*

No known research has investigated ACT in terms of stages of change or therapeutic stance, but there is growing evidence, typically using analogue methods (see section 3.3.1), to support the utility of each of ACT's techniques.

Analogue research on tolerance for physical discomfort and/or emotional distress supports the therapeutic utility of acceptance techniques. For example, using a variety of pain induction tasks and/or symptomatic patients (e.g., patients with lower back pain), acceptance has consistently been found to increase pain tolerance (e.g., Hayes, Bissett, et al., 1999; Vowles, McCracken, & Eccleston, 2007). For example, Takahashi, Muto, Tada, and Sugiyama (2002) randomized undergraduates to either (a) ACT rationale plus ACT exercise ('Leaves on the Stream' and physicalising techniques, see Appendix E) or (b) ACT rationale plus thought control exercise. Using hand submersion time in the cold pressor bath as a dependent variable, they found that participants in condition (a) demonstrated significantly greater pain tolerance than those in condition (b). This finding suggests that the *exercise*, rather than the rationale, improved pain tolerance. Similar findings have been reported for the reduction of affect (e.g., Karekla, Forsyth, & Kelly, 2004; Levitt, Brown, Orsillo, & Barlow, 2004). For example, Eifert & Heffner (2003) found that an acceptance rational was significantly more effective than a thought control rationale or no instruction at reducing fear and catastophising in anxious females prior to the inhalation of CO2 enriched air.

Perhaps because of the conceptual overlap between acceptance and defusion, dismantling research often fails to discriminate between these two techniques, referring both to acceptance and defusion in the methodology. One study has, however, investigated defusion per se (Masuda, Hayes, Sackett, & Twohig, 2004). Using an alternative treatments design (N = 8), authors compared the effect of three brief techniques on the distress and believability of idiosyncratic negative self-referents (e.g., "fat"). The techniques were (a) de-fusion rationale plus technique (quickly repeating the word for 3 minutes), (b) thought control rational plus technique (try not to think about X), and (c) a control task (reading about Japan). Authors reported that technique (a) was more likely to reduce the distress elicited by, and believability of, the negative self-referent (e.g., "fat"). Although this provides some preliminary support for defusion, further research is required.

Mindfulness has received a large amount of empirical interest. Overall, this research indicates that mindfulness training can reduce physical and psychological distress in symptomatic and asymptomatic participants (e.g., Bowen et al., 2006; Kenny & Williams, 2007; Kingston, Chadwick, Meron, & Skinner, 2007; Ostafin et al., 2006). For example, Davidson et al., (2003) evaluated the effect of an 8-week mindfulness intervention on brain functioning and antibody immunity in asymptomatic participants. They found that, relative to a WLC group, mindfulness trained participants had significantly greater activation in brain regions associated with positive affect (left-sided anterior). Furthermore, these participants had significantly greater antibody filters in response to an influenza vaccine. Research on DBT for BPD patients (e.g., Linehan et al., 2006), and MBCT for treatment resistant depression (e.g., Kenny & Williams, 2008), further suggest that mindfulness-based techniques may have specific utility for chronic disorders. Some preliminary research has also suggested that the effect of mindfulness on psychological distress is partially mediated by reductions in distractive and ruminative behaviours (Jain et al., 2007).

No known research has tested the effect of self-as-context techniques per se. However, the utility of cultivating a sense of self that has present moment focus and that is not defined by VB may be derived from the mindfulness research. For example, Farb, et al., (2007) conducted research into the differentiation between two temporally distinct forms of self-reference; a narrative self-reference (akin to self-as-content) and a sense of self centred in the present moment (akin to self-as-process). Using fMRI, they demonstrated that although these two types of self were usually neurologically integrated, they were dissociated in mindfulness trained participants. Although this does not speak to self-as-context techniques, it suggests that a sense of self grounded in the present moment can (a) exist in a way that is distinct from a more verbal sense of self and (b) be cultivated by mindfulness training. Similarly, mindfulness literature has also found that cultivating a perspective that observes thinking as a process rather than a source of self-definition (i.e., de-centred perspective) is significantly predictive of positive treatment outcomes (e.g., Ma & Teasdale, 2004).

Finally considering values and committed action, research from many schools of psychology suggests that values can play an important role in psychological well-being. For example, Creswell et al. (2005) randomised undergraduates to either a value-affirmation task (answering questions about their top ranked values) or a control task

(answering questions about their lower-ranked value). This was followed by a laboratory stressor; a 5-minute public speaking task evaluated by two confederates. They found that relative to controls, value affirmation participants had significantly lower post stressor cortisol levels and rated the task as significantly less stressful and threatening. Similar research has also found that affirming one's values reduces rumination after failure (Koole, Smeets, van Knippenberg, & Dijksterhuis, 1999) and defensiveness after receiving threatening information (Sherman, Nelson, & Steele, 2000). The literature on committed action is sparse; however, Amrhein, William, Yahne, Palmer, and Fulcher (2003) found that patients' degree of commitment to abstinence significantly predicted outcomes in an alcoholic cohort. Similarly, Kulik and Carlino (1987) found that public commitment procedures improved compliance with behaviour change.

2.2.3 *Evidence for ACT as an Integrated Treatment Package*

Consistent with the idea that common processes underlie dissimilar clinical disorders, ACT has shown promising effects when applied to the treatment of several different patient groups. These include trichotillomania, social phobia, smoking, polysubstance abuse, agoraphobia, depression, anxiety, interpersonal problems, psychosis, social anxiety disorder, chronic pain, and BPD (see Hayes et al., 2006). When summarised statistically, this outcome research suggests that compared to WLC, TAU, and/or placebo control, ACT had a weighted average effect size (ES) of d = 0.99 (total N = 284) at post-test and d = 0.71 (N = 176) at follow-up (average of 19.2 weeks; Hayes et al., 2006). Moreover, when compared to well-specified, disorder specific treatment packages such as CBT and psychoeductaion, results showed a weighted average ES in favour of ACT at post-test (d =0.48), which *increased* to d =0.63 at follow-up (mean 26 weeks). This rise in ES from post-test to follow-up reflects a trend often seen in ACT outcome trials (e.g., Gifford et al., 2004 (smoking cessation ACT vs. Nicotine replacement); Hayes, Bissett et al., 2004 (therapist burn out); Lundgren, Dahl, Melin, & Kies, 2006 (epileptic seizures and quality of life). That is, while comparison conditions tend to show, at best, gains that are sustained from post-test to follow-up, ACT often obtains *continued gains*.

These findings are promising and suggest that ACT may be at least as good as front line treatments (i.e., CBT). However, ACT is a relatively modern intervention and because of this, research is predominantly in the pilot phase of investigation (see chapter 3). Indeed, although a more recent meta-analysis reported similar ESs for ACT, and argued *for* its clinical efficacy, Ost (2008) highlighted a number of key methodological weaknesses. The most pointed of these included the use of inactive (i.e., WLC) or ill-defined TAU comparison groups, the failure to diagnose patients using standardised measures, insufficient sample sizes, and non-standardised treatment protocols. These methodological features, and their place in research, are discussed in chapter 3. For now, however, it is important to note that ACT outcome research, although promising, is nevertheless preliminary. The following section reviews the main outcome trials, starting first with ACT for acute disorders, followed by tentative research on ACT for more treatment resistant patients.

2.2.3.1 *ACT for Acute Disorders.* One of the largest ACT trials was conducted by Forman, Herbert, Moitra, Yeomans, and Geller (2007), who randomised treatment seeking university students (N = 101) to either ACT or CBT. Participants were described as having 'moderate to severe' mood and interpersonal problems, with a range of acute disorders reported (depressive disorder (34%), anxiety disorder (32%), adjustment disorder (11%), method of diagnosis not specified). Therapy was individual, self-terminating (mean of 15 sessions), and delivered by CBT trained clinical graduates (N = 23) who had received additional ACT training. Clinicians delivered both ACT and CBT and treatment fidelity was tested and verified. Post-treatment outcomes, measured using a range of patient and clinician ratings, indicated that both interventions obtained similar effects. Preliminary analyses of mechanisms of change were conducted by assessing whether *changes* in the hypothesised mediators (e.g., AAQ) co-occurred with *changes* in symptom severity. These analyses suggested ACT and CBT obtained effects through different mechanisms. In the ACT group, changes in the AAQ, acting with awareness, and acceptance were all significantly related to symptom change. Conversely, in keeping with Teasdale et al.'s (2002) findings, the ability to observe private events was significantly related to change in CBT. These preliminary investigations are insightful; however, because measures were taken at the same point in time, cause-and-effect cannot be determined (see section 3.1.2).

A similar trial has been conducted by Lappalainen et al. (2007) in Finland. These authors actively recruited community patients via newspaper advertisements and randomised them to either ACT or CBT. As in Forman et al.'s (2007) trial, patients presented with mood and/or interpersonal complaints (N = 28). Therapy sessions were individualised and interventions were delivered by CBT trained post-doctoral trainees who had received additional ACT training. Unlike the former trial, however, this trial reported medium to large between-group ES for reductions in global symptom severity that favoured ACT. Differential mechanisms of change were again implicated: CBT enhanced self-confidence and ACT did not; ACT decreased AAQ scores and CBT did not. Lappalainen et al. also reported that post-treatment symptom severity was significantly associated with self-confidence in the CBT condition and AAQ scores in the ACT condition. These analyses simply involved correlating post-treatment process and symptom scores and 6-month process and symptom scores (i.e., not assessing change or cross-lagged correlations). Thus, because these measures were taken at the same point in time, and were not based on change scores, they are also unable to elucidate causal relations.

Two trials have compared ACT versus TAU for psychosis (Bach & Hayes, 2002; N = 80; Gaudiano and Herbert, 2006; N = 40). Patients in both trials met diagnostic criteria for psychosis and approximately one third of patients in the first study and half of patients in the second study had co-morbid PD and/or Substance Dependency Disorder. These more severe characteristics are typically excluded from RCTs on CBT for psychosis (see Gould, Mueser, Bolton, Mays, & Goff, 2002). Despite brief intervention periods (4 and 3 sessions respectively), both trials reported that re-hospitalisation at 4-month follow-up was 50% lower in the ACT condition than TAU. Furthermore, significant reductions in psychiatric symptoms and hallucination distress and believability were also reported. Comparing the ES reported by Gaudiano and Herbert to those reported in a meta-review on CBT for psychosis (Gould et al.), showed that ESs were comparable (ACT d = 0.60, CBT d = 0.65). However, ACT obtained these effects using a briefer intervention period than CBT (mean 3.2 versus mean 13.6 respectively) and by treating a more complex patient group. Finally, Gaudiano and Herbert conducted a comprehensive assessment of mediation (Baron & Kenny, 1986; see section 3.4.3) and found that the effect of treatment on psychiatric symptoms was

mediated by reductions in hallucination believability. These mediational analyses are promising but were based on a small sample (N = 29) and thus require replication.

Two trials have piloted ACT for social anxiety disorder. In a repeated measures within-subjects design, Dalrymple and Herbert (2007) measured psychological distress and social functioning over a 4-week baseline control period. This was compared to post-treatment measures following 12-weeks of individualised ACT. Multiple measures, including self-report, clinician report, and behavioural tasks (e.g., public speaking task) were used to assess social functioning. Behavioural tasks were recorded and rated by assessors who were blind to the aims of the study and to the testing period of each recording they rated (i.e., pre-treatment versus post-treatment). Despite the small sample size (completers N = 12), patients rated themselves, and were rated by others (i.e., blind assessors), as significantly less anxious following treatment as compared to baseline. Preliminary analyses into mechanisms of change were again computed, indicating that changes in the AAQ during the first half of treatment preceded and predicted subsequent change in symptom severity. These findings are promising but, again, the sample size was particularly small.

The second study applying ACT to social phobia was conducted by Ossman, Wilson, Storaasli, and McNeil (2006), who used an uncontrolled trial to obtain preliminary information on group-based delivery of ACT for treatment seeking patients with Social Phobia (SP; 10-week intervention). They also found significant reductions in self-reported SP symptoms and anxiety, and gains continued over the 3-month follow-up. Exploratory investigations into mechanisms of change, using the same method adopted by Lappalainen et al. (2007), demonstrated a high correlation between symptoms and experiential avoidance. However, because variables were measured at the same time point, the direction of causation cannot be ascertained.

A further two trials have assessed the relative effects of an early ACT protocol (then called ‘Cognitive Distancing’; CD) to CBT for patients with depression (Zettle & Hayes, 1986, N = 18; Zettle & Raines, 1989, N = 31). In both these trials, clinicians were described as primarily ‘Beck-trained’ (see Hayes et al., 2006), receiving additional CD training. Both trials tested the relative effects of 12-weeks of CD versus CD+CBT or CBT alone. Results from the first trial showed that although there were significant reductions in all conditions, post-treatment and 2-month follow-up depression scores favoured CD relative to CBT. Furthermore, a reanalysis of these data suggested that

changes in the believability of depressogenic thoughts mediated superior treatment gains for the CD condition (see Hayes et al., 2006). Unlike the first trial, results of the second trial showed equivalent gains across groups. These trials tentatively suggest that CD was clinically beneficial for depressed patients, but the extent to which CD reflects current ACT protocols is unclear.

The ACT outcome trials that have been reviewed tentatively suggest that it may be as effective as CBT for some acute disorders. Findings have also been consistent with hypothesised mechanisms of change and some data suggests that ACT may achieve effects using shorter intervention periods than CBT (also see section 2.3.3.2). It is already clear from chapter 1, however, that CBT is usually quite effective for the treatment of acute disorders. A more pressing question, therefore, is how effective is ACT when applied to the treatment of patients typically more resistant to CBT (see section 1.2.4)? To date, research has not ventured into testing whether ACT may have utility for more chronic, co-morbid and/or PD groups. Three trials tentatively support such an application, however.

2.2.3.2 *ACT for Hard-to-Treat Patients.* The first of these trials was conducted by Hayes, Wilson et al. (2004), who investigated ACT versus methadone maintenance (MM) versus Intensive 12-Step Facilitation program (ITSF) for polysubstance abusing patients (N = 109). At baseline, most of this group had co-morbid disorders: 40% were reported to have a mood disorder, 42% an anxiety disorder, and 52% were reported to meet criteria for at least one PD (N = 57). Furthermore, patients had attended a substantial number of previous residential or outpatient treatments (mean = 6.5). Participants received 32 individual and 16 group sessions. Using urine specimens as a measure of abstinence, comparable post-treatment effects for ACT and IFTS were found, with both superior to MM. Moreover, although not significant, 6-month follow-up data suggested that ACT obtained long term effects that exceeded ITSF (Figure 2.4). The AAQ was not, however, associated with change. This makes it difficult to elucidate how ACT affected outcomes.

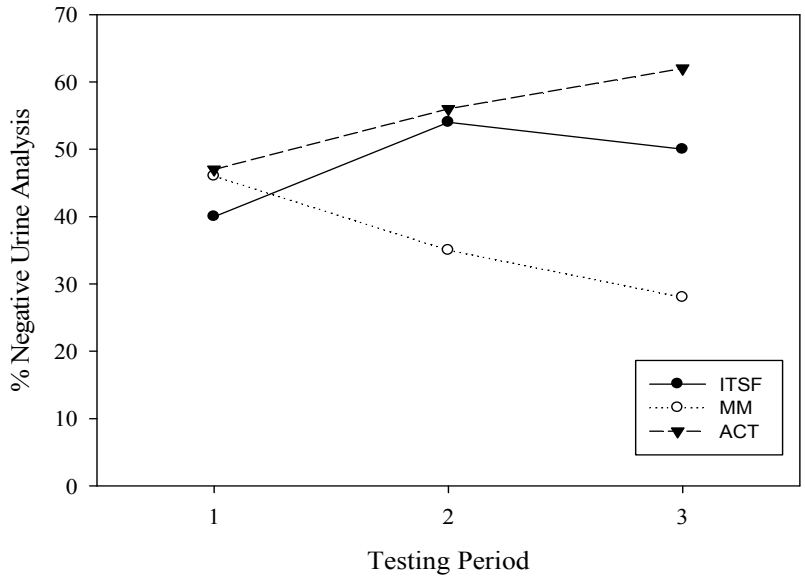


Figure 2.4 Outcome Data for Percentage of Clear Urine Analysis Samples in an Opiate Using Clinical Sample Following ACT, IPSF and MM

From *"A Preliminary Trial of Twelve-Step Facilitation and Acceptance and Commitment Therapy with Polysubstance-Abusing Methadone-Maintained Opiate Addicts,"* by S. Hayes, K. Wilson, E. Gifford, R. Bissett, M. Piasecki, S. Batten et al., 2004, *Behaviour Therapy, 35(4),* p. 667-688. Note: '1' = baseline, '2' = post-test and '3' = 6 month follow-up.

Results from a trial by Gratz and Gunderson (2006) also suggest that ACT may be useful for hard-to-treat patient groups. They randomised self-harming BPD patients (N = 24) to either 16-weeks of an 'ACT-DBT hybrid' plus TAU or TAU-alone. The ACT-DBT hybrid included approximately 70% ACT and 30% DBT. Significant between-group differences were obtained; patients receiving ACT-DBT+TAU were significantly less likely to self-harm relative to TAU at post-test. ACT-DBT+TAU patients also showed a significant reduction in BPD symptom severity. This was accompanied by significant reductions in the AAQ, but Gratz and Gunderson did not report whether this was related to behaviour change. Although the intervention was brief relative to other BPD interventions (e.g., Giesen-Bloo et al., 2006), approximately 50% of patients receiving ACT-DBT+TAU experienced clinically reliable reductions in global psychiatric functioning (using Jacobson & Truax's, 1991 criteria; see chapter 3). Because Gratz and Gunderson tested a hybrid of ACT and DBT, however, the specific affect of ACT techniques cannot be ascertained.

The final trial to implicate the effectiveness of ACT-like techniques for chronic disorders was conducted by Dimidjian et al. (2006). Dimidjian et al. randomised treatment seeking depressed patients (N = 241) to CBT, Behavioural Activation (BA), or antidepressant medication. BA bore noticeable similarity to key ACT techniques: "(BA) highlights the centrality of patterns of avoidance and withdrawal *(experiential avoidance)*…and seeks to identify and promote engagement with an individual's long term goals *(values and committed action)*... The model also includes an increased focus on the assessment and treatment of avoidance behaviours *(experiential avoidance)*... and on moving attention away from the content of thoughts *(cognitive fusion)* towards direct immediate experience *(mindfulness)*" (p. 672, brackets added). Dimidjian et al. found that although the effects of CBT and BA were comparable for less severely depressed patients, a significant difference emerged when considering the most severe subsample (N = 61). Patients with more severe depression experienced significant improvements following BA and medication *but not* following CBT. Similar findings have been reported by Elkin et al. (1995) and Ma and Teasdale (2004), and tentatively suggest that ACT-like techniques may be effective for the treatment of patients with more severe symptoms. Again, however, ACT was not being directly assessed.

To summarise: although the research is in its infancy, and the story is still unfolding, ACT appears to hold promise. It has produced notable effects for a range of clinical disorders, even when delivered as a brief and group-based intervention. Furthermore, when compared to CBT for acute disorders, the data tentatively suggest that ACT could be as effective as this front line treatment. Also, preliminary indications suggest that ACT may achieve change using fewer treatment sessions and through theory consistent mechanisms. Perhaps more importantly, preliminary data further suggest that ACT and ACT-like techniques may be useful for patients typically more resistant to traditional CBT. The key word, however, is *tentative*. ACT is a relatively new therapy and because of this, much of its research remains in a pilot phase of investigation (see chapter 3). For example, few trials have had sufficiently large samples to conduct powered analyses and only a small cluster has begun to investigate mechanisms of change. Furthermore, most of this research has been restricted to American samples. ACT is not yet an empirically supported treatment (Ost, 2008), and only with more rigorous and replicable investigations can its effects be thoroughly tested.

2.3 Chapter Summary and Global Aims of the Thesis

This chapter aimed to provide a thorough account of ACT by reviewing its theoretical underpinnings, therapeutic techniques, and outcome data. It will conclude by reiterating a few main points that will lead into the broad aims of the thesis. Firstly, not anomalous to the first and second wave treatments that pre-date it, the congruence between RFT theorising and ACT treatment techniques are arguably more analogous than empirical. As with these previous treatments, it is also likely that the clinical successes of ACT will not rest on the laboratory successes or failures of RFT. Secondly, although the applied work is largely supportive of ACT's effectiveness (Hayes et al., 2006), the full range of its applicability and the mechanisms through which it obtains effects are yet to be thoroughly tested. Investigations which compare ACT and CBT may be central to quantifying how—in terms of theory and practice—ACT differs from this main stream approach (Ost, 2008). Finally, this review has aimed to highlight one area of research that seems particularly worthy of attention; that is, the possibility that ACT may be useful for patients who have been resistant to, or relapsed following, standard psychological treatment. This is by virtue of the fact that, according to ACT, the range of symptoms that this group present with are commonly maintained by excessive experiential avoidance. In theory, therefore, ACT should be effective for this heterogeneous cohort even when delivered as a group-based treatment. Such an investigation would provide a good test of ACT's generic applicability, while also evaluating whether it has clinical benefits above and beyond existing techniques.

Archetypically, treatment resistant patients present with longstanding, chronic and co-morbid symptomatology. Additionally, they will often engage in a range of maladaptive behaviours which, according to ACT, are motivated by experiential avoidance (Hayes et al., 1996). Although a decade has passed since Hayes et al.'s seminal paper was published, this proposition is yet to be empirically tested. The empirical chapters of this thesis will adopt a systematic, theoretical and applied programme of research, designed to investigate the role of the experiential avoidance in co-occurring maladaptive behaviours and to pilot test ACT for treatment resistant patients. With these aims in mind, the following chapter discusses methodological issues and statistical techniques for evaluating complex psychological treatments.

CHAPTER III

Methodological Issues and Statistical Techniques

The previous two chapters identified ACT as a promising intervention for patients with a range of clinical disorders and perhaps even for those with more longstanding difficulties. Several gaps in knowledge were identified, however, both in terms of ACT theorising and outcome research. This thesis aims to address those gaps most relevant to ACT for treatment resistant groups. The purpose of this chapter, therefore, is to provide a concise summary of research methods and statistical techniques in common use in developing and validating novel clinical treatments. The chapter begins by discussing the major varieties of research design and the main techniques for measuring study variables. These discussions aim to inform *how* treatments may be evaluated, but do not consider when in the evaluation process particular approaches are most appropriate. Latter sections of this chapter thus discuss stages of treatment evaluation and statistical techniques. Some, but not all, of the techniques covered in this chapter feature in the thesis. Those used receive greater coverage.

3.1 Research Designs in Clinical Research

Clinical research is usually concerned with evaluating the effects of different treatments and/or testing the theoretical assumptions upon which they are based. For example, is ACT an effective intervention for treatment resistant patients, and do independent variables (IVs; e.g., treatment techniques) affect dependent variables (DVs; e.g., patient symptoms) in theory consistent ways? A variety of designs can be used to test these types of questions, and these differ mainly with regard to control procedures. Experimental research is designed to maximise internal validity; the validity of conclusions regarding cause-and-effect relations. This is achieved using rigorous control procedures. Although high internal validity is desirable, it is not always feasible, ethical, and/or appropriate in clinical settings. Less controlled approaches, such as quasi-experimental and non-experimental designs are thus frequently used. The following section discusses the major types of research design and allocates specific attention to Time as an important variable for determining causation.

3.1.1. *Control Procedures*

Experimental designs manipulate an IV and measure the effect of that manipulation on a DV(s). Although many variants of this model exist, the common principle is to hold all factors constant except the IV, thus isolating its effect from that of spurious variables.

The randomised control trial (RCT) is the most highly regarded experimental design for outcome research (e.g., Chambless & Hollon, 1998). Participants are randomly allocated to either the treatment or a control condition(s), and between-group comparisons are used to test whether the treatment has a significantly greater effect on the DV(s) than the control. Randomisation ensures between-group comparability at baseline, thus controlling for the possibility that post-treatment differences are attributable to pre-existing group differences. RCTs also typically sample patients with *homogeneous symptoms*, meaning that patients are as similar as possible on dimensions such as illness type and symptom severity. This is desirable because large within-group heterogeneity obscures the detection of small treatment effects by increasing variability in treatment response (Donenberg, Lyons, & Howard, 1999). This variability threatens statistical conclusion validity; the probability of rejecting the null hypothesis when it is false (Shapiro, 1996). Uncontrolled variation in the delivery of treatments also threatens statistical conclusion validity. Treatments are thus typically delivered in keeping with detailed treatment manuals and adherence is rigorously checked. This helps to ensure that treatments are delivered competently and with consistency across clinicians and treatment sites. Finally, the potential for experimenter bias is reduced by blinding assessors to group allocation. Together, these features provide a tightly controlled environment designed to isolate the specific effects of a treatment.

Although the RCT has good internal validity, it has some limitations. These mostly arise from the fact that the persons and settings studied in RCTs often poorly represent those of clinical practice (see Westen et al., 2004). This threatens external validity; the validity of generalised inferences regarding cause-and-effect. RCTs compromise external validity in several ways. For example, in everyday practice, therapy is seldom delivered according to treatment manuals; rather, it tends to be self-correcting and integrates techniques from different theoretical models (Clarke & Rienecke, 2003; Westen et al., 2004). Likewise, homogeneous sampling typically under-represents those patients usually seen in clinical practice. For example, Westen et al. (2004) found that RCTs *excluded* between 40% and 70% of standard care patients, with co-morbidity and

PD symptoms being the most common justification. In addition to issues of generalisability, RCTs can be difficult to conduct in clinical settings. For example, it is not always appropriate or ethical to randomise patients to a no-treatment control group. Furthermore, a greater proportion of participants allocated to control conditions drop-out of treatment (selective attrition), thus undermining the assumption of group equivalences at baseline (see section 3.4).

There are many alternatives to the RCT, such as *patient series*, *pre-post*, and *quasi-experimental* design. Patient series and pre-post design are based on within patient comparisons. Patient series is a naturalistic approach in which the clinician delivers treatment as they would in usual practice, but uses regular observations to quantify effects. This approach aims to maintain a "clear focus on the individual rather than the average group member" (Owens, Slade, & Fielding, 1996, p. 231), but uses a series of patients to systematically develop techniques over time. Pre-post design, on the other hand, simultaneously delivers the same treatment to a group of patients and assesses change by comparing the groups' pre-treatment scores to their post-treatment scores. *Quasi-experiments* are a further option. These mimic experimental design but do not use randomisation. For example, non-equivalent group design compares groups that are already intact, such as patients already enrolled on a course of treatment or groups with innate differences (e.g., males versus females). Compared to RCTs, these alternative designs have two main strengths; they are easier to conduct and provide a more accurate reflection of treatment as delivered in clinical practice. The main limitation, however, is that they lack internal validity. In all three of the approaches described, a range of uncontrolled rival hypotheses make it difficult to ascertain what proportion of an effect is attributable to the treatment.

Non-Experimental design is also used in clinical research. This approach does not manipulate an IV, nor does it procedurally control for extraneous variables. Instead it analyses the relationship between multiple variables to identify structural or functional relations. This approach is often used to test the theoretical model underpinning a treatment. Epidemiological research is a good example, which not only documents population rates of a disorder, but also aims to "detect factors associated with its origin, course and outcome" (Leighton, 1979, p. 235, cited in Cooke, 1996). Indeed, although non-experimental design does not manipulate an IV, it can contribute to knowledge regarding cause-and-effect relations. This is made possible by pre-specifying, based on

a firm theoretical foundation, the nature of the variables being tested (e.g., IV (predictor), DV (criterion)[10], mediator) and their anticipated relations. Multiple Regression (MR; section 3.4.2) or Structural Equation Modelling (SEM; section 3.4.3) can be used to test for those relations. For example, mediational analysis (section 3.4.3) is often used to test whether a predictor (e.g., negative affect intensity) predicts variance in a criterion (e.g., BPD symptomatology) indirectly and through the effect of a mediator (e.g., thought suppression; Cheavens et al., 2005).

Non-experimental design has several strengths. It is easy to conduct and requires few resources, but allows for the collection of large data sets. Furthermore, it is suited to testing multiple inter-variable relations. This is a common characteristic of models that underpin clinical treatments. Its main limitation, however, is that because it does not involve the manipulation of an IV, it is less internally valid than experimental design. Although it has been argued that finding evidence to support complex patterns of inter-variable relations, such as mediation, reduces the number of plausible competing hypotheses (Anderson & Bushman, 1997), non-experimental research is nonetheless unable to ascertain whether one variable *caused* another.

3.1.2 *Time as a Variable*

Time, as a variable, is also an important consideration for clinical research. Clinical research is typically cross-sectional or prospective. Cross-sectional research, usually non-experimental in design, tests the associations between variables that are measured at the same point in time. Although measured concurrently, one variable typically has antecedent status (i.e., it is conceptualised as the IV). For example, cross-sectional research has been used to test whether a historic event, such as a negative life event, predicts current levels of a criterion, such as depression (see Kessler, 1997). Prospective research, on the other hand, takes several measures at different time periods. The RCT is thus a typical prospective design because it measures the DV both before and after treatment. Although prospective research is usually experimental in design, non-experimental research can be used to obtain data sets from the same sample on two or

[10] Because non-experimental research does not involve the manipulation of an IV, the terms IV and DV do not correctly apply. Thus, non-experimental research use the terms ‘predictor’ and ‘criterion’ (respectively).

more occasions. These prospective non-experimental designs thus allow for an examination of lagged correlations, potentially identifying causal relationships without the direct manipulation of an IV.

Both approaches have strengths and limitations. Cross-sectional research is inexpensive, quick, and easy to conduct. It also lends itself to the collection of large datasets, which enable detailed and powered multivariate analyses (see section 4.1.3). These strengths are offset by some important limitations, however. Because cross-sectional research invariably measures the IV retrospectively, it is subject to memory biases and distortions (e.g., over-generalised and mood-congruent recall). These sources of error are often especially pronounced in clinical samples (e.g., Kuyken & Brewin, 1995; Russo, Fox, Bellinger, & Nguyen-Van-Tam, 2001). Additionally, it is impossible to obtain any objective baseline measure against which the effect of that IV can be compared. Cross-sectional research is thus low in internal validity; measuring variables concurrently, it is not possible to show that one preceded and caused the other. The strengths of prospective research parallel the limitations of cross-sectional. Because participants are followed over time, inferences regarding cause-and-effect are typically more valid than in cross-section research. This is because one can follow the predicted temporal path of relations between variables, systematically testing whether changes in one preceded and caused changes in the other. A disadvantage to this approach, however, is that it is time consuming and resource intensive. Furthermore, periodic testing (i.e., repeated measures) can produce reactive changes in measures of a DV that are not directly attributable to the IV.

Given the strengths and weakness of the several designs that have been considered, it seems reasonable to conclude that there is no one perfect design in clinical research. Rather, it has been described by Shapiro (1996) as a “creative compromise”, which involves balancing tensions of validity and practical feasibility. Resolving these tensions is influenced by the stage of evaluation that the treatment is in (see section 3.3.). Before considering these stages, however, the following section discusses the various methods that are available for the measurement of study variables.

3.2. Measures Used in Clinical Research

There is a wide range of choice in selecting how to measure study variables. Self-report, observational, and physiological measures have all been used. Each has relative advantages and drawbacks.

3.2.1 *Self-report*

Self-report measures, such as questionnaires or interviews, are a common procedure for explicitly measuring variables of interest. Self-report is useful for measuring clinical phenomena ethically and anonymously. It can also be used to efficiently summarise information about broad constructs (e.g., global symptom severity) and to measure events retrospectively. Questionnaires are easy, quick, and inexpensive to administer. Furthermore, standardised measures facilitate direct comparisons across patients, treatments and populations, which can be used to inform treatment decisions. Although interviews are more resource intensive than questionnaires, they are often favoured because they can provide less constrained information. Indeed, because questionnaires force participants to answer in pre-defined ways, they may bias findings and/or omit important information. Interviews can partly overcome this by using open-ended techniques, but the loss of anonymity can limit the quality and quantity of data. Although self-report techniques have many strengths, a key limitation is that people seldom have access to full and accurate accounts of past or present experiences. For example, people are often unaware of the contingencies that affect behaviour and verbally accessible accounts, at best, approximate true relations (e.g., Nisbett & Wilson, 1977). These measures can thus have high levels of subjective inference. Furthermore, they can be reactive to repeat testing.

3.2.2 *Observational*

Observational measures directly observe phenomena of interest. Observations can be taken from the natural environment, such as measuring how often, and when, a patient with OCD engages in checking behaviour within their own home. Alternatively, observations may be made in the clinical or laboratory setting. Observational measures are useful because they allow the researcher to quantify behaviour, its antecedents and consequences objectively, rather than relying on the participant's verbal codification of

those relations. Furthermore, observational data are less susceptible to subjective inference than self-report. For example, operational definitions and blind second ratings of observations heighten the objective reliability of these measures. Also, measures can be used to quantify activity that is not under voluntary control, such as eye gaze. These measures are useful because they are less reactive to repeat testing and demand bias. Because this approach uses direct observation, however, it is seldom used to investigate harmful behaviours unless through the means of an analogue procedure (see section 3.2.1). Furthermore, the introduction of observers and/or recording equipment may produce reactive change in the behaviour observed.

3.2.3 *Physiological*

Physiological measures quantify the activity of biological processes such as heartbeat, skin conductance, and cortical activity. These can be used in clinical research by, for example, providing a baseline assessment against which intervention effects may be measured. For example, the effects of a treatment on stress reactivity can be tested by comparing pre-post measures of cortisol. These modern methods are unique because they have the ability to identify, sometimes in real time, the locus of effects of experimental variables on biological functions (Coles, 2003). It is well recognised, however, that these parameters have many associated difficulties (Edelmann, 1996). They can be difficult to implement and intrusive. This is an important consideration for applied research. Additionally, these techniques are resource intensive, expensive, and often require specialists for their correct operation. Furthermore, psychophysiological measures are usually taken in laboratory settings. The correspondence between the behaviour of these processes in this versus naturalistic settings is relatively unknown. What is apparent, however, is that psychophysiological readings are often reactive to subtle changes in the stimuli and testing environment (Edelmann).

So far, this chapter has considered the many research designs and measurement approaches that are in common use in clinical research. Two other important considerations are reviewed in the sections that follow. The first is when in the research process particular approaches are more or less appropriate, and the second is the statistical techniques available to analyse the data obtained (although analysis is intimately related to the design adopted).

3.3 Stages of Clinical Research

In recent years, clinical research methodology has been codified on the basis of logic and the experience of a number of researchers (e.g., Medical Research Council (MRC); Campbell et al., 2000; Tull, Bornovalova, Patterson, Hopko, & Lejuez, 2008). It is now widely accepted that there is a four stage research process in the development and evaluation of a new clinical intervention. These stages, depicted in Figure 3.1, include (usually in this order) analogue research, pilot trials, RCTs, and field effectiveness (FE) research. Each stage is discussed below.

3.3.1 *Analogue Research*

Treatment evaluation usually begins by testing key theoretical assumptions upon which a treatment is based. In the psychological literature, this stage is often called analogue research: "research focused on gaining a clear understanding of the processes underlying the development and maintenance of psychopathology ... with the goal of understanding more basic processes, which may be used to guide additional research focused on the generalisability of findings to clinical phenomena" (Tull et al., 2008, p. 77). According to Tull et al., research can be analogue either with regard to procedures or sample characteristics. *Analogue procedures* artificially simulate clinical phenomena, such as experimentally inducing anxiety or fear, and then test theory-driven predictions about its nature and/or modification. For example, Eifert and Heffner (2003) have used the "biological challenge" (inducing hyperventilation through the inhalation of CO_2 enriched air) to compare the effect of acceptance, avoidance, and control techniques on anxiety. *Analogue sampling* refers to the use of non-clinical or sub-clinical samples to investigate clinical phenomena. For example, researchers often recruit asymptomatic participants (Cheavens et al., 2005) or those with sub-clinical symptoms (e.g., Foa, McNally, & Murdock, 1989).

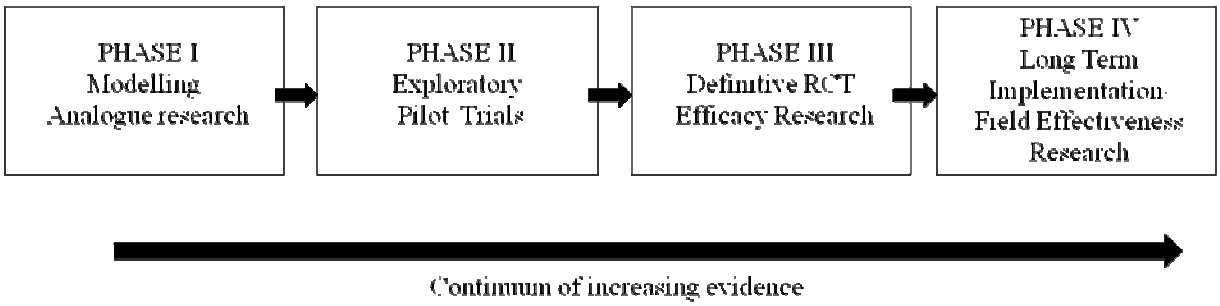


Figure 3.1 Phase-Based Approach to Developing and Evaluating Complex Treatments

Adapted from "Framework for Design and Evaluation of Complex Interventions to Improve Health" by M. Campbell, R. Fitzpatrick, A. Haines, A. Kinmonth, P. Sandercock, and D. Spiegelhalter, 2000, *British Medical Journal,* 321, p. 694. Note: Adaptation maps medical terms used by the MRC ("modelling", "exploratory", "definitive RCT" and "long term implementation") to corresponding terms used in psychological research ("analogue", "pilot trials", "efficacy", "field effectiveness" research, respectively).

It is not hard to discern why clinical research begins with analogue investigations. One reason is that laboratory analogues are tractable, offering a level of precision that is hard to achieve in clinical settings (Bandura, 1987; Watts, 1996). These conditions thus provide a useful means of addressing specific theoretical questions. For example, testing the effect of acceptance on naturally occurring anxiety in clinical practice may be confounded by variability in treatment implementation, co-morbid symptoms of the patient, patient therapist interactions, and/or demand characteristics. Analogue research, on the other hand, can overcome these confounds by using asymptomatic patients, artificially simulating clinical phenomena and standardising procedures.

Analogue procedures have proved particularly useful for investigating behaviours that cannot be investigated ethically in-situ, such as risk taking. For example, Lejuez et al. (2002) developed a computerised gambling task (the Balloon Analogue Risk Task, 'BART') in which participants have the opportunity to win or lose money, but where persistent risk taking increases the probability of losing. The BART thus measures the tendency to take risks in a way that is claimed to be analogous to real life risk-taking. Although procedures of this kind cannot capture the true phenomenology of risk taking, this approach can provide an ethical means of measuring, manipulating and better understanding processes thought to underlie it.

Analogue sampling has also long proved useful in clinical research, primarily because non-clinical samples do not incur the same ethical risks as clinical counterparts. For example, although scientifically desirable, it would be unethical to randomise emotionally vulnerable patients to a no-treatment control group and then induce anxiety (e.g., see Eifert & Heffner, 2003). Similar risks are incurred even when emotions are not directly manipulated, such as when investigating emotionally evocative variables (e.g., trauma and abuse). Theory testing in clinical samples is thus uncommon, but it is by no means impossible; rather, tackling these issues requires specific procedures. For example, ethics committees may require that clinical groups participate in a safe environment (i.e., a clinic), that they are currently in therapy, and that a therapist is available post-participation. Of course, this is not always possible and such provisions may affect the accuracy of the data collected. For example, disclosure may be compromised when patients participate at the clinic where they receive treatment. Similarly, responses may be influenced by that treatment. These difficulties are further compounded by the fact that clinical samples are notoriously hard to recruit and attrition is often high. Theory testing on clinical samples is thus time consuming and often suffers from low statistical power.

Although analogue research has many advantages, the external validity of this approach is frequently debated (e.g., Bandura, 1978; Weary, Edwards, & Jacobson, 1995; Vrendenburg, Flett, & Krams, 1993). The generalisability of analogue procedures rests on the extent to which they accurately simulate clinical symptoms. The generalisability of analogue samples rests on the extent to which 'clinical' samples differ quantitatively, but not qualitatively, from 'non-clinical' samples. Overall, research suggests that these approaches exhibit acceptable external validity. For example, reviewing the aggression literature, a meta-analysis by Anderson, Lindsay, and Bushman (1999) found strong congruence (r = .75) between the findings of laboratory-based and field-based studies (see also Anderson & Bushman, 1997). Similarly, research on depression and psychosis has shown evidence for phenomenological continuity across non-clinical, sub-clinical, and clinical samples (e.g., Enns, Cox, & Borger, 2000; Johns & Van Os, 2001).

3.3.2 *Pilot Trials*

According to the stage-based approach to treatment evaluation, the most logical progression from theory-testing is to 'test run' a treatment (Campbell et al., 2000; Lancaster, Dodd, & Williamson, 2002). Pilot trials are designed for this very purpose, aiming to obtain preliminary information about the effect of the treatment on small clinical samples (approximately 30 participants; Browne, 1995). Pilot trials provide vital information regarding the use of a treatment with certain patient groups, and this information may in turn be used to justify expensive and rigorous stage three trials (i.e., powered RCTs).

Pilot trials do not adopt one uniform research design. Instead, the design is usually guided by (a) the amount of information already available for the treatment and patient group in question, (b) the ethical risks associated with the patient group, and (c) practical constraints. When the application is novel and the group are considered to be high risk, patient series, pre-post and/or quasi-experiments are typically used (Shapiro, 1996; Spokas, Rodebaugh, & Heimberg, 2008). These designs are suited to pilot trials because they provide some indication of treatment effects in a research context that can flexibly adapt to the needs of the patient. For example, if aspects of the treatment prove to be ineffective or require modification, the clinician can adapt the protocol accordingly. This flexibility is usually not afforded in RCTs, but is vital for developing the treatment manuals that they use (Campbell et al., 2000). Pilot RCTs are particularly useful (Lancaster et al., 2004). This is not only because they implement more rigorous controls than other designs, but also because they more directly inform powered RCTs (section 3.3.3). For example, pilot RCTs can test run specific design procedures (e.g., randomisation), provide realistic estimates of recruitment duration, pilot measures, and explore optimal treatment delivery (e.g., mode of delivery and duration). They are also vital for making realistic and informed power calculations.

Although pilot trials are useful as an early stage of treatment evaluation, they cannot determine the efficacy of a treatment. They are usually underpowered, lack sufficient control procedures, have limited external validity, and, even when randomisation is used, imbalances can exist between groups at baseline (Lancaster et al., 2004). Thus, having test-run a treatment, stage three of evaluation is concerned with *efficacy* research.

3.3.3 *Efficacy Research*

Efficacy research is interested in discovering the specific effects of a treatment on an outcome (Wells, 1999). This requires optimal control procedures, isolating the treatment from non-specific treatment effects such as therapist contact and spontaneous remission. This stage of research is thus exclusively concerned with powered RCTs. A powered RCT recruits sufficient participants to ensure that the probability of detecting a significant between-group difference, should one exist, is at least 80% (adjusted cell size for attrition).

Although efficacy research is exclusively concerned with powered RCTs, the nature of the control group usually increases with rigour throughout this stage. Early RCTs typically evaluate the treatment against a wait-list control. In this design, control participants receive treatment after, rather than during the trial, and effects are evaluated relative to the passage of time. If found to be more effective than no treatment, the next type of comparison is usually TAU; control patients are assigned to an ecologically valid alternative treatment. Although the premise of TAU is to control for non-specific treatment effects, such as therapist contact and treatment duration, TAU trials are rarely sufficiently rigorous. In practice, TAU often refers to a culmination of various different treatment approaches with little or no indication of contact hours or the techniques used. The most rigorous comparison, therefore, is an active and well structured comparison treatment (Ost, 2008). The aim here is to control for all possible confounds, leaving only treatment techniques to vary between groups. Because it is unethical for the comparison to be inert, this approach usually involves comparing the treatment to one that has already been established as effective. This type of trial thus usually aims to test whether the new approach offers any additional benefits above-and-beyond existing techniques, such as superior outcomes or a better cost-effectiveness ratio.

As discussed at the start of this chapter, the RCT is an internally valid method for testing treatment efficacy in idealised settings. Some of its control procedures, however, impinge on external validity. The final stage of investigation, FE research, is thus exclusively concerned with the effects of a treatment in usual clinical settings.

3.3.4 *Field Effectiveness Trials*

FE research is interested in the applicability, generalisability, and applied impact of treatment techniques in practical and non-controlled settings (Strosahl, Hayes, Bergan, & Romano, 1998). This approach tends to follow powered RCTs, but only for pragmatic purposes (RCTs justify dissemination to standard care). Quasi-experimental and non-experimental designs are most suited to this stage, but FE work is better characterised by the following features: large, unrestricted and heterogeneous samples, active patient-driven selection of treatments, non-restricted termination of therapy, naturally correcting therapeutic procedures, and regular assessment of well-being (Seligman, 1995). An example, described below, helps to convey the main focus of this approach.

Stroshal et al. (1998) used an effectiveness trial to test whether clinicians trained in ACT obtained better outcomes than control clinicians who did not receive training. Stroshal et al. offered voluntary ACT training to clinical trainees working in an applied setting. Those who volunteered formed the experimental group and those who did not formed the control group. Before training occurred, a baseline measure of clinician effectiveness was obtained. This was achieved by assessing each patient seen by either the experimental or the control clinician before and after (naturally terminating) therapy. After volunteering therapists had received one year's ACT training, baseline procedures were repeated; the patients of both groups of clinicians were assessed at intake and re-assessed five months later. This trial showed that ACT trained clinicians obtained significantly better clinical outcomes following training as compared to non-trained counterparts. Although it is hard to ascertain what affected treatment gains, because of confounds such as non-randomised assignment to training and non-manualised treatments, the merit of this study is its ability to document whether ACT actually worked in real life conditions.

3.4 Statistical Analyses Used in Clinical Research

This final section now deals specifically with statistical techniques for analysing clinical research. Although statistical analysis is closely linked to research design, these topics and some of their complications are dealt with separately. Experimental and quasi-experimental research is a form of hypothesis testing based on between-group comparisons. These designs usually rely on analysis of variance and covariance (ANOVA and ANCOVA, respectively), but calculating individual change (e.g., clinical significance of change) is another valued approach. Although useful for quantifying change, research is also interested in more detailed information, such as mechanisms of change. Regression-based analytic techniques are most suited to this, as well as to testing complex theoretical models. For example, SEM has been developed to explore large and complex multivariate relations. These analytic techniques are described below.

3.4.1 *Group Comparisons*

ANOVA, a family of statistical techniques, tests for significant differences between two or more group means. This is called an analysis of *variance* (rather than of means) because the null hypothesis of equal means is based on the statistical significance of the ratio between within-group (unexplained) and between-group (explained) variability. If the null is true, variance estimates based on within-group and between-group variability should be comparable (Tabachnick & Fidell, 2001). ANOVA makes three main assumptions: (1) that the variances of each population are the same (*homogeneity of variance*), (2) that scores are normally distributed around their mean (*distribution normality*), and (3) that observations are independent from one another (*independence of observation*). Because clinical trials often take multiple observations from the same individual (e.g., at baseline and following treatment), the third of these assumptions is often violated. *Repeated measures* ANOVA is thus a useful variant of the model that estimates and removes variance attributable to dependence imposed by repeated measures. Other useful variants include ANCOVA and multivariate-analysis of variance (MANOVA) and covariance (MANCOVA). ANCOVA allows one to test for between-group differences whilst partialling out variance attributed to covariate(s). MANOVA

and MANCOVA mirror ANOVA and ANCOVA, but simultaneously compare groups across multiple DVs.

Complications can arise when using ANOVA to evaluate a treatment. The most common complication arises from selective attrition; unequal drop-out across conditions. This is problematic because analyses based on those participants completing treatment (*analysis per protocol*) cannot assume between-group equivalence at baseline (nor do sample sizes remain equivalent). Thus, although the most logical way to evaluate a treatment is to assess its effects on those patients who received it, an alternative *intention-to-treat* (ITT) procedure is also commonly used (Altman et al., 2001). ITT analysis uses the data of *all* participants initially randomised to the trial regardless of whether they subsequently received treatment. This upholds randomisation, but because those discontinuing treatment seldom complete post-treatment measures, this approach is complicated by missing data. *Last observation carried forward* (LOCF) is a common method for dealing with this missing data (Spokas et al., 2008). LOCF uses the participant's last observation as an estimate of their missing observation and is thus based on an assumption of no change. This assumption is not always true, however. Furthermore, because LOCF artificially inflates the degrees of freedom, it increases the probability of Type I error. Other more complex approaches such as the Expectation-Maximization (EM) algorithm and the mixed linear model (MLM) compute estimates of missing data based on the observed sample data. This approach can also be problematic, however, because it assumes that those discontinuing treatment are not qualitatively different from those who continue treatment. Dealing with selective attrition is thus an important, and often unavoidable, complication when evaluating clinical trials.

3.4.2 *Clinical Significance of Change*

The analysis of between-group differences is useful for summarising group means and how they may differ, but it is often criticised for insensitivity to individual change. The clinical significance of change has been developed for this purpose, designed to quantify whether the magnitude of change, per individual, is sufficiently large to be meaningful and reliable. Thomas and Truax's (2008; see also Jacobson & Truax, 1991) method for determining clinical significance is based on two criteria: "(1) the amount of

change is large enough that it is unlikely to be due to measurement error (*reliable change*), and (2) the post-treatment level of functioning is closer to the non-clinical population than the clinical population" (crossing the "*cut-off point*"; p. 319).

The reliable change index (RCI) tests the first criterion, examining whether the magnitude of change is greater than that expected from random error in the measurement tool. This value is computed by dividing the pre-treatment to post-treatment difference score by the standard error of the difference score ($RCI = X_2 - X_1 / S_{diff}$ ($S_{diff} = \sqrt{2}(SE)^2$)). Crossing the cut-off point (i.e., criterion 2) can be defined one of three ways: (a) two standard deviations from the clinical population mean (in the direction of functionality), (b) two standard deviations from the non-clinical population mean (in the direction of dysfunctionality) or (c) half way between the two means. Jacobson and Truax (1991) recommend the third of these options as the least arbitrary. Thomas & Truax (2008) and Jacobson, Roberts, Burns, and McGlinchey (1999) have proposed four categories of change: (a) *recovered,* the patient meets both criteria; (b) *improved,* the patient shows a significant RCI without moving into the non-clinical range; (c) *same,* the patient does not meet either criteria; and (d) *deteriorated,* the patient shows a reliable worsening of symptoms.

3.4.3 *Regression Analyses*

In addition to quantifying change, clinical research is also interested in understanding more complex multivariate relations such as mediation and moderation. Regression analysis is useful for these purposes (Aiken, West, & Taylor, 2008). Regression is a family of statistical techniques that aim to summarise the dependence of one variable (the criterion) upon another (the predictor). *Simple* regression predicts a criterion from one predictor and *multiple* regression (MR) from a set of predictors. Regression analysis fits a predictive model to sample data and uses that model to predict values of the criterion. In the regression equation, these values are a function of the predictor(s), a constant, and an error term (unexplained variance). This process assigns a weight, called a *partial regression coefficient*, to each of the predictors, which can be tested for statistical significance. In practical terms, regression coefficients summarise how much unique variance in the criterion each predictor accounts for (Tabachnick & Fidell, 2001).

One common use of MR in treatment evaluation research is to test for mediation. The principle of mediation is to test whether a treatment (IV) affects a change in symptoms (DV) only indirectly and through its effect on an intervening variable (a mediator; M). For example, does ACT effect a change in symptoms because it reduces experiential avoidance? Baron and Kenny (1986) proposed four conditions that, when met, would strongly support inferences regarding mediation. These conditions, which are typically tested using a series of four regressions, state that: (1) IV correlates with DV, (2) IV correlates with M, (3) M correlates with DV, and (4) the effect of IV on DV, controlling for M, is not significantly different from zero. An important limitation of this traditional approach, however, is that the critical mediated (or indirect) path is not itself tested, rather the IV to M and M to DV paths are tested separately. Sobel's (1982) test for indirect effects is thus usually added as a fifth step, which tests whether the association between the IV and DV is significantly reduced when controlling for M. Full mediation is thus implied if, controlling for M, the IV to DV relationship is significantly reduced and is not significantly different from 0. Partial mediation is inferred if, controlling for M, the IV to DV relationship is significantly reduced but is significantly different from 0. Although some researchers have advanced this classic model and proposed different analysis strategies for testing it (e.g., SEM, bootstrapping), the fundamental premise remains. Furthermore, it is currently used in various research designs, such as experimental and cross-sectional design (Aiken et al., 2008).

Although MR is a useful tool for testing multivariate relations, it is gradually becoming superseded by a more complex yet flexible analysis strategy; structural equation modelling (SEM). One of the main reasons for this is that SEM can *simultaneously* test multiple multivariate relations. For example, whereas MR tests for mediation using a series of independent tests, SEM is unique in its ability to test all paths concurrently (Byrne, 2001).

3.4.4 *Structural Equation Modelling (SEM)*

SEM is a family of statistical techniques that test integrated, and often complex, multivariate relations. SEM begins with the specification of a theoretical model using *path diagrams*. These visually represent hypothesised relations between variables (Figure 3.2). Drawn using computer packages (e.g., AMOS), path diagrams electronically synchronise with datasets (e.g., SPSS), thus allowing one to test the model using the data. In SEM, variables are latent (depicted using ellipses). This means they are not measured directly; rather, they are comprised of the shared variance among a set of *manifest indicators* such as questionnaire items or observations (depicted using rectangles). Furthermore, latent variables can be specified either as predictors (*exogenous*) or criterion (*endogenous*) variables, using single-headed arrows to denote uni-directional paths and double-headed arrows to denote bi-directional paths. Finally, a path can either be fixed to a constant (have a pre-assigned value) or be freely estimated. To be 'freely estimated' means that SEM uses the covariance matrix of the sample to determine a regression coefficient for that path.

Two main models are considered here. *Measurement models* test theory driven relations between manifest indicators, latent variables (often called factors for consistency with factor analysis), and inter-factor relations. *Structural models* (e.g., Figure 3.2) test theory driven associations between latent variables only. (An example of each is provided later in section 3.4.4). Before a model can be tested it must be *identified*. This means that the amount of known information in the sample data must exceed unknown information in the model (Byrne, 2001). In order for this criterion to be satisfied, one manifest indicator per latent variable must be fixed to a constant. This fixed value allows for all other values to be freely estimated. Additionally, all manifest indicators must have an associated error term ('e') and all endogenous variables an associated disturbance term ('d'). Error and disturbance terms denote residual variances (per variable) that are not accounted for by paths in the model.

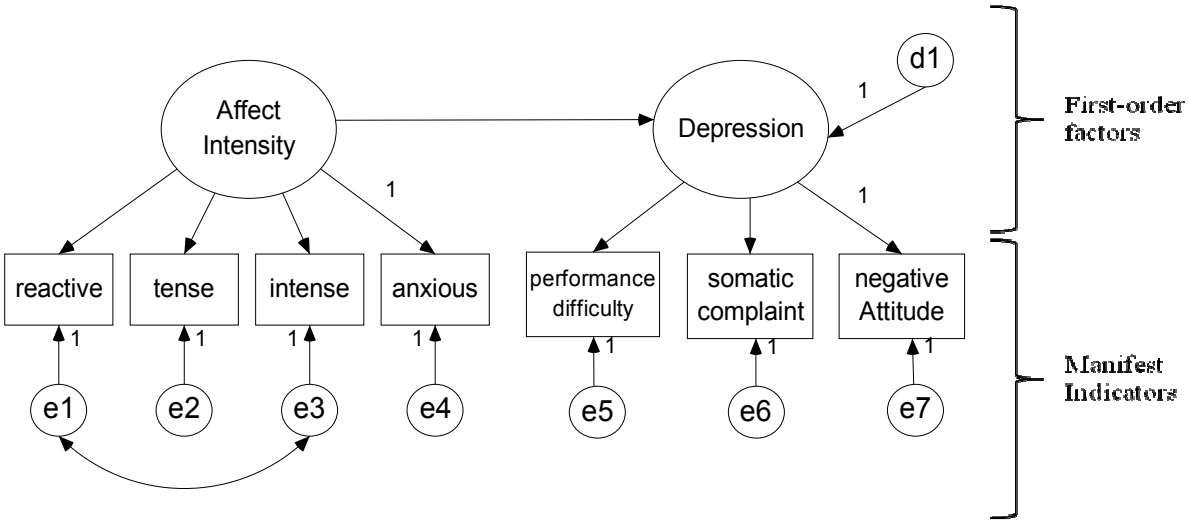


Figure 3.2 Exemplar Structural Model for Hypothesised relations between Affect Intensity (Exogenous variable) and Depression (Endogenous variable).

Note: 'd' denotes disturbance term, 'e' denotes error terms. Rectangles represent manifest indicators (questionnaire items), ellipses represent latent variables.

Once a model has been specified, statistical analysis programmes (e.g., AMOS, LISREL) are used to evaluate how well relations specified in the model are reflected in the sample data. The process of *fitting* the model begins by computing estimated regression values for freely estimated paths. This is done using an iterative process that minimises the difference between the implied (model) and observed (data) covariance matrix. The final parameters thus represent the most accurate estimates that can be made, given the model specified and the sample matrix. Once these values have been estimated, an evaluation is made of the extent to which the integrated model is reproduced in the observed covariances of the sample matrix.

The chi-square test statistic (χ^2) is the primary indicator of model fit. Because χ^2 has a monotonic relationship with sample size, however, Type I error is high in large samples (Byrne, 2001). Several additional goodness-of-fit indices have thus been developed to describe model-data congruence in less sample-size-dependent ways. Typically these are interpreted collaboratively, but there is little consensus in the literature as to which set of indices best discriminates a well-fitting model. One set, designed to reduce Type I and II error, is proposed by Hu and Bentler (1999). This includes the normed chi-square (NC), comparative fit index (CFI), and root-mean-square error of approximation (RMSEA; see Byrne, 2001). The NC is a proxy measure which divides χ^2 by the degrees

of freedom (values of 2.00 indicate adequate fit (Bollen, 1989; Tanaka, 1993)). The CFI is a normed (zero-one) index that compares the specified model to a baseline model that assumes no inter-variable relations (values of .90 - .95 indicate acceptable fit and ≥ .95 indicate good fit; Bentler, 1990). The RMSEA is also normed and calculates the discrepancy between observed and predicted covariances (values of ≤ .05 indicates good fit, .08-.10 average fit, and ≥ .1 inadequate fit; see Byrne, 2001). Finally, parsimony is an important goal of any well-defined model. The Parsimony Adjusted CFI (PCFI) is a non-normed index that penalizes models for lack of parsimony.

If a model obtains inadequate fit statistics it may be rejected, but more often than not a *model generating approach* is adopted (Joreskog, 1993). This aims to 'discover' a model that exhibits better congruence with the sample matrix *and* continues to make theoretical sense (Byrne, 2001). This involves re-specifying the model. This process begins by identifying sources of mis-specification, indicated by Modification Indices (MIs) and regression weights. MIs indicate paths that could be added to the model to improve fit, and regression estimates indicate redundant or non-significant paths that add no predictive value to the model. Re-specification is useful because it can guide model development. Because it is data-driven, however, changes may capitalise on chance. Modifications should thus only be made when they are consistent with theory and should be re-tested in a separate sample. To consolidate this discussion, two examples are described below.

3.4.4.1 *Example One.* The first example involves a type of measurement model called the Higher Order Factor (HOF) model. The HOF model is a hierarchical model which tests whether the covariation between *first-order* factors (endogenous latent variables) can be accounted for by their common association with a single HOF (exogenous latent variable). For example, can the relationships between factors in a questionnaire (first-order factors) be accounted for by their common association with a single underlying construct (a HOF)? This is tested by specifying a model that has no direct paths between first-order factors; rather, these factors are indirectly associated via their common relationship with the HOF (Figure 3.3). If adequate fit is found, it can be inferred that a common HOF efficiently explains factor-factor covariances.

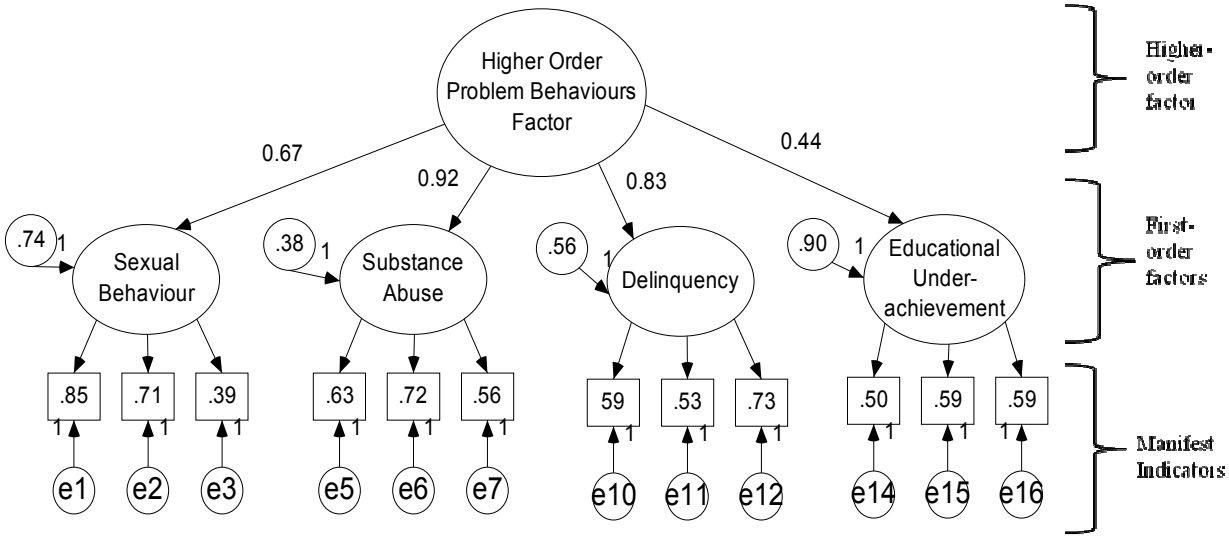


Figure 3.3 Higher Order Factor Model (standardised regression values reported).

From "Personality and the Predisposition to Engage in Risky or Problem Behaviours During Adolescence," by M. Cooper, P. Wood, H. Orcutt, and A. Albin, 2003, *Journal of Personality and Social Psychology*, *84,* p. 398. Note: For ease of communication, beta values for paths from manifest indicators to latent variables are denoted in the manifest indicator boxes.

Several researchers have used the HOF model to test whether a common factor underlies dissimilar risk behaviours (Cooper, Wood, Orcutt, & Albino, 2003; Donovan & Jessor, 1985; McGee & Newcombe, 1992). For example, Cooper et al. (see Figure 3.3) tested whether a single HOF could account for covariation between four delinquent behaviours in an adolescent sample. The authors found acceptable fit: although χ^2 and χ^2/df values were large ($\chi^2_{(50)} = 373.9$, $p < .001$; $\chi^2/df = 7.48$), other indices were acceptable (CFI = .94; RMSEA = .058 (± .052, .063)). Furthermore, all first-order factors significantly loaded on the HOF and the HOF accounted for between 20% and 86% of first-order factor variance. In an attempt to elucidate the nature of the HOF, authors further tested how well four exogenous variables (impulsivity, negative affect, sensation seeking and avoidant coping) predicted this covariation. They found that avoidant coping was its strongest predictor ($\beta = .34$, $p < .001$). Cooper et al. concluded that, despite the apparent dissimilarity of behaviours measured, avoidant coping was a shared causal factor. This interpretation should be treated with caution, however, because the data were cross-sectional. A less assuming interpretation is that behaviour

covariation was effectively represented by one HOF and that avoidant coping significantly predicted a proportion of that covariance.

3.4.4.2 *Example Two.* The second example uses a structural model to test for mediation. In this example, Lynch, Cheavens, Morse, and Rosenthal (2004), using a cross-sectional design and self-report measures, tested whether emotional inhibition mediated the relationship between negative affect intensity (predictor) and depression (criterion). To test for mediation, two models were statistically compared. The first specified that the relationship between predictor and criterion was only indirect (i.e., mediated by emotional inhibition), whereas the second included an additional direct path from predictor to criterion. The second model thus proposed both direct and indirect effects (see Figure 3.4). In this case, mediation was tested by computing a χ^2 difference test ($\Delta \chi^2$), which subtracts the χ^2 and *df* values of one model from the other, and tests whether that difference is statistically significant. If the addition of the direct path (i.e., from predictor to criterion) does not significantly improve fit, full mediation is implied. If, however, the direct path does significantly improve model fit, partial mediation is implied. Lynch et al. found evidence for full mediation. Thus, although cross-sectional in design, the study produced findings that were consistent with the prediction that vulnerability towards heightened negative affect is associated with depression because it fosters heightened emotional inhibition.

In summary: this chapter has aimed to provide a thorough but concise review of research methods and statistical techniques in common use in developing and validating novel clinical treatment. This review has aimed to show that there are a variety of techniques available for testing the theoretical underpinning and clinical effects of a psychological intervention. Each has several associated strengths and weaknesses and thus the process of designing research will often involve a "creative compromise" (Shapiro, 1996). The final section will now provide an overview of the application of some of these techniques to the aims of the current thesis.

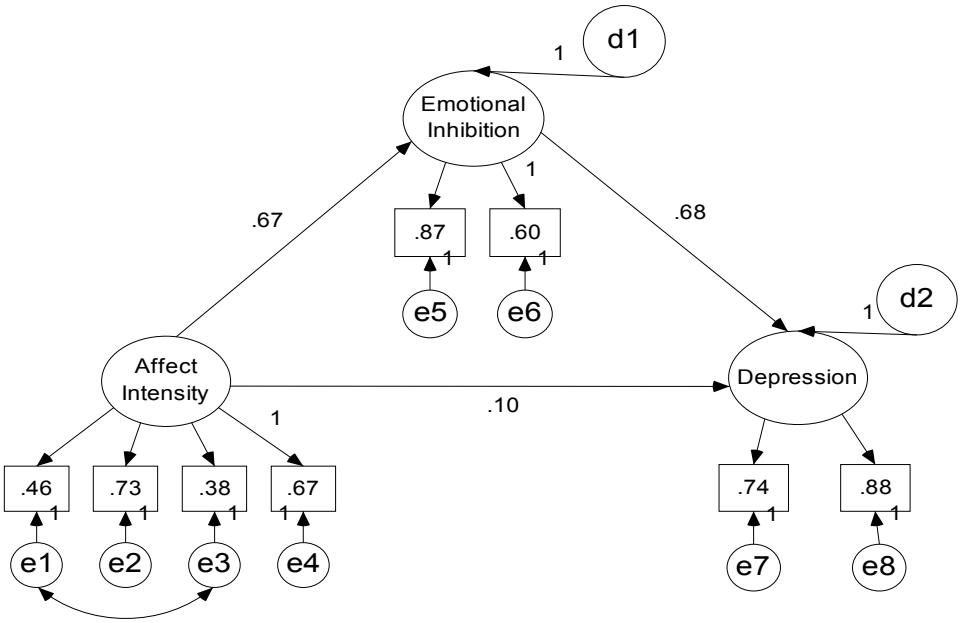


Figure 3.4 Structural Model Proposing that Emotional Inhibition Mediates the Relationship between Negative Affect Intensity and Depression (standardised regression weights reported).

From "A Model Predicting Suicidal Ideation And Hopelessness In Depressed Older Adults: The Impact of Emotion Inhibition and Affect Intensity," by T. Lynch, J. Cheavens, J. Morse, and Z. Rosenthal, 2004, *Aging and Mental Health, 8,* p. 486-497. Note: For ease of communication, beta values for paths from manifest indicators to latent variables are denoted in the manifest indicator boxes.

3.5 The Present Thesis

Informed by discussions raised in the past three chapters, the empirical work of this thesis was designed to pioneer novel investigations into ACT for treatment resistant groups. Theoretically-orientated and clinically-orientated approaches were used. Before embarking on the novel application of ACT for this group, it was first important to gather empirical evidence, based on ACT theorising, that supported such an application. As discussed in chapter 2 (and 6), some research was already available (e.g., Chapman et al., 2005; Gratz & Gunderson, 2006). There was a noticeable gap, however, in understanding the role of experiential avoidance in co-occurring maladaptive behaviours (e.g., dysfunctional eating, DSH and substance abuse). Engagement in co-morbid maladaptive behaviours is common in many, but by no means all, treatment

resistant patients. Addressing this gap was the primary focus of studies 1 and 2. Study 1 employed SEM to test Hayes et al.'s (1996) central theoretical prediction that experiential avoidance underlies several topographically dissimilar, maladaptive behaviours. To obtain a more detailed understanding of this theoretical proposition, study 2 extended the complexity of the model by testing whether known risk factors for maladaptive behaviours would affect behavioural engagement only indirectly and through experiential avoidance. Such mediation-based investigations are central to substantiating the theoretical principles underlying applied work. This is because they can help to elucidate whether and how the processes addressed in therapy relate to surrogate or real outcomes.

Studies 3 and 4 extended these theoretically-orientated investigations by piloting the novel, group-based application of ACT to a heterogeneous group of treatment resistant patients. Because ACT had not previously been delivered to this patient group or trialled on a UK clinical sample, a cautious approach was considered most appropriate. Study 3 recruited a small sample to a pre-post uncontrolled trial. Effects of the intervention were assessed before and after treatment, and at 6-month and 12-month follow-up. Study 4 was designed to evaluate ACT using more rigorous control procedures. In this study, a pilot RCT was used to evaluate the effects of ACT relative to an ecologically valid, active comparison; Cognitive Behaviour Therapy-based Treatment as Usual (CBT-TAU). Both trials made preliminary investigations into mechanisms of change and both strived to achieve acceptable levels of external and internal validity. Together, this systematic and integrated programme of research was designed to advance theoretical and applied knowledge regarding ACT for treatment resistant groups.

CHAPTER IV

Study 1. Experiential avoidance and Maladaptive Behaviours Part I: Developing, Validating, and Testing the Structure of a Maladaptive Behaviours Questionnaire

4.1 Introduction

'Treatment resistant' is a term used within the literature to describe patients whose symptoms are resistant to standard psychological and/or pharmacological intervention (e.g., see Amsterdam et al., 2001; Kenny & Williams, 2007). These patients are heterogeneous with regard to symptomatic complainant, but characteristically exhibit a range of deficiencies in emotional, interpersonal, cognitive, and behavioural functioning (Strosahl, 2005). Trends in the literature suggest that treatment resistant patients have higher symptomatology at baseline, co-morbid mood and/or personality disordered symptoms, and often engage in *maladaptive behaviours* (MBs). MBs can be defined as behaviours that interfere with everyday functioning, that are potentially damaging to the self or others, that are socially defined as a problem, and that usually elicit some form of social control response[11]. These include, for example, substance abuse, deliberate self-harm (DSH), and dysfunctional eating (e.g., Grant, Stinson, Dawson, Chou, Ruan, et al., 2004; Grant, Stinson, Dawson, Chou, Dufour, et al. 2004; Nurnberg, Rifkin, & Doddi, 1993; O'Brien & Vincent, 2003; Sansone, Levitt, & Sansone, 2005; Thomas, Melchert, & Banken, 1999; Lacey & Evans, 1986). The primary aim of this study was to test whether experiential avoidance predicts the tendency to engage in multiple MBs.

4.1.1 *An ACT Conceptualisation of Maladaptive Behaviours*

The syndrome-based classification of clinical disorders (e.g., DSM-IV-TR, 2000), currently the most widely used diagnostic system, conceptualises MBs topographically and therefore distinct from one another. This has lead to a number of models proposing that MBs emanate from separate psychosocial motivations, thus requiring separate explanations, measurement tools, and treatment programmes (McGee & Newcombe, 1990). For example, Anorexia Nervosa (AN) and Substance Dependeny (SD) are

[11] This definition is based on Jessor, Donovan, and Costa's (1991) definition of problem behaviours (p. 24), which is used more specifically to describe adolescent-specific risk taking.

differentiated diagnostically, resulting in the development of assessment batteries and therapeutic interventions that treat these behaviours largely as independent entities.

In contrast to this syndromic approach, ACT theorists propose a more parsimonious, *functional* interpretation of MBs. From this perspective, despite their formal dissimilarity, MBs constitute a functional response class whose shared function is the capacity to prevent, escape, or reduce contact with negatively reinforcing private events (Blackledge & Hayes 2001; Hayes et al., 1996; Gratz, 2006). Despite their deleterious long term consequences, these behaviours are maintained primarily because, in the short term, this *experiential avoidance* function is fulfilled.

Consistent with this theorising, several MBs have clear avoidance functions. For example, substances such as alcohol and cocaine have direct mood altering effects, can reduce the awareness and potency of negative cues, increase dopamine activity, and alleviate physiological withdrawal (see Sher & Grekin, 2007). Similarly, it has been hypothesised that DSH and binge eating may function as focused distracters, able to shift attention away from negative affect eliciting cues, reduce physiological arousal, and facilitate the regulation of mood (Heatherton & Baumeister, 1991; Linehan, 1993; Chapman, Gratz, & Brown, 2005). Furthermore, researchers have speculated that other less obvious behaviours, such as internet addiction, restrictive eating, and impulsive spending could also have an experiential avoidance function (Li & Chung, 2006; Miltenberger, 2005; Wilson & Roberts, 2002). Although a common experiential avoidance hypothesis has not before been tested, numerous independent studies suggest that a common cause(s) or function(s) may underlie several formally dissimilar MBs.

4.1.2 *Maladaptive Behaviour Commonalities*

One of the most striking findings of research in this area is that MBs commonly co-occur in both the general population and in clinical samples (e.g., see Christo et al., 2003; Greenberg, Lewis, & Dodd, 1999; Gossop, 2001). For example, up to 50% of DSH hospital admissions abuse alcohol (Haw, Hawton, Casey, Bale, & Shepherd, 2005; Merrill, Milker, Owens, & Vale, 1992), approximately 25% of patients diagnosed with an eating disorder engage in DSH (Sansome & Levitt, 2002), rates of substance abuse are significantly elevated in disordered eating populations (e.g., Holderness, Brooks-Gunn, & Warren, 1994; Newman & Gold, 1992), and dysfunctional eating is

significantly related to aggression (Thompson, Wonderlich, Crosby, & Mitchel, 1999). Moreover, reducing the frequency of one type of MB has been found to co-occur with increases in the frequency of others (*behaviour switching;* Donovan, 1988).

In addition to the functional interpretations outlined above, research has also identified similarities on the behavioural, biological, and psychological level of analysis. For example, Griffiths (2005) identified common addictive features in gambling, exercise, and internet use (salience, tolerance, conflict, mood modification, withdrawal and relapse) despite the non-pharmacological nature of these behaviours. Similarly, altered dopaminergic and serotonergic functions have been observed in individuals engaging in substance abuse (Di Chiara, 1995), disordered eating (see Kaye, et al., 2005; Kuikka, et al., 2001), and excessive exercise (see Adams & Kirkby, 2002). Finally, on the psychological level, MBs appear to share common associations with trait-based constructs such as impulsivity and avoidant coping (e.g., see Anderson, Simmons, Martens, Ferrier, & Sheehy, 2006; Cooper et al., 2003; Dawe & Loxton, 2004; Grano, Virtanen, Vahtera, Elovainio, & Kivimaki, 2004). These observations further suggest that MBs may share a common cause(s) or function(s).

4.1.3 *SEM and Maladaptive Behaviours*

Chapter 3 described how SEM could be used to investigate whether common mechanisms underlie diverse problem behaviours. Specifically, the HOF model was identified as a means of testing whether the covariation between behaviours can be accounted for by one single HOF. By employing these methods for the investigation of small clusters of adolescent-specific risk behaviours, several independent researchers have obtained data in support of the 'common cause' hypothesis. For example, McGee and Newcombe (1992) and Donovan and Jessor (1985) both found good fit statistics for a HOF model. The former study measured drug use, academic orientation, social conformity and criminal behaviour; the latter measured alcohol use, drug use, delinquency and promiscuous sex.

More recently, Cooper et al. (2003) extended this work in an attempt to identify predictors of risk behaviour covariance (see section 3.4.4.1; sexual behaviours, substance abuse, delinquency and educational underachievement). They reported that Avoidant Coping, a composite of aggressive expression and avoidant coping, was the

strongest predictor of HOF variance. This work was limited on two accounts, however. Firstly, the authors adopted an inductive, rather than theory-testing approach. Secondly, the use of an aggression-based avoidance measure may have confounded findings, because aggression is known to co-vary with other MBs (e.g., Thompson, 1999). The present study aimed to extend this work by testing the ACT-derived hypothesis that experiential avoidance accounts for a significant proportion of MB covariance. A broad and clinically relevant range of MBs were considered: DSH, sexual promiscuity, excessive exercise, restrictive eating, binge eating, excessive internet use, impulsive spending, smoking nicotine, excessive alcohol use, drug use, and aggression.

4.1.4 *Methodological Considerations*

MBs are difficult to research ethically in-situ, and this is especially the case when investigating *multiple* MBs. Researchers have overcome this by using self-report methods (e.g., Cooper et al., 2003). These provide an explicit means of measuring multiple constructs, ethically and anonymously. Although many inventories exist to measure different behavioural topographies in isolation, no truly wide-ranging measure of MBs currently exists. Because of this, the current study began with the development and validation of one such measure (see section 4.2.3.1).

In terms of study design, a cross-sectional approach was selected as most appropriate for the validation of a new measure. This is because it offers a resource unintensive method of collecting large amounts of data. This is necessary for scale validation because factor analysis and SEM are large sample techniques. Furthermore, I also considered the recruitment of a large community sample as most appropriate for the aims of the study. Large clinical samples are notoriously hard to recruit and, from an ethical perspective, they are not suitable for the piloting of new measures. The use of a community sample was supported by evidence suggesting phenomenological continuity across general population and clinical groups for a range of MBs. For example, the co-morbidity of dysfunctional eating and substance abuse is strikingly similar in student and inpatient samples; 35% of student problem drinkers report dysfunctional eating and 40% of eating disordered inpatients abuse substances (see Christo et al., 2003). Also, many correlates, predictors, and maintenance factors are common to both samples (e.g., dysfunctional eating (McFarlane, McCabe, Jarry, Olmsted, & Polivy, 2001), DSH

(Gratz, Conrad, & Roemer, 2002), substance use/misuse (Cooper, et al., 2003; Dawe & Loxton, 2004; Moeller & Dougherty, 2002)). Despite having good justification for the use of a non-clinical sample, efforts were made to obtain preliminary information on the scale's ability to discriminate between self-declared clinical and non-clinical participants (see section 4.2.1).

4.1.5 *Synopsis of the Present Study*

A review of existing literature revealed that few validated inventories exist to measure MBs concurrently[12]. Before investigating patterns of covariation, therefore, it was first necessary to develop and validate a composite Maladaptive Behaviours Questionnaire (MBQ). On the basis of clinical experience and research evidence, I designed the MBQ to measure DSH, sexual promiscuity, excessive exercise, restrictive eating, binge eating, excessive internet use, impulsive spending, smoking nicotine, excessive alcohol use, drug use, and aggression. In constructing such a scale, I deliberately avoided confounding the behavioral measure itself with possible predictor variables, such as experiential avoidance and impulsivity. Items thus aimed to measure one's tendency to engage in certain behaviours rather than motivations to act. Having tested the psychometric properties of the MBQ (e.g., validity and reliability), confirmatory factor analysis (CFA) SEM was used to test whether a HOF model could adequately account for behaviour covariances. If acceptable fit were to be found, a further model would be specified to test whether experiential avoidance predicted a significant proportion of HOF variance.

4.2 Method

4.2.1 *Design*

A cross-sectional design was used. The construct validity of the MBQ was assessed by evaluating its convergence with trait impulsivity (positive correlation predicted), satisfaction with life (negative correlation predicted), and Borderline Personality

[12] To my knowledge, The PROMIS (Christo, et al., 2003) is the only existing scale that approximates a composite measure of maladaptive behaviours. This scale was designed, however, to measure *attitudes* towards and *motivations* to engage in *addictive* behaviours. Consequently, many items confounded engagement with motivation to engage (e.g., "I have used alcohol as both a comfort and a strength"). Furthermore, many behaviours of interest were not measured by this scale.

Disorder (BPD; positive correlation predicted). The construct validity of MBQ subscales was assessed by evaluating their convergence with well-validated scales designed to measure each of the behaviours in isolation (see section 4.2.2). The MBQ's ability to discriminate between clinical and non-clinical samples (concurrent validity) was assessed by comparing the scores of participants who had received treatment for a psychological disorder versus those who had not (see section 4.2.2). Finally, internal consistency and test retest reliability (2-week, 2-4, and 8-14 months) were also assessed.

4.2.2 *Participants*

An opportunity sample (N = 722), consisting of University of Southampton students (N = 423) and members of general public (N = 299), was recruited using advertisements posted on the World Wide Web (www) and on the University's Psychology Intranet. This sample completed all questionnaires (except the measure of BPD, see section 4.2.3) and their data were used for exploratory factor analysis (EFA), construct and concurrent validity, and confirmatory factor analysis (CFA). This sample was asked to indicate whether they were currently, or had in the past, received therapy for a psychological problem (see section 4.2.3). Those indicating 'yes' are referred to as the 'self-declared clinical sample' (N = 183) and those indicating 'no' are referred to as the 'non-clinical sample' (N = 539). A second sample (N = 42) was recruited from the Dorset Healthcare Foundation Trust (DHFT) waiting list for psychological treatment[13]. This 'DHFT clinical sample' completed the MBQ, the Million-III (BPD subscale) and the AAQ (see section 4.2.3) as part of baseline assessment for participation in study 4 of this thesis. This sample was used to assess the MBQ's convergent validity with BPD symptoms and, in conjunction with the opportunity sample, to assess the MBQ's concurrent validity (that is, the scale's ability to discriminate between clinical and non-clinical samples). Table 4.1 reports demographic statistics for these samples.

[13] This sample completed the MBQ *after* it had been piloted on the large opportunity sample.

Table 4.1

Demographics Statistics Split by 'Clinical Status'

Demographics	Opportunity Sample		DHFT Clinical Sample
	Non-clinical (N = 539)	Self-Declared Clinical (N =183)	(N = 42)
Mean Age (SD)	21.0 (5.0)	25.53 (10.2)	44.1 (14.2)
Gender (% Female)	78%	88%	60%
Occupation (%):			
Student Psychology	75%	53%	0%
Student Other	16%	26%	9%
Employed	7%	16.5%	42%
Unemployed	2%	4.5%	49%
Country of Origin (%):			
England	83%	67.9%	86%
USA	6%	21.4%	0%
Europe (other)	6%	6.2%	0%
Asia	3%	2.2%	0%
Other	2%	2%	14%
Mean no. of therapies (range)	0	1.78 (1-8)	2.50 (1-6)
Mean no. of sessions (range)	0	40.64 (2-385)	36.55 (10-75)

Note. 'Non-clinical' refers to those participants who indicated that they had never sought help for a psychological problem. 'Self-declared clinical' refers to members of the opportunity sample who reported that they were receiving, or had received, therapy for psychological problem. 'DHFT clinical' refers to those participants who were recruited from the DHFT waiting list for therapy.

A third and final sample was recruited for assessing the MBQ's test retest reliability (N = 53; not in Table 4.1). This sample consisted of psychology students recruited using the Psychology Intranet. The mean age was 19 years and 92% were female. The Southampton University Psychology Ethics Committee approved recruitment of the opportunity and student samples, and the Local Research Ethics Council (LREC) of the DHFT approved recruitment of the clinical sample. University of Southampton Psychology students were offered course credits for participating.

4.2.3 Materials

4.2.3.1 *The Maladaptive Behaviour Questionnaire: Construction and Description.*

Eleven behaviours were chosen for inclusion in the MBQ (Excessive Alcohol Use, Illicit Drug Use, Smoking Nicotine, Excessive Exercise, Aggression, Sexual Promiscuity, DSH, Restrictive Eating, Binge Eating, Impulsive Spending and Excessive Internet Use[14]). This selection was made following an extensive literature review (PsychLit search terms: problem behaviour, maladaptive behaviour, at-risk behaviour, risk taking, antisocial behaviour, dysfunctional behaviour, addictive behaviour, and self-defeating behaviour) and consultation with a specialist in entrenched psychological disorders[15].

To generate a scale that was valid, reliable, and specific to each behavioral domain, the MBQ's construction was based on a range of well-validated inventories. Item construction began by aggregating inventories that measured one behavioral topography in isolation and these were used to identify features characteristic of the behaviour being 'problematic' or 'at-risk'. Based on this, an initial set of sixty six questionnaire items were generated (six items per behaviour, two of which were reverse coded). Where possible, and with the permission of all authors, these items were closely derived from the validated inventories. Items were discussed and refined in collaboration with consultant clinicians and doctoral level researchers, before being piloted on a small sample of undergraduate and postgraduate students (N = 30). These students were asked to provide written feedback on their experience completing the questionnaire. Based on this feedback, and on descriptive statistics, items underwent a final stage of refinement.

[14] A Gambling subscale was initially included in the MBQ, but was dropped owing to floor effects.

[15] Professor Susan Clarke (co-supervisor of the PhD)

The response format chosen asked participants to rate how characteristic each behaviour was of them using a scale ranging from (1) "very like me" to (6) "very unlike me". This scale was chosen to ensure that all participants could respond to all questions, even if they had never engaged in the behaviour described. This ensured standardisation across procedures, avoided selection bias, and helped to obtain composite scores that were comparable across participants. To control for order effects, items were randomly distributed throughout the questionnaire.

4.2.3.2 *Demographics*. Participants were asked to indicate age, gender, country of birth and residence, and occupation. They were also asked to indicate whether (a) they had ever attended therapy for a psychological problem (prompts of, "For example depression, anxiety, bereavement, an eating disorder" were used), (b) the number of different types of psychotherapy they had attended, and (c) the approximate number of sessions per type [16] (see Appendix A).

4.2.3.3 *Construct Validation Questionnaires.* The following well-validated questionnaires were used to test the construct validity of MBQ composite and subscales.

The *Alcohol Use Disorders Identification Test* (*AUDIT:* Babor & Grant, 1989). The AUDIT is a10-item scale of harmful drinking that measures consumption, dependence, and harmful consequences arising from drinking behaviour (e.g., "Have you or someone else been injured as an effect of your drinking?"). Although developed in a clinical population, psychometric data supports its sensitivity to student drinking. The scale's reliability and validity has been well reported (e.g., $\alpha = .80$; Fleming, Barry, & MacDonald, 1991).

The *Sociosexual Orientation Inventory* (*SOI;* Simpson & Gangestad, 1991). The SOI is a 7-item measure of attitudes towards and willingness to engage in casual sex (e.g., "With how many partners have you had sex on one and only one occasion?"). This measure has been used both in clinical and non-clinical populations and has evidenced satisfactory psychometric properties (e.g., internal consistency, $\alpha = .77$ (women) and $\alpha = .76$ (men); test retest reliability, $r = .94$, 2-month delay; Simpson & Gangestad).

The *Drug Problem Index* (*DPI;* Simons & Carey, 2002). The DPI is a modified version of Rutgers Alcohol Problem Index (White & Labouvie, 1989), specifically developed to measure illicit drug use in student populations. Items measure the

[16] The use of medicinal treatment was not measured in this study. It was, however, measured in study 2.

frequency of negative consequences following drug use (e.g., "Neglected your responsibilities"), rated on a scale ranging from 0 (never) to 6 (> 50 times). The authors reported good psychometric properties in a student population (e.g., α = .88).

The *Internet Addiction Test* (*IAT;* Young, 1996). The IAT is a 20-item measure of internet addiction. Items assess the addictive qualities of internet use (e.g., "How often do you become defensive or secretive when anyone asks you what you do on-line?") and are rated on a scale ranging from 1 (rarely) to 5 (always). The author has reported adequate internal consistency in a student sample (α = .78).

The *Impulsive Buying Tendency Scale* (*IBTS;* Verplanken & Herabadi, 2001). The IBTS is a 20-item scale measuring cognitive (e.g., absence of deliberation) and affective (e.g., emotionally driven purchases) reasons for impulse buying (e.g., "I usually only buy things that I intend to buy"). Participants rated their agreement with statements using a scale ranging from 1 (strongly agree) to 7 (strongly disagree). The authors reported good internal consistency in an undergraduate sample (α = .86).

The *Modified Fagerström Tolerance Questionnaire* (*MFTQ;* Prokhorov, Pallonen, Fava, Ding, & Niaura, 1996). The MFTQ measures nicotine addiction with specific sensitivity to adolescent populations. Items measure patterns of smoking behaviour (e.g., "How soon after you wake up do you smoke your first cigarette?") and satisfactory psychometric properties have been reported (e.g., 2-month test retest = .71; Prokhorov, Koehly, Pallonen, & Hudmon, 1998).

The *Deliberate Self-Harm Inventory* (*DSHI;* Gratz, 2001). The DSHI measures whether participants engage in any of 16 predetermined forms of DSH (e.g., "Burned myself with a cigarette") using a 'yes/no' response format. Gratz reported good internal consistency (α = .82) and adequate test-retest reliability (*r* = .68, 2-4 week delay).

The *Obligatory Exercise Questionnaire* (*OEQ;* Thompson & Pasman, 1988). The OEQ is a 20-item questionnaire measuring one's personal obligation to exercise (e.g., "When I don't exercise I feel guilty"). Items are rated on a 4-point scale ranging from 1 (never) to 4 (always). Authors reported good internal reliability (α = .94) and adequate 4-week test-retest reliability (*r* = .69).

The *Three Factor Eating Questionnaire* (*FEQ;* Stunkard & Messick, 1985). The FEQ measures three forms of dysfunctional eating. Subscales used in this study included Restraint (e.g., "I deliberately take small helpings as a means of controlling my

weight") and Disinhibition. Items are rated using a 'yes/no' response format. Authors reported good internal reliability in clinical and student samples (e.g., α = .90 and α = .87 respectively).

The *Aggression Questionnaire* (*AG;* Buss & Perry, 1992). The AG is a 29-item measure of physical and verbal aggression, hostility, and anger (e.g., "I have become so mad that I have broken things"). Items are rated on a scale ranging from of 1 (extremely characteristic of me) to 5 (extremely uncharacteristic of me). Adequate internal consistency (α = .77) and test retest reliability values have been reported (*r* = .75, 2-month delay; Harris, 1997).

The *UPPS Impulsivity Scale* (*UPPS;* Whiteside & Lynam, 2001). The UPPS measures four aspects of Impulsivity (Urgency, Premeditation, Perseverance, and Sensation Seeking) using a 'true/false' response format. Only the Urgency subscale was used in this study. This subscale measures the tendency to engage in impulsive behaviours to alleviate negative emotions (e.g., "I have trouble controlling my impulses"). Authors reported that this subscale has shown good internal reliability (α = .89) and construct validity.

Satisfaction with Life (*SWL;* Diener, Emmons, Larsen, & Griffins, 1985). The SWL is a 5-item measure assessing how satisfied the individual is with their life and their desire to change the past (e.g., "So far I have gotten the important things I want in life"). Items are rated on a scale ranging from 1 (strongly agree) to 7 (strongly disagree). Authors reported good internal consistency (α = .87) and test retest reliability (*r* = .82, 2-month delay).

The *Acceptance and Action Questionnaire* (*AAQ;* Hayes, Stroshal, et al., 2004). The AAQ is a 9-item measure of experiential avoidance, with items measuring: the tendency to evaluate, the unwillingness to experience, the desire to control, and the inability to take action when experiencing private events (e.g., "I rarely worry about getting my anxieties, worries and feelings under control"). Items are rated on a scale ranging from 1 (never true) to 7 (always true). Authors reported adequate internal (α = .77) and test retest reliability (*r* = .64, 4-month period).

The *Millon Mutliaxial Clinical Inventory III* (*MMCI-III;* Millon, 1994). The MMCI-III is a175-item questionnaire that measures 14 personality disorders and 10 Axis I syndromes based on DSM-IV classification system. Items are rated using a 'yes/no'

response format. Devised as a diagnostic tool, scores of greater than 85 are indicative of clinical levels of symptomatology and scores of greater than 75 are indicative of trait tendency. Only the BPD subscale was used in this study and only the 'DHFT clinical' sample completed it.

4.2.4 *Procedure*

Opportunity sample participants either completed the full validation pack on-line, or they collected paper questionnaires from outside the experimenter's office and completed them in a place of their choosing. Participants were pre-warned that questions were of a sensitive nature and advised to complete them in private. The order of questionnaires was randomised across participants to control for order effects. To ensure anonymity, participants returning questionnaires to the experimenter's office were asked to seal them in the envelope provided and to deposit them in a secure box. The DHFT clinical sample received questionnaires in the post, completed them in their own time, and returned them to the clinic prior to treatment allocation. The test retest sample received the MBQ twice via email. To ensure completion at pre-specified times, they were asked to highlight their answers and return them electronically. ID numbers were allocated to all participants to ensure anonymity. Owing to the personal nature of the questionnaires, participants were also given the contact details of help organisations (e.g., Samaritans) and the author's email address for questions or comments.

4.2.5 *Analysis Strategy*

Following the recommendations of Mulaik and Millsap (2000) *Phase One* of scale analysis employed EFA with Maximum Likelihood (ML) extraction procedures. EFA is a data-driven analytic technique that allows an iterative assessment of scale structure by imposing few theory driven constraints. This is achieved by reproducing observed relationships among questionnaire items, (initially) without specifying the number of factors to be extracted (Brown, 2005). EFA was thus used to determine, in an unbiased way, the number and nature of latent factors (Brown, 2005; Mulaik & Millsap, 2000). This analysis used the data of the opportunity sample only. Following this, *Phase Two* of analysis assessed the psychometric properties (construct and concurrent validity, internal and test retest reliability) of the composite MBQ and its subscales (factors). Analyses in this phase used the data of all samples. Finally, *Phase Three* used theory-

driven measurement modelling (see section 3.4.4) to test the hypothesised structure of the scale. Specifically, a HOF model was used to test whether a single HOF could efficiently account for MB covariances. If so, a further model was specified to test whether the AAQ was significantly predictive of HOF variance (see Cooper et al., 2003; section 3.4.4.1). This phase of analysis used the data of the opportunity sample only.

4. 3 Results

4.3.1 *Preliminary Statistics*

4.3.1.1 *Missing data and outliers.* Inspection of the raw data indicated that 22 participants (3%) had either (a) > 10% data missing for any one inventory or (b) a succession of missing cells across different inventories. The data of these individuals was excluded from analyses. Following the recommendations of Tabachnick and Fidell (2001), all other missing data were replaced by the group mean. Box plots and Mahalanobis distance values were used to identify univariate and multivariate outliers, following which the data from nine participants were excluded from analyses. This resulted in: self-declared non-clinical sample (N = 521), self-declared clinical sample (N = 168), DHFT clinical sample (N = 42), and test retest reliability sample (N = 53).

4.3.1.2 *Clinical versus Non-Clinical Comparisons.* Consistent with the proposition that the self-declared clinical sample were drawn from a clinical population, MANCOVA (co-varying for country of origin, occupation, and gender) showed that this group scored significantly higher than the non-clinical group on the AAQ ($F_{(2, 684)} = 31.64$, $p < .001$), SwL ($F_{(2, 684)} = 31.77$, $p < .001$), DSHI ($F_{(2, 684)} = 66.48$, $p < .001$), MFTQ ($F_{(2, 684)} = 15.20$, $p < .001$), DPI ($F_{(2, 684)} = 11.32$, $p = .001$), SOI ($F_{(2, 684)} = 10.80$, $p = .001$), AG ($F_{(2, 684)} = 8.69$, $p < .01$), and FEQ (Restraint) ($F_{(2, 684)} = 5.07$, $p < .01$). The non-clinical group, however, scored higher on the AUDIT ($F_{(2, 684)} = 8.28$, $p < .01$).

4.3.2 *Phase One: EFA* (opportunity sample data)

Because behaviour covariation was anticipated, a non-orthogonal extraction procedure (oblique rotation; Direct Oblimin) was used. Delta was set at ‘0’ to allow for “fairly high” inter-factor correlations (Tabachnick & Fidell, 2001). Inventories with a large number of items can be analysed using one of several extraction procedures, all of

which are likely to provide the same or similar solution (Tabachnick & Fidel, 2001). The ML extraction procedure was used for consistency with the CFA.

The first iteration of EFA included all 66 questionnaire items (see Appendix A for the full set of items) and allowed for items to cluster naturally, rather than pre-specifying the number of factors to be extracted. Following the recommendations of Gorsuch (1974), two methods were used to interpret the output. Inspection of the scree-plot implied a multi-component scale with the point of inflection[17] occurring at the 11th factor. These 11 factors accounted for 61.88% of the variance and converged in 20 iterations. Examining eigenvalues > 1[18], however, implied a 14-factor solution accounting for 65.52% of total variance. Inspecting the content of these factors (i.e., item clusters) showed that only the first 11 were meaningful. Factor 12 consisted of one low-loading item (Binge Eating item), factor 13 of a reverse Sexual Promiscuity item, and factor 14 of three low-loading items (two reverse DSH items and one reverse Impulsive Spending item). Based on this information a second EFA was run, excluding these items and specifying an 11 factor solution. This converged in 14 iterations and accounted for 62.66% of total variance.

Factors were next refined by removing low loading and cross-loading items. Comrey and Lee's (1992) criteria for extracting low loading items (items with eigenvalues < 0.55) and Osborne and Costello's (2005) criteria for removing cross-loading items (items loading on two or more factors with $\beta \geq .32$) were used. This resulted in the removal of a further 12 items. The final EFA, run on the remaining 49-items and specifying an 11 item solution, converged in nine iterations, accounted for 68.46% of total variance, and had a $\chi^2_{(692)} = 1482.0$. Factors and Eigenvalues (in parenthesis) were as follows (see Figure 4.1 for the final scree plot and Table 4.2 for item factor loadings): Nicotine Smoking (8.04), DSH (4.29), Excessive Alcohol Use (4.02), Drug Use (3.43), Restrictive Eating (2.77), Impulsive Spending (2.24), Excessive Internet Use (2.00), Binge Eating (1.97), Excessive Exercise (1.81), Sexual Promiscuity (1.53), and Aggression (1.46). Descriptive statistics (Table 4.3) showed that Nicotine Smoking, Drug Use, and DSH were not normally distributed. This was corrected using logarithmic transformations.

[17] The point of inflection is where the slope first deviates from the plateau.
[18] The Kraiser-Guttman rule states that eigenvalues > 1 represent nontrivial factors (see Gorsuch, 1974).

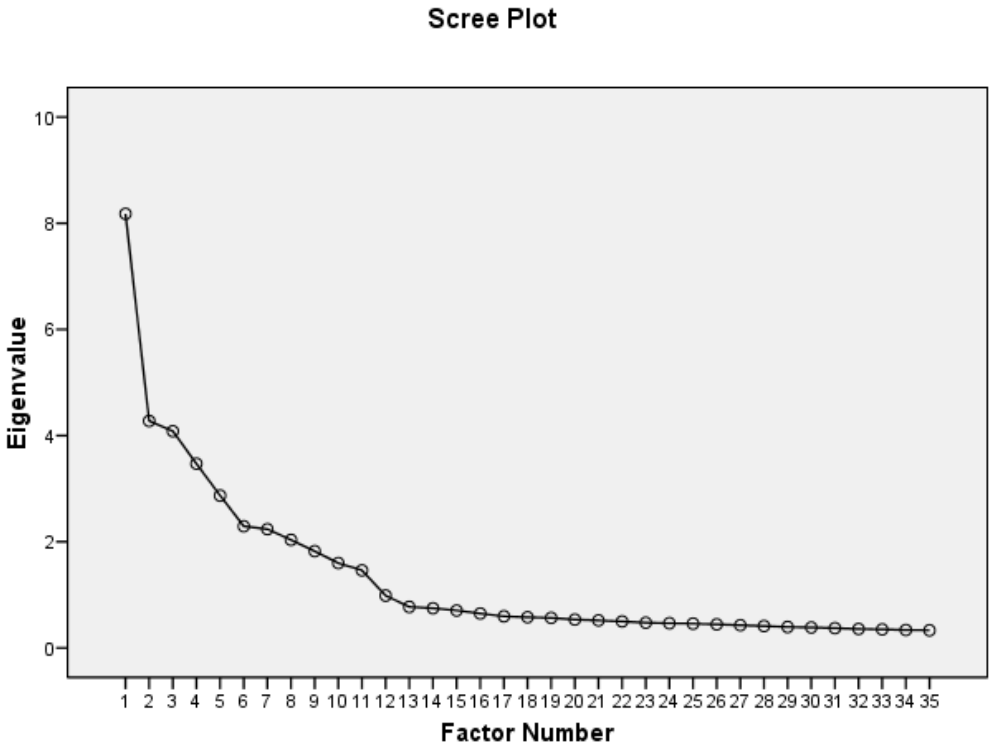


Figure 4.1 Scree Plot for Third Iteration of EFA using ML Extraction

Table 4.2

Factor Loadings for MBQ Eleven Factor Solution

MBQ Subscale items	Factor loading										
It's like me.....:	1	2	3	4	5	6	7	8	9	10	11
Smoking:											
(13) ... to smoke tobacco	.92										
(52) ... to feel the urge to have a cigarette	.90										
(20) ... to feel irritation/frustration if I am in a non-smoking environment	.78										
(4) ... to be pre-occupied by thoughts about smoking when smoking is prohibited	.77										
(29) ... to prefer being in places where smoking is prohibited (R)	.58										
Deliberate Self-Harm:											
(37) … to sometimes cause myself direct bodily harm by, for example, cutting or burning myself		.96									
(62) ... to feel the urge to intentionally harm myself		.90									
(11) ... to sometimes intentionally prevent scars or wounds from healing		.71									
(21) ... to sometimes scratch or bite myself to the point of scarring or bleeding.		.70									
Excessive Alcohol Use:											
(5) ... to sometimes consume more than 6 alcoholic drinks in one evening			.84								
(24) ... to drink a lot more alcohol than I initially intended			.80								
(26) ... to feel excitement and/or tension in anticipation of getting drunk			.68								
(54) ... to go out with friends who are drinking, but opt to stay sober (R)			.66								
(64) ... to sometimes feel that I need an alcoholic drink			.62								
Drug Use:											
(41) ... to be excited by the opportunity of taking drugs, including cannabis				.87							
(19) ... to sometimes actively seek out drugs for personal use, including cannabis				.81							
(3) ... to say no to drugs, including cannabis (R)				.78							
(53) ... to sometimes feel that I need to take drugs (this includes cannabis)				.76							
(15) ... to generally have no interest in taking drugs, including cannabis (R)				.74							
(58) ... to sometimes think that I might have a drugs problem (this includes cannabis)				.67							

Restrictive Eating:					
(32) ... to deliberately take small helpings as a means of controlling my weight	.75				
(59) ... to avoid eating when I am hungry	.74				
(8) ... to ignore dietary details (e.g., calorie content) when choosing something to eat (R)	.64				
(25) ... to have a long list of things that I dare not eat	.63				
(65) ... to sometimes claim I have already eaten when this is not true	.63				
Impulsive Spending:					
(50) ... to sometimes feel a strong impulse to buy things that I don't really need		.90			
(48) ... to sometimes buy things for the sake of it, rather than because I actually need them		.85			
(68) ... to sometimes experience a powerful urge to spend money		.68			
Excessive Internet Use:					
(22) ... to sometimes feel pre-occupied with the internet/computer games			.82		
(17) ... to find that my work performance or productivity suffers because of my internet/video game use			.78		
(14) ... to surf the net/play computer games before doing something else that needs doing			.70		
(39) ... to unsuccessfully try to cut back my use of the internet/computer games			.68		
(51) ... to easily limit my use of the internet or video games (R)			.66		
Binge Eating:					
(28) ... to always stop eating when I feel full (R)				.83	
(38) ... to only eat when I am hungry (R)				.74	
(60) ... to find it difficult to stop eating after certain foods				.62	
(35) ... to sometimes eat to the point of physical discomfort				.62	
Excessive Exercise:					
(33) ... to exercise more than three times a week					.79
(27) ... to be content if I am prevented from exercising for a week (R)					.72
(9) ... to exercise even when I am feeling tired and/or unwell					.69
(36) ... to sometimes feel tension and/or excitement in anticipation of doing exercise					.67
(23) ... to skip doing exercise for no good reason					.65

Sexual Promiscuity:		
(47) ... to sometimes have more than one sexual partner	.85	
(16) ... to sometimes engage in sexual activities with someone I have only just met	.76	
(49) ... to sometimes engage in sexual actives with someone when really I shouldn't	.72	
Aggression:		
(31) ... to control my temper (R)		.70
(18) ... to never resort to violence (R)		.63
(61) ... to be aggressive when sufficiently provoked		.62
(43) ... to sometimes get so angry that I beak something		.58

Note: All factor loadings of below 0.30 are not reported. DSH = Deliberate Self-Harm. Numbers to the right hand side of each item correspond to the version in the Appendix A.

Table 4.3

Descriptive Statistics for MBQ subscales (full opportunity sample)

MBQ	Mean (SD)	Skew	Kurtosis
Composite (49)	2.57 (0.54)	0.58	0.46
DSH (4)	1.67 (1.16)	2.05	3.53
Nicotine Smoking (5)	1.99 (1.33)	1.48	1.07
Excessive Alcohol Use (5)	3.45 (1.32)	-0.27	-0.92
Restrictive Eating (5)	2.74 (1.17)	0.53	-0.34
Aggression (4)	2.13 (0.97)	1.00	0.77
Excessive Internet Use (5)	2.74 (1.22)	0.39	-0.72
Drug Use (6)	1.73 (1.10)	1.81	2.79
Binge Eating (4)	3.19 (1.16)	-0.24	-0.69
Sexual Promiscuity (3)	2.01 (1.21)	1.00	-0.35
Impulsive Spending (3)	3.61 (1.16)	0.19	-0.59
Excessive Exercise (5)	3.11 (1.22)	0.28	-0.59

Note: Bracketed numbers denote number of items per subscale.

4.3.3 *Phase Two: Validity and Reliability*

4.3.3.1 *Construct validity.* Values of convergent validity, reported in Table 4.4, support the construct validity of the MBQ and its subscales. Consistent with prediction, composite scores were significantly, positively associated with the tendency to engage in impulsive behaviours (UPPS Urgency) and significantly negatively correlated with life satisfaction. This latter finding suggests the MBQ successfully tapped problematic, rather than recreational, patterns of behaviour. Furthermore, in the DHFT clinical sample, composite MBQ scores were significantly positively correlated with BPD symptoms ($r = .60$, $p < .001$); a disorder characterised by the engagement in multiple risk behaviours. Testing associations between each subscale and its corresponding inventory further supported the MBQ's construct validity. Eight subscales were multicollinear ($r \geq .70$, Tabachnick & Fidell, 2001) with their corresponding validated scale. This suggests that they measured the same, or similar, constructs. The remaining three (Binge Eating, Restrictive Eating and Sexual Promiscuity), although not multicollinear, were nonetheless significantly correlated with their corresponding inventory.

Table 4.4

MBQ's Construct Validity, Internal Reliability, and Test Retest Reliability.

	VALIDITY				RELIABILITY			
	Construct (*r*)				Test-retest (*r*)			Internal (α)
MBQ	Validated scale	UPPS (Urgency)	SwL	AAQ	2-4 wks (N = 20)	2-4 mths (N = 15)	8-14 mths (N = 19)	
Composite	-	.43*	-.29*	.29*	.97*	.87*	.91*	.87
DSH†	.80*	.29*	-.39*	.40*	.98*	.80*	.92*	.90
Nicotine Smoking †	.90*	.18*	-.18*	.11	.95*	.89*	.86*	.90
Excessive Alcohol Use	.76*	.27*	-.04	.06	.89*	.80*	.80*	.85
Restrictive Eating	.58*	.09	-.07*	.20*	.95*	.91*	.88*	.78
Aggression	75*	.38*	-.23*	.26*	.73*	.69*	.85*	.78
Excessive Internet Use	.70*	.17*	-.20*	.18*	.73*	.74*	.65*	.85
Drug Use†	.71*	.16*	-.19*	.12	.91*	.73*	.78*	.90
Binge Eating	.50*	.29*	-.16*	.18*	.75*	.73*	.81*	.80
Sexual Promiscuity	.56*	.15*	-.06	.02	.95*	.70*	.76*	.83
Impulsive Spending	.74*	.39*	-.15*	.23*	.90*	.57*	.57*	.86
Excessive Exercise	.73*	-.08	.14	-.09	.95*	.87*	.75*	.84

* $p \leq .001$ (adjusted for multiple comparisons). † Correlations based on transformed data. Note: Colum entitled "validated inventory" reports correlations between MBQ subscales and corresponding validated inventory (see methods section). AAQ = Acceptance and Action Questionnaire, UPPS = Impulsivity Scale, SWL = Satisfaction with Life; mths = months. Bracketed values following subscale names denote the number of items per subscale.

Table 4.4 also reports correlations between the AAQ and MBQ, indicating that the two were significantly, moderately associated. This suggests that participants higher in the tendency to avoid unwanted internal experiences were more likely to engage in MBs. Breaking this down into subscales showed differential effects, however. In the full opportunity sample, the AAQ was significantly related to many (e.g., DSH, Aggression, and Impulsive Spending) but not all (e.g., Excessive Alcohol Use, Sexual Promiscuity, and Drug Use) subscales (at the $p \leq .001$ level).

Inter-factor correlations are reported in Table 4.5. These show that, consistent with previous research, many of the subscales were significantly correlated. These correlations were sufficiently low, however, to support subscale specificity. Moreover, low correlations between each subscale and non-corresponding validated inventories (range $r = .08$ to $r = .34$) further suggested that subscales measured related, but differentiable, behaviours. Unexpectedly, Restrictive Eating and Excessive Exercise were not associated with the remaining nine behaviours, however. Furthermore, neither of these subscales was related to the Urgency UPPS subscale, and Excessive Exercise was positively, rather than negatively, related to life satisfaction (Table 4.4). The relationship between these and other subscales was tested in the SEM.

Table 4.5

Inter-factor Correlations (opportunity sample)

MBQ Subscale	2.	3.	4.	5.	6.	7.	8	9.	10.	11.
1. DSH$_{\dagger}$	.22*	.10*	.20*	.30*	.19*	.22*	.20*	.14*	.19*	.06
2. Nicotine Smoking $_{\dagger}$		.24*	.07	.24*	.04	.39*	.05	.28*	.14*	-.13*
3. Alcohol Use			.06	.26*	.20*	.30*	.17*	.40*	.28*	-.03
4. Restrictive Eating				.03	-.02	.08	.12	-.03	.09	.27*
5. Aggression					.21*	.25*	.18*	.24*	.29*	-.04
6. Internet Use						.01	.23*	.19*	.19*	-.02
7. Drug$_{\dagger}$							.07	.01	.07	.08
8. Binge Eating								.10	.28*	.12
9. Sexual Promiscuity									.13*	-.03
10. Impulsive Spending										.09
11. Excessive Exercise										

* $p \leq .001$ (Bonferroni adjustments for 55 tests). $_{\dagger}$ = correlations based on transformed data.

4.3.3.2 *Concurrent Validity.* Preliminary investigations into the MBQ's ability to discriminate across samples was obtained by comparing the scores of the clinical (self-declared clinical plus DHFT clinical sample; N = 210) and non-clinical (participants reporting that they had never received treatment for a psychological disorder; N = 521) subgroups. Comparisons were made using MANCOVA, co-varying for gender, country of origin and occupation. Results, reported in Table 4.6, showed that the clinical sample scored significantly higher on total MBQ scores. Decomposing this across subscales showed that the clinical subgroup scored significantly higher on DSH, Nicotine Smoking, Aggression, and Restrictive Eating subscales. The non-clinical group, however, scored significantly higher on Excessive Alcohol Use. In order to judge these findings relative to a standard benchmark, they can be compared to the between-group comparisons reported in section 4.3.1.2, which compared these two groups using the well-validated measures (e.g., the AUDIT). Results from those analyses suggested further between-group differences for Sexual Promiscuity and Drug Use that were *not* detected by the MBQ.

Table 4.6

MBQ Descriptive Statistics Split by Clinical Status and MANCOVA of Between-Group Differences

	Non-clinical subgroup (N = 521)	Clinical subgroup (N = 210)	Non-clinical vs. Clinical
MBQ	Mean (SD)	Mean (SD)	MANCOVA (*F*)
Composite	2.52 (0.50)	2.65 (0.60)	8.23*
DSH†	1.43 (0.88)	2.25 (1.52)	80.49*
Nicotine Smoking†	1.81 (1.16)	2.43 (1.60)	27.00*
Excessive Alcohol Use	3.59 (1.27)	3.12 (1.40)	19.66*
Restrictive Eating	2.37 (1.07)	2.80 (1.27)	17.64*
Aggression	2.06 (0.91)	2.30 (1.09)	9.50*
Excessive Internet Use	2.80 (1.20)	2.62 (1.26)	3.10
Drug Use†	1.69 (1.07)	1.82 (1.18)	1.57
Binge Eating	3.17 (1.08)	3.25 (1.25)	0.88
Sexual Promiscuity	2.03(1.19)	2.00 (1.07)	0.44
Impulsive Spending	3.62 (1.28)	3.61 (1.42)	0.01
Excessive Exercise	3.10 (1.19)	3.10 (1.02)	0.09

* = $p \leq .01$ (adjusted for 12 comparisons). † Variables displayed in non-transformed state, but independent t-tests computed using transformed data. Note: High values indicate greater behavioural engagement. 'Non-clinical sample' = self-declared non-clinical sample; 'Clinical sample' = self-declared clinical plus DHFT clinical sample.

Finally, correlations between the AAQ and MBQ were computed in the clinical and the non-clinical subgroups (see Table 4.7). Results from this analysis suggested that the AAQ was related to a greater range of behaviours in the clinical than the non-clinical group. Furthermore, the size of those associations tended to be greater in the clinical than non-clinical group. These trends might suggest that experiential avoidance was a greater determinant of MBs in the self-declared clinical sample.

Table 4.7

Correlations between the AAQ and MBQ in the Clinical and Non-clinical Sample

	Non-clinical subgroup (N = 521)	Clinical subgroup (N = 211)
MBQ	AAQ (*r*)	AAQ (*r*)
Composite (49)	.22***	.34***
DSH† (4)	.32***	.40***
Nicotine Smoking † (5)	.04	.11
Excessive Alcohol Use (5)	.06	.17*
Restrictive Eating (5)	.16*	.16*
Aggression (4)	.21**	.21**
Excessive Internet Use (5)	.18**	.20**
Drug Use† (6)	.08	.16*
Binge Eating (4)	.14	.24**
Sexual Promiscuity (3)	.03	.01
Impulsive Spending (3)	.26**	.12
Excessive Exercise (5)	.06	.16*

*$p < .05$, **$p < .01$, *** $p < .001$. Note: Bracketed numbers denote the number of items per subscale. 'Non-clinical' = self-declared non-clinical sample; 'clinical' = self-declared clinical plus DHFT clinical sample.

4.3.3.3 *Reliability.* The MBQ also demonstrated good internal and test retest reliability (Table 4.4). Internal reliability values were all above the widely accepted benchmark of $\alpha = 0.7$ (Tabachnick & Fidell, 2001), thus suggesting that subscale items strongly converged with one another. Furthermore, test retest values, obtained over varying delay periods, indicated that subscales were stable over time.

4.3.4 *Phase Three: Confirmatory Factor Analysis*

Allowing for inter-factor correlations, and allowing all items to cluster freely, EFA analysis suggested an 11 factor solution with all factors evidencing acceptable psychometric properties. Phase three of analysis aimed to develop this further by testing the structural relations between those factors. Based on theory, it was predicted that the

covariation of these behaviours could be accounted for by one single HOF. This HOF model was tested using SEM with ML Estimates in AMOS Graphics.

4.3.4.1 *Evaluating the HOF Model.* An example of the HOF model is depicted in Figure 4.2 (this Figure depicts the final, rather than the first, iteration of the HOF Model and thus only depicts nine latent variables). The first HOF model tested specified 11 latent variables (first-order factors), each represented by between three and six manifest indicators (questionnaire items). The manifest indicators used for each subscale were questionnaire items selected from the previous EFA. Manifest indicators were restricted to load only onto the latent variable they were designed to measure and, for identification purposes, one manifest indicator per factor was restricted to '1' (see section 3.4.4). As demonstrated in Figure 4.2, an exogenous HOF was specified to model common variance among the 11 first-order factors. Sample moments, freely estimated parameters, and *dfs* indicated that this model was over-identified (i.e., there were sufficient *dfs* to test the model).

The first evaluation of the model indicated some sources of misspecification (Table 4.8, 'Model A_1'; see section 3.4.3 (p. 72) for a review of fit statistics). Although RMSEA was good, χ^2 was large relative to *dfs* and CFI was below the acceptable range. Following recommendations of Joreskog (1993) a data-driven, model generating approach was thus adopted. Inspecting standardised regression weights between first-order factors and the HOF revealed that Restrictive Eating and Excessive Exercise were not significantly related to the HOF ($\beta = .08$, $p > .05$ and $\beta = -.10$, $p > .05$ respectively). The model was thus re-specified with these paths removed. This significantly improved model fit ($\Delta \chi^2_{(423)} = 1394.7$, $p < .001$) and parsimony (see Table 4.8, 'Model A_2').

Table 4.8

Fit Statistics for Initial and Trimmed Measurement Models

Model Tested	χ^2	*df*	χ^2/df	CFI	RMSEA (± 90% CI)	PCFI
Model A_1	3160.0	1116	2.8	0.89	.052 (±.050, .054)	0.84
Model A_2	1765.3	693	2.6	0.93	.046 (± .044, .049)	0.87
Model A_3 (final model)	1594.5	690	2.3	0.94	.044 (± .041, .046)	0.87
EA → Model A_3	2384.0	1067	2.2	0.92	.042 (± .040, .045)	0.87

Note: 'Model A_1' = first iteration of the HOF; 'Model A_2' = second iteration of the HOF model; 'Model A_3'= final iteration of the HOF model; 'EA → Model A_3' = final model with EA predicting HOF variance.

Next, consultation of the modification indices (MI) suggested a further source of misspecification. Specifically, three pairs of errors were correlated. This finding suggests that three pairs of manifest indicators (questionnaire items) share unique variance that is not shared by other indicators for that factor. This often occurs when two questionnaire items are particularly similar in content, such as when they are reverse coded (Byrne, 2001). In this case, the highest MI value (MI = 59.04) identified covariation between two Drug Use items that measured more severe drug use than the remaining four ("I sometimes feel that I need to take drugs" and "I sometimes think that I might have a drugs problem"). Because of the intended clinical use of the MBQ, both items were retained and their error terms were free to co-vary (see Figure 4.2). This was also the case for two DSH and two Nicotine Smoking items (items 37 & 21, and items 20 & 4 respectively, see Table 4.2). With these covariances added, acceptable fit was found (Table 4.8, Model A_3). All manifest indicators loaded significantly on to the respective first-order factor (all $\beta \geq 0.60$) and all first-order factors loaded significantly on the HOF (see Figure 4.2).

4.3.4.2 *Predicting HOF Variance.* Having evaluated and refined this model, the final stage of analysis tested whether the AAQ significantly predicted HOF variance. This model was a development of Model A_3 in which the AAQ (measured using nine manifest indicators (questionnaire items)) was hypothesised to be an exogenous latent variable that was causally related to the HOF (see Cooper et al., 2003; section 3.4.4.1). Testing this model (see Table 4.7 'EA → Model A_3') revealed that the AAQ significantly predicted the HOF ($\beta = .35$, $p < .001$), accounting for 12% of its variance.

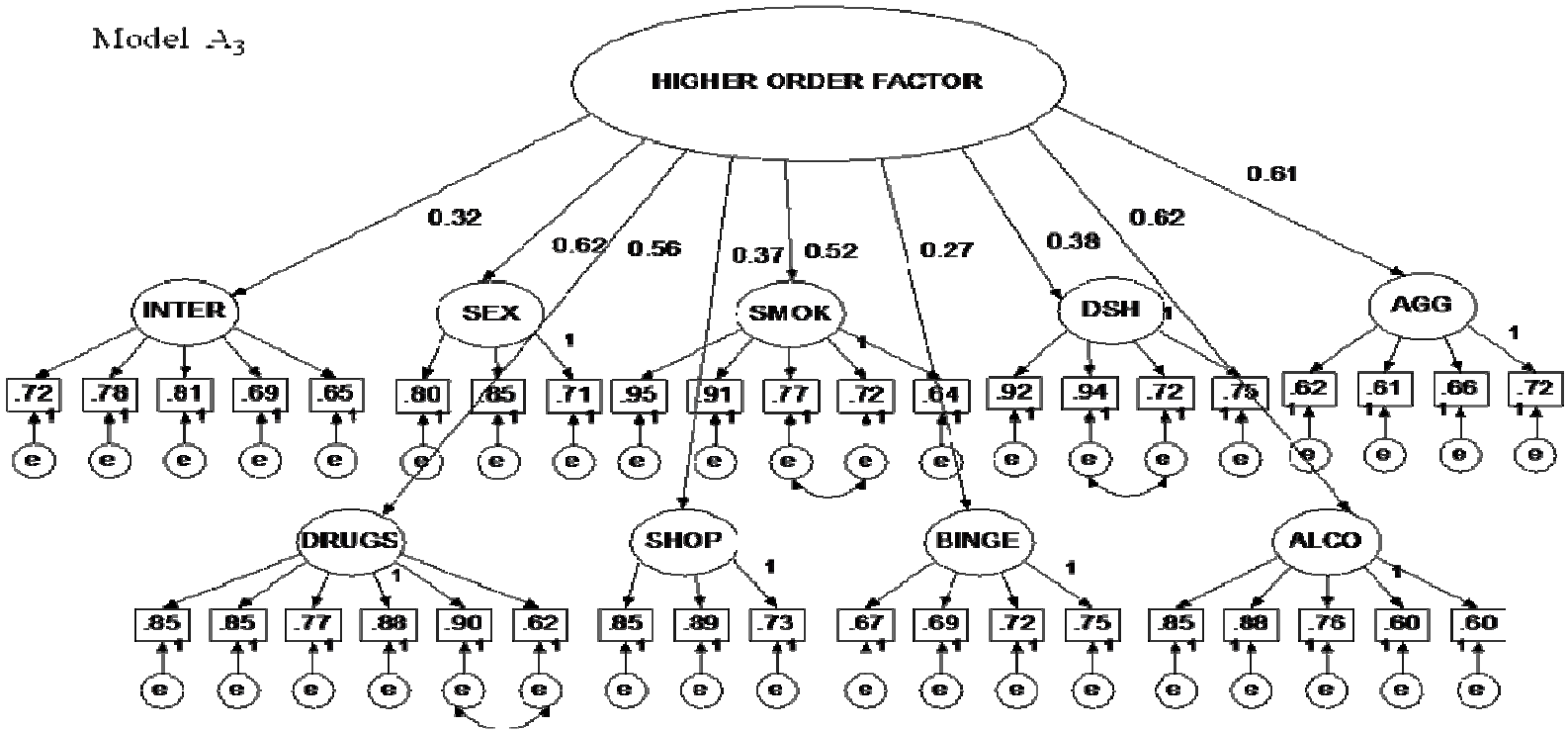


Figure 4.2 Standardised Regression Weights for trimmed HOF Model (i.e., Model A_3)

All paths significant at $p < .001$. Note: For ease of communication, error and disturbance values are not reported. Also, values for paths from factor to manifest indicator are denoted in the manifest indicator box and values from the HOF to latent factors are reported to the **right** of the path. SMOK = Nicotine Smoking; SHOP = Impulsive Spending; INTER = Excessive Internet Use; DRUG = Illicit Drug Use; BINGE = Binge Eating; DSH = Deliberate Self-Harm; SEX = Sexual Promiscuity; AGG = Aggression; ALCO = Excessive Alcohol Use. Bidirectional arrows linking 'e' indicate correlated error terms.

4.4 Discussion

4.4.1 *Study Findings*

Over a decade ago Hayes et al. (1996) proposed that, despite their formal dissimilarity, MBs constitute a functional response class whose underlying commonality is the capacity to prevent, escape, or reduce contact with negatively reinforcing private events. Although previous research has investigated covariation between small clusters of adolescent-specific risk behaviours (e.g., Cooper et al., 2003), and found evidence to support the existence of a shared HOF, this analytic technique has never before been used to examine covariation between such a wide range of clinically relevant behaviours. Research focused on identifying common factors or functions is important not only because problem behaviours commonly co-occur, but also because it may inform parsimonious treatments. To this end, this study (a) developed an easy to administer, simultaneous measure of MBs, (b) tested whether a common HOF could account for covariation between those behaviours, and (c) tested whether experiential avoidance was significantly predictive of that covariation.

Evaluation of the MBQ provided evidence to support the aspects of reliability and validity that were assessed. Because the measures against which the MBQ was evaluated were themselves well validated, it can be concluded that the MBQ exhibited good construct validity. Furthermore, adequate to good values of internal consistency suggested that items tapped common constructs, and good test retest reliability values supported the scale's stability over time. Preliminary data also suggested that the MBQ could discriminate between clinical and non-clinical samples. This having been said, however, the subscales for Drug Use and Sexual Promiscuity were not as sensitive as their validated counterparts. Furthermore, because there was no way of verifying the nature of the self-declared clinical group, these findings are preliminary and require further assessment. Indeed, scale validation is an on-going activity, and the data reported here represent the first stage of that process. Future research should evaluate the scale's applicability to other samples, particularly those drawn from a clinical population. Such work should test whether the MBQ is sufficiently sensitive to discriminate between samples with different behavioral problems (e.g., AN versus SD) and whether it can detect meaningful shifts over time.

The second aim of this study was to test structural relations between subscales (or behaviours). This analysis showed that, having undergone some modest re-specifications, the HOF model provided adequate fit statistics. Despite the range of behaviours being far broader than in previous research, the fit statistics reported in this study were comparable to, if not better than, those reported by Cooper et al. (2003; χ^2/df = 7.48; CFI = .94; RMSEA = .057 (± .052, .063)). The present findings thus extend previous research to suggest that a wide range of clinically relevant behaviours shared a common function, or common causal mechanism, of some kind. In an attempt to elucidate the nature of this commonality, it was found that the AAQ accounted for a significant proportion of HOF variance. This proportion was also comparable to the value reported by Cooper et al. for the optimal of the four predictors they investigated (Avoidant Coping: $\beta = 0.34$). These findings thus suggest that one of the reasons why MBs co-vary is because they function to alter, distract, avoid or escape from unwanted private experiences. Preliminary trends further suggested that associations between the AAQ and MBQ were greater and wider ranging in the clinical than the non-clinical subgroup. The sample size did not permit subgroup HOF modelling, however. This trend should thus be explored in future research.

Although many theory consistent findings were found, results nevertheless also indicated that some behaviours were not related to the AAQ. Similarly, in keeping with previous work (e.g., Cooper et al., 2003), a significant proportion of HOF variance was *not* explained by the hypothesised predictor (experiential avoidance). Moreover, differences emerged in the amount of variability that the HOF explained per behaviour. The fact that some behaviours (e.g., binge eating and excessive internet use) were under-represented relative to others, suggests that unique factors were more important determinants of their occurrence. This highlights the importance of establishing the relative contributions of both common and unique factors, which may help to elucidate how a common cause(s) or function(s) manifests in different behavioral topographies (see chapter 8). According to Joreskog (1993), findings such as these should be expected; “most often, the independent constructs in the model account for only a fraction of the variation and covariation in the dependent constructs, because there are many other variables that are associated with the dependent constructs that are not included in the model for various reasons” (Joreskog, 1993, p. 296). Future research

should thus build more detailed models investigating how other factors independently or interactively account for additional variance (see chapter 5 & 8).

4.4.2 *Methodological Limitations*

These findings should be considered in the light of the study's limitations. Firstly, although self-report was deemed most appropriate for the measurement of multiple risk behaviours, it has significant weaknesses. Several sources of systematic and unsystematic error, such as retrospective bias and social desirability, are likely to have affected the accuracy of findings (see section 3.2.1). Secondly, the current hypothesis was based on the assumption that experiential avoidance precedes behavioural engagement, but the use of cross-sectional data means that alternative temporal hypotheses cannot be disproved. For example, it is possible that individuals become increasingly avoidant as a result of engaging in MBs. This is possible considering that MBs often lead to aversive consequences. If this were to be the case, a different genesis, such as the seeking of positive affect, could play a primary etiological role; whereas experiential avoidance may be implicated as a developmental and maintenance factor. It is most likely that some feedback loop does exist, whereby individuals become increasingly more avoidant as the use of MBs becomes more entrenched. Such subtleties could not, however, be detected using cross-sectional design. This should be a consideration for future research and could be addressed using a longitudinal cross-sectional approach.

The third limitation concerns sample characteristics. Although a broad range of respondents took part, students were over-represented, and clinical and male participants were under-represented. These demographics are likely to have impacted on the findings. For example, preliminary analyses suggested that relations between the AAQ and MBQ were greater in participants drawn from a clinical sample; however, the sample size was too small to explore this more fully. Another concern regarding sampling was that, although the 'self-declared clinical' versus 'non-clinical' distinction appeared to have some validity, this approach was limited in two main ways. Firstly, a problem that is inherent to work that uses the 'clinical' versus 'non-clinical' distinction is that many people with 'clinical symptoms' do not seek treatment (see Bandura, 1978). Secondly, the method of categorisation failed to accommodate participants who

had sought medicinal, rather than psychological, help for a psychological problem. Future research should evaluate the MBQ's applicability to other samples, especially better defined clinical samples.

The last two considerations relate to the statistical procedures that were used. Firstly, this study used the same data set for EFA and CFA analysis, which may prove circular. For example, adequate CFA statistics could in part reflect the fact that item clustering had been determined using the same sample's data. Nevertheless, the EFA was not used to influence factor-factor interrelations in the measurement model; rather, the HOF model was determined exclusively by previous research and theory. This crucial aspect of the modelling was therefore uninfluenced by results of the EFA. Future research should re-evaluate this model in a second sample to test its structural reliability (see chapter 5). Secondly, the use of SEM to identify underlying factors warrants some consideration. This statistical method is based on the analysis of latent constructs, which can only be inferred through component manifest indicators. Similarly, the nature and function of the HOF can only be investigated indirectly and through the use of predictor variables (e.g., experiential avoidance). To avoid unfounded assumptions, therefore, it is best to conceptualize the HOF as a factor that models common variance across first-order factors, but that may or may not cause those first-order factors.

4.4.3 *Implications*

This study has several implications. Firstly, on a practical level, the data have been largely supportive of the MBQ as a measurement tool, which may prove useful for monitoring co-occurring MBs. For example, it may assist professionals in monitoring both target and non-target behaviours during treatment, enable pre-post change analysis (see chapter 7), and help in the identification of phenomena such as behaviour switching. Furthermore, the implication that a common factor exists to unite these behaviours suggests that they may be usefully considered as related, rather than independent constructs. This tool may help future research to obtain a more detailed understanding of common factors.

The current findings also have treatment implications. If findings generalise to clinical samples then interventions designed to reduce experiential avoidance, such as acceptance-based and mindfulness-based interventions, may prove effective for treating

independent and co-occurring problem behaviours. This is an important treatment implication, given that these behaviours are known seldom to occur in isolation *and* given that their co-occurrence is known to challenge traditional cognitive interventions (see section 1.2.4.1). To date, early indications suggest that ACT can be therapeutically useful for patients with polysubstance abuse (Hayes, Wilson, et al., 2004), DSH (Gratz & Gunderson, 2006), and nicotine addiction (Gifford et al., 2004); and mindfulness-based approaches have utility in the treatment of substance abuse (Marlatt, et al., 2004). However, no known research has extended the application of ACT to the treatment of co-occurring MBs. These findings suggest that the reduction of experiential avoidance may be one promising technique for disrupting the process through which these behaviours hang together.

4.4.4 *Summary*

This study has presented one feasible and promising method for measuring MBs and their co-occurrence. Furthermore, evidence was found to suggest that experiential avoidance may account for some of the covariation between them. Although this provides important information about the relationship between experiential avoidance and MBs, it says little about the broader context within which these associations are typically embedded. For example, what variables predict heightened levels of experiential avoidance and how does experiential avoidance relate to other factors known to influence MBs? With these questions in mind, study 2 was designed to obtain a more detailed and integrated understanding of the association between experiential avoidance and MBs. Study 2 was also designed to re-test the structural reliability of the HOF, and to explore the implicated differences between clinical and non-clinical groups.

CHAPTER V

Study 2. Experiential Avoidance and Maladaptive Behaviours Part II: Does Experiential Avoidance Mediate the Relationship Between Negative Affect Intensity, Childhood Trauma and Maladaptive Behaviours?

5.1 Introduction

As predicted, study 1 showed that the HOF model provided a parsimonious and acceptable account of MB covariance. Furthermore, the AAQ explained a significant proportion of that covariance. The current study was intended to develop this work further, by gaining a more detailed understanding of how experiential avoidance may be implicated in the tendency to engage in these behaviours. In the literature, many risk factors have been identified as predisposing individuals for co-morbid behaviour problems. Of these, intense negative affect and childhood trauma appear to be most prominent. The processes through which these factors affect future functioning, however, are not well known. According to ACT, experiential avoidance may be crucial to elucidating this process. From an ACT perspective, it can be predicted that negative affect and childhood trauma predict MBs only indirectly and through the mediating effect of experiential avoidance (Figure 5.1). This study aimed to test these predictions.

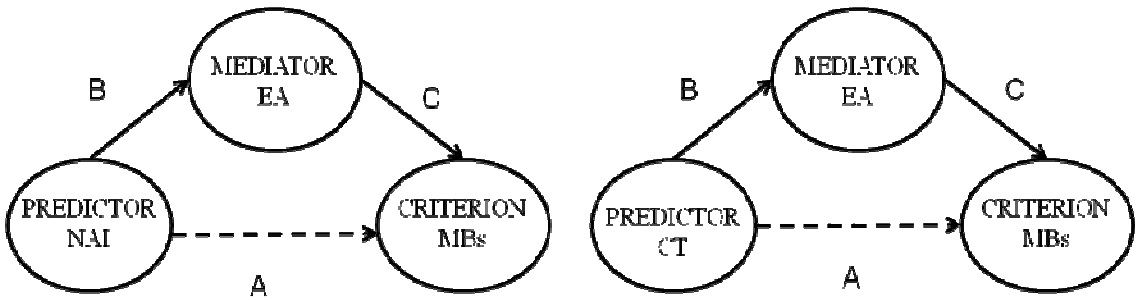


Figure 5.1 Simplified Version of Predicted Mediational Models with Experiential Avoidance Mediating the Relationship between Negative Affect Intensity, Childhood Trauma and Maladaptive Behaviours.

Note: CT = Childhood Trauma; NAI = Negative Affect Intensity; EA= Experiential Avoidance; MBs = Maladaptive Behaviours. Solid lines indicate direct relations; dashed lines indicate indirect (mediated) relations. 'A' 'B' and 'C' denote labels for the three hypothesised pathways.

5.1.1 *Pathway A: Risk Factors for Maladaptive Behaviours*

Research suggests that heightened levels of negative affect often precede and predict a variety of MBs. These have included, for example, DSH (see Chapman et al., 2006), binge eating (Agras & Telch, 1998; Deaver**,** Miltenberger, Smyth, Meidinger, & Crosby, 2003), substance use/abuse (see Baker, Piper, McCarthy, Majeskie, & Fiore, 2004; Kassal, Stroud, & Paronis, 2003), aggression (Verona, Patrick, & Lang, 2002), and excessive internet use (Yen et al., 2008). Based on these findings, it is intuitive to predict that the trait-based construct of *Negative Affect Intensity* (NAI) —the predisposition to experience intense negative affect (Bryant, Yarnold, & Grimm, 1996)—may also be significantly predictive of the tendency to engage in MBs. Although research on NAI and MBs is sparse, research has shown a significant association between NAI and DSH (Gratz & Roemer, 2008), BPD symptoms (Cheavens et al., 2005; Rosenthal et al., 2005; Yen, Zlotnick, & Costello, 2002), substance abuse (Thorberg & Lyvers, 2006), and suicidality (Lynch et al., 2004).

Traumatic experiences in one's childhood (*Childhood Trauma* (CT); Bernstein et al., 2003), such as being the victim of abuse (e.g., sexual, physical, emotional) and/or neglect (e.g., physical, emotional), is also significantly predictive of the tendency to engage in MBs. For example, using a prospective cohort design, Spatz, Marmorstein, and Raskin (2006) found that participants reporting CT were significantly more likely to abuse substances than matched controls. Similarly, longitudinal research has shown that a wide range of traumatic experiences in childhood are significantly predictive of disordered eating (Johnson, Cohen, Kasen, & Brook, 2002; see also Smolak & Murnen, 2002) and suicidality (Johnson, Cohen, Gould et al., 2002). Furthermore, individuals reporting CT, relative to non-abused counterparts, are more likely to engage in a range of health risk activities (e.g., alcohol use, risky sexual practices; Rodgers et al., 2004), to be aggressive (e.g., Herrenkohl et al., 2004) and to engage in DSH (Santa Mina & Gallop, 1998).

5.1.2 *Path B: NAI, CT and Experiential Avoidance*

Is there a relationship between the NAI, CT, and the tendency to avoid unwanted experiences? Research suggests so. Individuals high in NAI are more likely to engage in avoidant behaviour after an experimental stressor (e.g., Bijttebier & Vertommen, 1999).

Similarly, cross-sectional research has established a link between NAI and different indices of avoidance (e.g., emotional inhibition (e.g., Lynch et al., 2004; Lynch et al., 2001) and thought suppression (Cheavens et al., 2005; Rosenthal et al., 2005)). Individuals reporting CT are also likely to develop avoidant attachment styles (e.g., see Shapiro & Levendosky, 1999), and to engage in thought suppression (Batten, Follette, & Aban, 2001) and avoidant coping (Hyman, Paliwal, & Sinha, 2007; Kuyken & Brewin, 1994). Focusing specifically on experiential avoidance, Marx & Sloan (2002) have also reported that sexual abuse is significantly predictive of AAQ scores.

5.1.3 *Path C: Experiential Avoidance and Maladaptive Behaviours*

Finally, does experiential avoidance predict MBs? Study 1 of this thesis certainly suggests so, showing that the AAQ predicted 12% of MB covariance. These data converge with previous research, which has found that substance misusers often report escape from aversive psychological states as a central motivation for consuming addictive substances (e.g., see Baker et al., 2004). Research has also shown that individuals who use alcohol to alleviate distress are more likely to develop an abuse problem (Cooper et al., 1992). Additionally, current negative reinforcement models of non-pharmacologically based MBs, such as DSH (Chapman et al., 2006) and dysfunctional eating (Heatherton & Baumeister, 1991; Deaver et al., 2003; see also Miltenberger, 2005) have received some empirical support (e.g., study 1; Najmi et al., 2007; McManus & Waller, 1995; Meyer, Waller, & Watson, 2000).

The literature reviewed above suggests that a predisposition towards heightened negative arousal and/or a history of childhood trauma increase the probability of engaging in MBs. An ACT-derived interpretation of these associations, however, would suggest that these links are indirect and mediated by the intervening effect of experiential avoidance (e.g., Hayes et al., 1996). This interpretation is plausible, given that experiential avoidance is predicted by these risk factors *and* is itself predictive of MBs (see Figure 5.1). From this perspective, NAI and CT increase the probability of engaging in MBs through the fostering of heightened experiential avoidance. This study was designed to test these hypotheses.

5.1.4 *Methodological Considerations*

The design of the present study was strongly influenced by the techniques available for measuring the key constructs of NAI, CT, experiential avoidance and MBs. To date, this technique is self-report questionnaires (some authors have used interviews for MBs and CT). For example, the most comprehensive instrument designed for the measurement of CT is the Childhood Trauma Questionnaire (CTQ-SF; Bernstein et al., 2003), which provides a means of anonymously measuring five separable facets of trauma (see section 5.2.3.2). Although NAI, experiential avoidance, and MBs could be measured using behavioural procedures, no validated methods have been established.

Questionnaire-based research lends itself either to the use of longitudinal or cross-sectional design. Although longitudinal design is a more powerful method for drawing inferences of cause-and-effect, it did not naturally lend itself to the aims of the current study. This is because it would be impossible to take any form of baseline measures (i.e., experiential avoidance and MB scores before and after an abusive episode). Informed by much of the current work in this area of research (e.g., Chapman et al., 2005; Lynch et al., 2004; Najmi et al., 2007), therefore, a cross-sectional design was used. In keeping with study 1, a large community sample was recruited with the aim of accessing a self-identified clinical subgroup. To improve on study 1's recruitment, however, this study was advertised on a broader range of websites. Furthermore, the present study included an item to establish whether participants had taken, or were currently taking, prescribed medication for a psychological problem. It was reasoned that the failure to add this to the last study could have lead to the mis-categorisation of some participants.

5.1.5 *Synopsis of the Present Study*

This study aimed to test the mediational models depicted in Figure 5.1; that is, the prediction that experiential avoidance would mediate the relationship between NAI, CT, and the tendency to engage in MBs. To obtain a full and detailed understanding of the proposed relations, a step-wise method of analysis was adopted. First, the models were tested using Baron and Kenny's (1986) mediational criteria (see section 3.4.2 and 5.3.2). This stage aimed to test the mediational models when predicting composite MBQ scores and also when predicting each behaviour in isolation (e.g., Restrictive

Eating, DSH etc.). Following this, SEM was used to test whether experiential avoidance mediated the effect of NAI and CT on MB covariation (i.e., the HOF).

5.2 Method

5.2.1 *Design*

A cross-sectional, questionnaire-based design was employed. Predictor variables included childhood trauma and negative affect intensity. The tendency to engage in maladaptive behaviours was the criterion variable and the mediator variable was experiential avoidance.

5.2.2 *Participants*

An opportunity sample of participants was recruited (N = 719). Psychology students from the University of Southampton (N = 287), recruited via electronic and paper advertisements, received course credits for participation. Participants from outside the University (N = 432) were recruited via electronic advertisements on the www. This method of recruitment was similar to that of study 1. Thus, although study 2 was conducted almost 12-months after study 1, it ran the risk of recruiting some of the same participants. Cross-referencing student ID numbers across studies revealed that approximately 40% of the Southampton student sample (i.e., N = 115) had also participated in study 1. Despite this, demographic variables (Table 5.1) suggested that the samples of study 1 and 2 were distinguishable from one another.

5.2.3 *Materials*

Measures for the measurement of MBs and experiential avoidance are described in study 1 (section 4.2.2). Demographic variables were measured in the same way as those described in study 1 except participants were additionally asked to indicate whether they were currently taking, or had in the past taken, prescribed medication for a psychological problem (see Appendix A). The following measures were also used.

Table 5.1

Demographic Statistics Split by 'Clinical Status'

	Opportunity Sample	
Demographics	Self-Declared Non-clinical (N = 410)	Self-Declared Clinical (N =309)
Mean Age (SD)	22.11 (7.0)	26.26 (9.7)
Gender (% Female)	76.7%	87.2%
Occupation (%):		
Student Psychology	63.7%	43.1%
Student Other	21.4%	21.3%
Employed	12%	23.4%
Unemployed	2%	5.4%
Not specified	0.9%	5.8%
Country of Origin:		
England	56.4%	34.7%
USA	30.2%	57.3%
Europe (other)	6.2%	4.2%
Asia	2.4%	1.2%
Other	4.8%	2.6%
Mean no. of therapies	0	1.56
Mean no. of sessions	0	34.34

Note: 'Self-declared clinical' were those participants indicating that they were either currently receiving, or had in the past received, therapy for a psychological difficulty. The 'self-declared non-clinical' sample were those indicating that they had not.

5.2.3.1 The *Affect Intensity Measure – Negative Intensity subscale (AIM-NI;* Larsen & Diener, 1987; Bryant et al., 1996). The AIM-NI is a 6-item subscale of the Affect Intensity Measure. This subscale assesses how strongly negative emotions are experienced when they occur, in a manner that does not confound intensity with the frequency of those emotions (e.g., "My emotions tend to be more intense than those of most people"). Items are rated on a 6-point scale ranging from 1 (never) to 6 (always). Bryant et al. reported acceptable internal reliability ($\alpha = .70$) and good test retest reliability over a 2 year period ($r = .71$). This supports a temperamental interpretation of the nature of NAI.

5.2.3.2 The *Childhood Trauma Questionnaire Short Form (CTQ-SF;* Bernstein et al., 2003). The CTQ-SF is a 28-item self-report inventory measuring five facets of CT: Emotional, Physical, and Sexual Abuse, and Emotional and Physical Neglect. Items (e.g., "When I was growing up I felt hated by my family" for Emotional Neglect) are rated on a scale ranging from 1 (never) to 5 (very often true). Adequate psychometric properties have been reported for both clinical and non-clinical populations (e.g., $\alpha = .92$; Wright et al., 2001).

5.2.4 *Procedure*

This was an online study that required participants to complete a consent form before accessing questionnaires. The form pre-warned participants that some questionnaires were of a personal nature. Questionnaire packs were completed in the participants' own time and they were advised to complete them in private. To control for order effects, the sequence in which the questionnaires were presented, and the order of questions per questionnaire, was randomised. University of Southampton Psychology students were required to indicate their ID number on a separate web page to ensure the allocation of course credits. These details were stored in separate data files from questionnaire responses. Owing to the potentially upsetting nature of some of the questionnaire items, participants were provided with a debriefing statement that included the contact details of help organisations (e.g., The Samaritans). They additionally received the researcher's contact details for questions or comments.

5.2.5 *Analysis Strategy*

Preliminary statistics assessed variable distributions, tested for predicted correlations and for between-group (i.e., clinical versus non-clinical subgroups) differences. Next, *Phase One* of analysis used Baron and Kenny's (1986) 4-step process to test for mediation (see section 3.4.3 and 5.3.2). Sobel's (1982) test for indirect effects was added as a fifth step to test whether the indirect effect was significant. Separate mediation analyses were computed for testing the effect of NAI, CT, and experiential avoidance on (a) composite MBQ scores and (b) each MBQ subscales in isolation (e.g., Restrictive Eating, DSH etc.). *Phase Two* addressed the same fundamental questions, but using the more flexible and integrated approach offered by SEM. First, the structural reliability of the HOF model and the predictive value of the AAQ identified in study 1 was re-tested with the new dataset. Following this, a structural model was developed to test whether the AAQ mediated the relationship between AIM-NI, CTQ-SF, and the MBQ HOF. Two models were tested and statistically compared. The first model proposed indirect effects of AIM-NI and CTQ-SF on the HOF (i.e., mediated through the AAQ), and the second proposed that AIM-NI and CTQ-SF had both direct and indirect effects. Chi square difference tests ($\Delta \chi^2$) were computed to compare the fit of the two models (Kline, 2005; see also section 3.4.4).

5.3 Results

5.3.1 *Preliminary Statistics*

5.3.1.1 *Missing Data, Outliers, and Distributions.* Inspection of the raw data showed that 13 participants had high levels of missing data (see section 4.3.1). These individuals' data were excluded from further analysis. Small amounts of missing data (i.e., < 10%) were replaced with the group mean value for that item. Descriptive statistics (stem and leaf graphs) and Mahalanobis distance values further identified four univariate and twelve multivariate outlier participants. These individuals' data were also removed from further analysis, resulting in a final N = 690. Finally, measures of skew and kurtosis showed that DSH, Nicotine Smoking, Drug Use, and the CTQ-SF composite and subscales were positively skewed. Logarithmic transformations corrected the skew of all these variables except Sexual Abuse, which remained highly skewed. The Sexual Abuse subscale was thus excluded from the SEM modelling (items were, however, included in the composite

measure that was used for regression analysis). All other variables were normally distributed (see Table 5.2 for means, standard deviations and internal reliability).

5.3.1.2 *Clinical versus Non-Clinical Subgroups*. Consistent with Study 1, MANCOVA (controlling for country of origin, occupation and gender) revealed several significant differences between the self-declared clinical and non-clinical subgroups (see Table 5.2). These differences supported the idea that the clinical group was drawn from a clinical population. Because sample sizes permitted, analyses were conducted on full sample data, followed by the data of clinical and non-clinical subgroups.

5.3.1.3 *Bivariate Correlations.* Bivariate correlations between MBQ subscales showed that, consistent with study 1, Excessive Exercise and Restrictive Eating were unrelated to most other MBQ subscales. Excessive Exercise was not significantly related to *any* other MBQ subscale and Restrictive Eating only to DSH ($r = .32$, $p < .001$). The replication of this finding justified their exclusion from the HOF modelling.

Bivariate correlations between predictor, mediator, and criterion variables (excluding Excessive Exercise and Restrictive Eating) are reported in Table 5.3. Consistent with prediction, MBQ composite scores were significantly related to AIM-NI, CTQ-SF, and the AAQ. This was the case for the full group and for each subgroup (i.e., clinical and non-clinical group). Analyses using the MBQ subscales provided a more detailed account of these relations. In the full sample, the AAQ was significantly related to all MBQ subscales *except* Sexual Promiscuity; whereas CTQ-SF and AIM-NI were only related to a subset of these subscales. Furthermore, consistent with study 1, some differential trends emerged across subgroups. For example, the AAQ was again related to a broader range of behaviours in the clinical than the non-clinical group (specifically: Sexual Promiscuity, Nicotine Smoking, and Excessive Alcohol Use). Conversely, the correlations between AIM-NI and MBQ subscales were generally higher in the non-clinical group than the clinical group (e.g., Excessive Alcohol Use and Binge Eating).

Table 5.2

Means (SD) and Internal Reliability Values for all study Variables in the Full Sample and Clinical/Non-Clinical Subgroups.

	Total Sample (N = 690)			Non-Clinical (N = 389)			Clinical (N = 301)			Non-Clinical vs. Clinical
	Mean	SD	α	Mean	SD	α	Mean	SD	α	MANCOVA (*F*)
AAQ	4.00	0.93	.75	3.69	0.76	.70	4.37	0.97	.79	43.26***
AIM-NI	3.71	1.05	.82	3.43	1.01	.82	4.15	0.95	.75	30.52***
CTQ-SF	1.61	0.68	.94	1.45	0.58	.94	1.87	0.75	.92	30.43***
MBQ Composite	2.62	0.58	.86	2.51	0.51	.85	2.70	0.63	.88	8.65***
DSH†	1.81	1.23	.88	1.54	0.97	.84	2.17	1.43	.89	20.32***
Nicotine Smoking†	2.22	1.43	.88	2.00	1.23	.86	2.51	1.62	.90	8.82***
Restrictive Eating	2.55	1.10	.71	2.40	1.02	.73	2.74	1.14	.74	5.53**
Excessive Internet Use	2.92	1.22	.81	2.85	1.15	.74	3.01	1.30	.83	3.03*
Drug Use†	1.93	1.31	.81	1.80	1.13	.88	2.07	1.50	.88	3.00*
Excessive Exercise	3.07	1.20	.76	3.16	1.14	.78	2.94	1.26	.81	2.57
Binge Eating	3.09	1.24	.81	2.99	1.12	.77	3.23	1.37	.88	2.17
Excessive Alcohol Use	3.21	1.33	.87	3.26	1.30	.88	3.14	1.36	.86	1.11
Aggression	2.30	1.04	.75	2.26	0.97	.73	2.33	1.11	.76	1.10
Sexual Promiscuity	1.97	1.19	.79	1.95	1.17	.77	2.00	1.21	.72	0.68
Impulsive Spending	3.46	1.42	.76	3.40	1.34	.83	3.52	1.52	.89	0.34

*$p < .05$, ** $p < .01$, *** $p < .001$. † Transformed scores were used in the MANCOVA. AIM-NI = Affect Intensity Measure –Negative Intensity Scale; CTQ-SF = Childhood Trauma Questionnaire; AAQ = Acceptance and Action Questionnaire; MBQ Composite = Maladaptive Behaviour Questionnaire composite scores.

Table 5.3

Inter-variable Correlations for the Full Sample and Clinical/Non-Clinical Subgroups

Predictors/Mediator/Criterion	Total Sample (N = 690)			Non-Clinical (N = 389)			Clinical (N = 301)		
	AAQ	AIM-NI	CTQ-SF	AAQ	AIM-NI	CTQ-SF	AAQ	AIM-NI	CTQ-SF
AAQ	-	.55*	.30*	-	.46*	.22*	-	.53*	.22*
MBQ Composite	.35*	.27*	.25*	.29*	.24*	.25*	.34*	.23*	.25*
DSH†	.38*	.29*	.35*	.36*	.24*	.30*	.37*	.27*	.31*
Nicotine Smoking†	.14*	.09	.23*	.06	.00	.21*	.14*	.08	.19*
Excessive Alcohol Use	.15*	.10	.11	.13	.14*	.11	.24*	.10	.05
Restrictive Eating	.23*	.23*	.23*	.20*	.16*	.16*	.18*	.23*	.18*
Aggression	.24*	.23*	.22*	.30*	.28*	.34*	.20*	.18*	.16*
Excessive Internet Use	.23*	.07	.10	.18*	.03	.19*	.25*	.08	.08
Drug Use†	.12*	.09	.17*	.11	.08	.21*	.07	.05	.16*
Binge Eating	.20*	.21*	.03	.20*	.28*	.02	.17*	.10	.06
Sexual Promiscuity	.09	.03	.12*	.02	.03	.12	.19*	.10	.12
Impulsive Spending	.20*	.22*	.01	.23*	.28*	.03	.17*	.15*	.05
Excessive Exercise	.13*	.07	.03	.10	.07	.03	.10	.00	.00

*$p \leq .001$ (adjusted for multiple comparisons). Note: AIM-NI = Affect Intensity Measure – Negative Intensity Scale; CTQ-SF = Childhood Trauma Questionnaire Short Form; MBQ Composite = Maladaptive Behaviour Questionnaire composite scores. For all scales, high scores denote greater values of that variable. Correlations concerning transformed variables are computed in transformed state but means are presented in non-transformed state to ease interpretation.

5.3.2 *Phase One Mediation Analysis: Step-Wise Regression*

Baron and Kenny's (1986) 4-step process was used to test whether the AAQ mediated the relationship between predictor variables (CTQ-SF and AIM-NI) and the criterion (e.g., MBQ composite scores). Sobel's (1982) test for indirect effects was added as a fifth step. These steps are summarised below:

Step 1: predictor significantly correlates with criterion

Step 2: predictor significantly correlates with mediator

Step 3: mediator significantly correlates with criterion

Step 4: the effect of the predictor on the criterion, controlling for the effect of M, is not significantly different from 0

Step 5: the indirect effect is significant

Partial mediation was inferred when the relationship between the predictor and criterion was significantly reduced (i.e., significant Sobel's test), but remained significantly different from 0 (Frazier, Tix, & Barron, 2004). Because a prerequisite for a mediational interpretation is the observation of significant associations between the predictor, mediator, and criterion (step 1, 2 & 3), mediational analyses were computed only for sets of variables meeting all these requirements (as indicated by correlations in Table 5.3). The following sets of variables were thus tested:

Full Sample: AIM-NI predicting Binge Eating, Aggression, Impulsive Spending, DSH and Restrictive Eating. CTQ-SF predicting Drug Use, Nicotine Smoking, Aggression, Sexual Promiscuity, DSH, and Restrictive Eating.

Clinical Subgroup: AIM-NI predicting Aggression, Impulsive Spending, Restrictive Eating and DSH. CTQ-SF predicting Nicotine Smoking, DSH and Restrictive Eating.

Non-clinical Subgroup: AIM-NI predicting Binge Eating, Aggression, Impulsive Spending, DSH, and Restrictive Eating. CTQ-SF predicting Aggression, DSH, Excessive Internet Use, and Restrictive Eating.

5.3.2.1 *Full Group Analyses.* Evidence supporting the first three of Baron and Kenny's (1986) steps are reported in Table 5.3. Step four was tested by regressing the criterion (e.g., MBQ composite) onto the predictor (AIM-NI or CTQ-SF) in block 1 of the

analysis and onto the AAQ in block 2. Results, reported in Table 5.4, indicated that the AAQ *fully* mediated the effect of AIM-NI→MBQ composite scores (β = .27 to β = .11). The AAQ also *partially* mediated the effect of AIM-NI→Binge Eating (β = .21 to β = .15), Aggression (β = .23 to β = .14), Impulsive Spending (β = .22 to β = .16), DSH (β = .31 to β = .12), and Restrictive Eating (β = .23 to β = .15). With regard to the CTQ-SF, the AAQ *partially* mediated the effect of CTQ-SF→MBQ composite scores (β = .28 to β = .19), Aggression (β = .25 to β = .19), DSH (β = .36 to β = .26), and Restrictive Eating (β = .21 to β = .16). The AAQ did not, however, predict unique variance in Nicotine Smoking. Furthermore, in block 2 of analysis, neither the AAQ nor the CTQ-SF predicted unique variance in Sexual Promiscuity.

5.3.2.2 *Clinical Subgroup Analyses.* Evidence supporting mediational steps one to three in the clinical subgroup is reported in Table 5.3. Testing step four (see Table 5.4), it was found that the AAQ *fully* mediated the relationship between AIM-NI→MBQ composite scores (β = .23 to β = .08), Aggression (β = .18 to β = .09), and DSH (β = .27 to β = .13). For the case of Restrictive Eating, however, the effect of the AAQ was non-significant in block 2 of analysis. Testing step four with CTQ-SF as the predictor indicated that the AAQ *partially* mediated the effect of CTQ-SF→MBQ composite scores (β = .25 to β = .18), DSH (β = .31 to β = .24), and Restrictive Eating (β = .18 to β = .15).

5.3.2.3 *Non-Clinical Group.* Evidence supporting the first three steps of mediation in the data of the non-clinical subgroup is reported in Table 5.3. Results for step four, reported in Table 5.4, indicated that the AAQ *partially* mediated the effect of AIM-NI→MBQ composite scores (β. = .24 to β. = .13). Furthermore, the AAQ *fully* mediated the relationship between AIM-NI→DSH (β. = .24 to β. = .09) and Restrictive Eating (β. = .16 to β. = .09) and *partially* mediated the effect of AIM-NI→Aggression (β = .28 to β = .18) and Impulsive Spending (β = .28 to β = .22). The AAQ was not, however, predictive of Binge Eating when controlling for the effect of AIM-NI. Finally, analyses with CTQ-SF as the predictor showed that the AAQ *partially* mediated the effect of CTQ-SF→MBQ composite scores (β = .25 to β = .19), Aggression (β .34 to β = .29), DSH (β. = 30 to β. = .23), Excessive Internet Use (β = .19 to β. =.15), and Restrictive Eating (β .16 to β = .13).

Table 5.4

Regression Analysis Testing Step Four of Baron and Kenny's Mediation Criteria in the Full Sample, and Clinical/ Non-Clinical Subgroups.

Full Sample (N = 690)												
AIM-NI												
Criterion	MBQ Composite		Binge Eating		Aggression		Impulsive Spending		DSH		Restrictive Eating	
Predictor	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β
AIM-NI	.27***	.11	.21***	.15**	.23***	.14**	.22***	.16***	.31***	.12*	.23**	.15**
AAQ	-	.29***	-	.12**	-	.16***	-	.11*	-	.34***	-	.15**
F of step	54.46***	51.45***	32.07***	19.99**	37.96***	26.10***	35.85***	21.23**	72.00***	72.06***	37.86**	24.69**
Overall R^2	.067	.131	.045	.060	.051	.068	.050	.060	.095	.175	.052	.067
Sobel	6.25***		2.73***		3.61**		2.47***		7.56***		3.25***	
CTQ-SF												
Criterion	MBQ Composite		Nicotine Smoking		Aggression		Sexual Promiscuity		DSH		Restrictive Eating	
Predictor	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β
CTQ-SF	.28***	.19***	.24***	.22***	.25***	.19***	.12*	.09	.36*	.26*	.21*	.16*
AAQ	-	.29***	-	.07	-	.18*	-	.06	-	.33*	-	.18*
F of step	57.12***	62.33***	41.30***	21.30***	44.56***	34.15***	9.60*	6.08*	100.7***	99.72***	31.92*	27.35**
Overall R^2	.077	.154	.057	.061	.061	.091	.014	.017	.128	.223	.045	.074
Sobel	5.66***		1.69		4.08***		1.56		6.27***		4.03**	

***p = <.001 **p = <.01 *p = < .05.

Clinical Subgroup (N = 301)

AIM-NI

Criterion	MBQ Composite		Aggression		DSH		Restrictive Eating	
Predictor	Block 1 β	Block 2 β	Block 1 β	Block 2β	Block 1 β	Block 2 β	Block 1 β	Block 2 β
AIM-NI	.23***	.08	.18**	.09	.27***	.13	.23***	.19**
AAQ		.28***	-	.15*	-	.27***	-	.08
F of step	17.00***	18.60***	9.48***	7.14***	23.21***	20.93***	17.11***	9.21***
Overall R^2	.054	.112	.031	.046	.073	1.24	.055	.059
Sobel	4.07***		2.12*		3.19***		1.13	

CTQ-SF

Criterion	MBQ Composite		Restrictive Eating		DSH	
Predictor	Block 1 β	Block 1 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β
CTQ-SF	.25***	.18***	.18**	.15*	.31***	.24***
AAQ	-	.29***		.15*		.28***
F of step	18.89***	23.82***	10.34***	8.43***	30.89***	30.35***
Overall R^2	.060	.139	.034	.054	.097	.171
Sobel	3.06***		2.09***		3.05***	

***p = <.001 **p = <.01 *p = < .05

Non-Clinical Subgroup (N = 389)

AIM-NI

Criterion	MBQ Composite		Binge Eating		Aggression		Impulsive Spending		DSH		Restrictive Eating	
Predictor	Block 1 β	Block 2 β	Block 1 β	Block 2β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β
AIM-NI	.24***	.13*	.28***	.24***	.28***	.18**	.28*	.22***	.24***	.09	.16***	.09
AAQ	-	.27***	-	.09	-	.21***	-	.13	-	.32***	-	.16***
F of step	17.00***	18.60***	33.03***	18.01***	32.00***	24.15***	32.60***	19.14***	23.62***	31.00***	9.96**	9.06**
Overall R^2	.054	.112	.079	.086	.077	.112	.078	.091	.060	.140	.025	.045
Sobel	4.32***		1.65		3.65***		2.24***		5.13***		2.73***	

CTQ-SF

Criterion	MBQ Composite		Aggression		DSH		Excessive Internet Use		Restrictive Eating	
Predictor	Block 1 β	Block 1 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β	Block 1 β	Block 2 β
CTQ-SF	.25***	.19***	.34***	.29***	.30***	.23***	.19***	.15**	.16**	.13*
AAQ	-	.27***		.23***		.32***		.15**		.21***
F of step	25.52***	24.43***	49.53***	37.98***	37.89***	43.10***	14.50***	11.58***	10.63***	11.10***
Overall R^2	.062	.113	.114	.165	.090	.183	.036	.057	.030	.070
Sobel	3.44***		3.26***		3.69***		2.42**		2.67**	

***p = <.001 **p = <.01 *p = < .05

In summary: this network of findings suggested that experiential avoidance was an important variable for understanding the relationship between AIM, CT and MBs. The AAQ tended to fully mediate the effect of AIM-NI, and partially mediate the effect of CTQ-SF, on *composite* MBQ scores. This was echoed in subscale and subgroup analyses. Overall, these analyses showed that the AAQ reduced a substantial proportion of the effect of AIM-NI, and a slightly more moderate proportion of the effect of CTQ-SF, on many of the criterion measures (e.g., Aggression, DSH). Having found some support for the hypothesised models using the traditional Baron and Kenny (1986) approach, SEM was employed as a more integrated and thorough test of whether the AAQ mediated the relationship between AIM-NI, CTQ-SF, and the MBQ HOF.

5.3.3 *Phase Two Mediation Analyses: SEM*

Phase two of analysis began by re-testing the structural reliability of the HOF model. This was tested in the full sample and in clinical and non-clinical subgroups, using statistical procedures previously described in section 4.3.5.

Results (see Table 5.5, 'Model A_3') showed that the HOF model provided an adequate fit of the full sample data, but differential effects emerged between subgroups. Specifically, the HOF model adequately fitted the correlation/covariation matrix of the self-declared clinical, but not the non-clinical, subgroup. For the non-clinical subgroup, although χ^2/df and RMSEA values were acceptable, CFI was not (see Table 5.5). MI values suggested that this mis-specification was attributable to two unique first-order factor relations (Excessive Alcohol Use: Sexual Promiscuity and Nicotine Smoking: Drug Use) that were not accounted for by the HOF. This suggests that, in this subgroup, these pairs of subscales shared unique variance that was not shared with the remaining seven subscales. The model was not re-specified to accommodate these factor-factor relations, because adding paths between first-order factors would undermine the theoretical proposition of a HOF model (i.e., that all first-order factor-factor relations can be accounted for by a single HOF). No further SEM analyses were computed in the non-clinical group, therefore, because results could not be reliably interpreted (Byrne, 2001). Testing whether the AAQ predicted HOF variance in the full and clinical subgroup (Table 5.5 'Model A_3 + EA') showed that the AAQ predicted 28% ($\beta = 0.53$, $p < .001$) and 22% ($\beta = 0.47$, $p < .001$) respectively.

Table 5.5

HOF Model Fit Statistics for the Full Sample and Clinical/Non-clinical Subgroups

Model Tested	Sample	χ^2	*df*	χ^2/df	CFI	RMSEA (90% CI)	PCFI
Model A_3	Full	1837.2	690	2.66	.92	.049 (±.047, .052)	.86
Model A_3	Clinical	1204.8	690	1.75	.93	.050 (±.045, .055)	.87
Model A_3	Non-clinical	1520.1	690	2.20	.89	.056 (±.052, .060)	.83
Model A_3 + EA	Full	2627.1	1067	2.46	.91	.046 (±.044, .048)	.86
Model A_3 + EA	Clinical	1722.2	1067	1.61	.92	.045 (±.041, .049)	.87

Note: 'Model A_3' = final HOF model (as tested in Study 1); 'Model A_3 + EA' = HOF model predicted by experiential avoidance (as tested in Study 1).

To test for mediation, the relative fit of two models was compared for each predictor (AIM-NI and CTQ-SF). The first of these models ('Model FM' (full mediation)) proposed that the effect of the predictor on the criterion was indirect and mediated by the AAQ. That is to say, paths relating the predictor to the mediator, and the mediator to the HOF were included in the model, but a direct path from predictor to the HOF was *not*. The second model ('Model PM' (partial mediation)) evaluated the competing hypothesis of partial mediation, in which a direct path from predictor to the HOF was added. This second model thus proposed direct and indirect effects. Models were statistically compared using a chi square difference test ($\Delta\ \chi^2$), which simply subtracts the $\chi^2_{(df)}$ values of 'Model PM' from 'Model FM' and tests whether the change in χ^2 is significant. If the addition of the direct path *does not* significantly improve model fit, mediation is implied (see section 3.4.4).

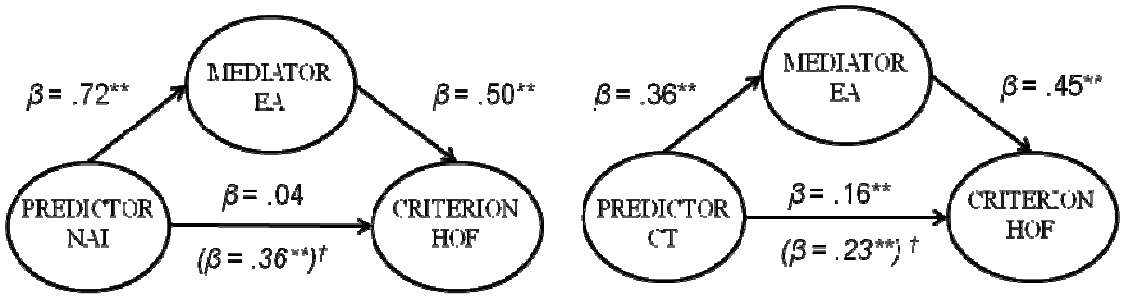
In each model, AIM-NI was modelled as a latent variable with six manifest indicators (AIM-NI questionnaire items), CT as a latent variable with four manifest indicators (CTQ-SF subscales, excluding Sexual Abuse), experiential avoidance as a latent variable with nine manifest indicators (AAQ questionnaire items), and MBs as a HOF with nine first-order factors (MBQ subscales) and 40 manifest indicators (MBQ questionnaire items). In all analyses that follow, factor loadings (paths from latent variable to manifest indicator) for each of the scales were significant at the $p = .01$ level. Furthermore, a large correlated error continued to be implicated between two AIM-NI

items (“My emotions tend to be more intense than those of most people” and “My friends say that I am emotional”). This error was thus freely estimated in all analyses.

5.3.3.1 *Full Sample Analyses.* Models were first tested using the full sample data. AIM-NI was the predictor in the first set of models and CTQ-SF in the second. The overall fit of ‘Model FM’ with AIM-NI as the predictor was adequate. Although χ^2 was large and significant ($\chi^2_{(1362)} = 3187$, $p > .001$), its ratio to the *dfs* was close to the optimal value of 2.00 ($\chi^2/df = 2.33$), CFI was within the acceptable range (CFI = 0.90), RMSEA was good (RMSEA = .044 (±.042, .046)) and PCFI was acceptable (PCFI = .86). As expected, AIM-NI significantly predicted the AAQ ($\beta = .72$, $p < .001$), which significantly predicted the HOF ($\beta = .53$, $p < .001$). Adding the direct path from AIM-NI to the HOF (i.e., Model PM) *did not* significantly improve χ^2 ($\chi^2_{(1361)} = 3186.8$; $\Delta\chi^2 = 0.2$, $p > .05$) and fit indices were unaltered (CFI = .90, RMSEA = .044 (±.042, .046) and PCFI = .86). Moreover, accounting for the indirect effect of the AAQ on the HOF, the direct effect of AIM-NI→HOF was non-significant (Figure 5.2 A). These findings suggest that the effect of AIM-NI on the HOF was mediated by the AAQ.

The overall fit of ‘Model FM’ with CTQ-SF as the predictor was also adequate: although χ^2 was significant ($\chi^2_{(1260)} = 3151.9$), the χ^2/df ratio was close to the optimal value of 2.00 ($\chi^2/df = 2.50$), CFI was within the acceptable range (CFI = .90), RMSEA was good (.047 (±.045, .049)), and PCFI acceptable (.86). Beta values indicated that CTQ-SF significantly predicted the AAQ ($\beta = .38$, $p < .001$), which significantly predicted the HOF ($\beta = .55$, $p < .001$). Adding the direct path from CTQ-SF to the HOF did not alter CFI, RMSEA and PCFI values. This direct path *was*, however, significant (Figure 5.2 A), and χ^2 was significantly improved ($\chi^2_{(1259)} = 3130.3$; $\Delta\chi^2 = 21.6$, $p > .05$). These results suggest that the AAQ partially mediated the effect of CTQ-SF on the HOF.

A



B

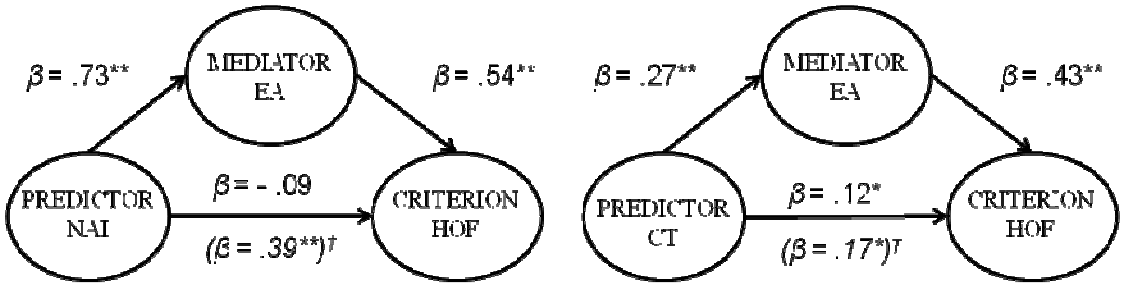


Figure 5.2 Simplified SEM Mediation Models showing Beta Coefficients for Direct and Indirect Effects for the Full Sample (A) and Self-Declared Clinical (B) Subgroup.

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* $p < .05$, ** $p < .001$. † = Beta coefficient for the direct effect of the predictor (NAI, CT) on the HOF when the effect of experiential avoidance is *not* controlled for. CT = Childhood Trauma, NAI = Negative Affect Intensity; HOF = Higher Order factor.

5.3.3.2 *Clinical Subgroup Analyses.* In the clinical subgroup, the overall fit of 'Model FM' with AIM-NI as the predictor was adequate: although χ^2 was large and significant ($\chi^2_{(1362)} = 2138.3$), its ratio to the *dfs* was good ($\chi^2/df = 1.57$) and the CFI value was acceptable (CFI = .91). Furthermore, RMSEA (.044 (±.040, .047)) and PCFI values were good (.87). Beta values indicated that AIM-NI significantly predicted the AAQ ($\beta = .72$, $p < .001$), which significantly predicted the HOF ($\beta = .47$, $p < .001$). Adding the direct path from AIM-NI to the HOF *did not* significantly improve model fit ($\chi^2_{(1361)} = 2137.8$; $\chi^2 \Delta = 0.5$, $p < .05$) and fit indices were unaffected by the addition of this path. Furthermore, accounting for mediated effects of the AAQ, the direct path from AIM-NI→HOF was not significant (Figure 5.2. B).

The overall fit of 'Model FM' with CTQ-SF as the predictor was also acceptable ($\chi^2_{(1260)} = 2040$, $\chi^2/df = 1.62$, CFI = .91, RMSEA = .046 (±.042, .049) and PCFI = .86).

CTQ-SF significantly predicted AAQ (β = .29, $p < .001$) and the AAQ significantly predicted the HOF (β = .48, $p < .001$). Adding the direct path from CTQ-SF to the HOF (i.e., 'Model PM') did not affect fit indices (CFI = .91, RMSEA = .046 (±.042, .049) and PCFI = .86), but again, χ^2 was significantly improved at the α = 0.05 level of significance ($\chi^2_{(1259)} = 2035$; $\chi^2 \Delta = 5$, $p > .05$). Furthermore, the direct path from CTQ-SF→HOF was significant (Figure 5.2 B). Results from the clinical subgroup analyses thus suggest that the AAQ fully mediated the effect of AIM-NI on HOF and partially mediated the effect of the CTQ-SF.

5.4 Discussion

5.4.1 *Study Findings*

Previous research has shown that intense negative affect and childhood trauma are reliably predictive of engaging in MBs. These relationships have been reported by studies using both cross-sectional and longitudinal designs, and 'clinical' and 'non-clinical' samples. These studies have, however, typically focused on the prediction of one type of behaviour in isolation (e.g., alcohol abuse or dysfunctional eating). If childhood trauma and intense negative affect do increase the risk of engaging in MBs, it is important to elucidate the processes through which these factors have their effects. This is because the identification of underlying psychological processes can guide focused treatments designed to target them. Such research will be especially useful if it can identify common processes that underlie a range of clinically relevant behavioural problems. This is not only because these problems commonly co-occur but also because such research may guide parsimonious and focused treatments. With this in mind, this study aimed to test an ACT-derived conceptualisation of how these variables interrelate. It was predicted that individuals who are predisposed to NAI and/or who have experienced CT are more likely to engage in MBs in an attempt to prevent, escape, or reduce contact with unwanted private experiences. A fully elaborated account of these relations was obtained by using two different statistical techniques and by using three different variants of the criterion (i.e., MBQ composite scores, MBQ subscales, HOF).

Before discussing the main mediational analysis in detail, four more general aspects of the data will be considered. Firstly, additional support for the MBQ was obtained with a new sample. Internal reliability remained good. Theory consistent relations

between the MBQ, CTQ-SF and AIM-NI were found. Furthermore, the MBQ was again able to detect significant differences between the clinical and non-clinical subgroups. This was obtained using a more refined definition of 'clinical', which included the measurement of medicinal treatment for a psychological problem. The present findings thus replicate and extend the reliability and validity values of the MBQ that were reported in study 1.

Secondly, consistent with study 1, descriptive statistics suggested that the current method for accessing a clinical group had some validity. Relative to their non-clinical counterparts, these participants were more likely to report CT, to experience intense negative affect, and to have high levels of experiential avoidance. Thus, although there is little detail about the nature of this group's treatment history, data support the idea that they were drawn from a clinical population.

Thirdly, also consistent with study 1, correlations showed that Restrictive Eating and Excessive Exercise tended not to co-vary with the other behaviours. One explanation for this may be that these two behaviours are associated with other variables such as trait-perfectionism and the need for control, both of which are undermined by, for example, intoxication and drug use. Although Restrictive Eating was not found to be co-morbid with other behaviours, it was still associated with high AAQ scores. This pattern of results may thus be indicative of a more complex mediation/moderation model in which other variables, such as perfectionism or impulsivity, moderate the expression of experiential avoidance. Further research into the manifestation of different behavioural topographies is required (see chapter 8).

Fourthly, consistent with study 1, the AAQ tended to be correlated with a greater range of behaviours in the clinical group than the non-clinical group. Although these patterns are merely trends, if established reliably, they could support the proposition that high levels of experiential avoidance relate to a broader range of behavioural topographies. Taking this further, MBs may be more likely to hang together (or co-vary) in groups where experiential avoidance is high. Although speculative, this interpretation is consistent with the SEM analyses, which found that the HOF model adequately fitted the correlation/covariation matrix of the clinical, but not the non-clinical, group. In the non-clinical group, several factor-factor correlations were found, suggesting that covariances could not be accurately modelled by one single factor. Interesting, this was not consistent with the results of study 1, which found that in a predominantly non-

clinical group, the HOF model provided a good overall fit of the data. One interpretation of this finding is that, if high experiential avoidance is a mechanism through which the behaviours hang together, then deliberately sampling a group of individuals with low levels of that variable (i.e., the non-clinical group) would tend to undermine the integrity of the HOF model. This interpretation cannot be verified, however, because other between-group differences that were not measured may also have produced the same outcome.

Now considering the main findings, support for the proposed mediational models was found. AIM-NI and CTQ-SF were both found to be significantly predictive of AAQ and MBQ scores. In keeping with study 1, the AAQ was also significantly predictive of the tendency to engage in MBs. In fact, the present study found that, as compared to study 1, the AAQ predicted a far greater amount of HOF variance. This may be accounted for by demographic differences. For example, relative to study 1, the present sample had a greater proportion of 'clinical' respondents, a smaller proportion of student respondents, and a tendency towards greater experiential avoidance.

With regard to the mediational role of experiential avoidance, both regression and SEM analyses suggested that the AAQ accounted for a meaningful proportion of the effect of AIM-NI and CTQ-SF on most of the behavioural criterion variables (i.e., MBQ composite scores, independent subscales and HOF). Broadly speaking, the AAQ tended to fully mediate the effect of AIM-NI and partially mediate the effect of CTQ-SF on individual and co-occurring behaviours. These findings thus support the conclusion that experiential avoidance is an important variable for understanding how MBs become established and maintained. Bearing in mind the limitations of the present study's cross-sectional design, results are supportive of the suggestion that people who experience negative affect intensely are more likely to rely on avoidance strategies to try to escape or modify that experience. This tendency to engage in experiential avoidance, rather than the experience of intense negative affect per se, appeared to contribute to the development of MB patterns. Similarly, for the case of CT, results supported the hypothesis that early traumatic experiences affect future risk taking, in part, because they foster heightened experiential avoidance. It therefore seems reasonable to suggest that, consistent with Hayes et al.'s (1996) theorising, MBs can be usefully understood as a behavioural class, which is negatively reinforced by its ability to provide temporary relief from unwanted private experiences.

5.4.2 *Methodological Limitations*

Several methodological limitations of the present study must be taken into account. Most importantly, cross-sectional research cannot determine causation. Although the constructs on which the predictor measures were based have clear antecedent status, cross-sectional research cannot unpack the temporal relationships between them and mediator/criterion variables. Complementary longitudinal research (e.g., using latent growth curve analysis; see Hoyle, 2008) may be a suitable method for confirming whether these causal paths hold when tested across time. Although this is a particularly challenging task, natural experiments could provide one possible method. For example, Rutter and his colleagues have conducted a series of longitudinal studies following a cohort of English and Romanian Adoptees (ERA) from early childhood to adolescents (e.g., Rutter, 2004). This design has enabled them to track the effect of early childhood experiences on later functioning in a manner that exhibits acceptable internal validity.

A second limitation for consideration is the reliance on self-report data. Although this measurement approach is methodologically justifiable, it is subject to many sources of bias (see section 3.2.1). This is an inevitable limitation shared by most published research investigating childhood trauma. The final limitation concerns sample bias. These findings were again obtained from a predominantly female sample. Furthermore, community sample participants were restricted to those individuals with access to the internet. Similarly, the clinical subgroup was 'self-declared' (although demographically it resembled a true clinical sample). Given that the model was supported in this sample, future research should now extend it, in a more confirmatory way, to a better defined clinical group. This could include, for example, a group currently awaiting treatment from the psychological services.

5.4.3 *Implications*

The current findings contribute to a growing evidence base that identifies concepts of avoidance and escape from private experience as central to the understanding of MBs (e.g., Batten et al., 2001; Cooper et al., 2003; Hayes et al., 2001; Hayes et al., 1996). These findings are the first, however, to suggest that such a wide range of behavioural topographies share a common experiential avoidance function. Moreover, they are the first to identify experiential avoidance as a mechanism through which risk factors affect

comorbid risk taking. For this reason, the present analogue study has substantial implications for clinical practice.

Personality predispositions and aversive childhood experiences are particularly difficult, if not impossible, to modify in treatment. For example, personality predispositions are rigid and resistant to change, especially in PD populations (Beck, Freeman, & Davis, 2001). Similarly, past experiences cannot be altered. If experiential avoidance is one of the vehicles through which these risk factors impact on maladaptive behavioural patterns, as the present data suggest, any technique designed to reduce it is, at least in theory, likely to reduce MBs. For example, teaching patients to experience negative affect without attempts to escape or modify those experiences (e.g., acceptance techniques) should in principle reduce the occurrence of MBs. A similar effect should also occur if the associative link between idiosyncratic unwanted private experiences and behavioural engagement can be extinguished (e.g., mindfulness techniques). Because ACT can effectively reduce experiential avoidance, it presents itself as a good candidate treatment for reducing co-occurring MBs. Similarly, other treatments that aim to break down the link between internal experiences and mindless reaction to those experiences should also, in principle, have valuable clinical effects. Early data supports this proposition. For example, preliminary data suggests that mindfulness-based and acceptance-based techniques are useful for reducing the occurrence of substance misuse and DSH (e.g., Hayes, Wilson, et al., 2004; Gratz & Gunderson, 2006; Marlatt et al., 2004).

5.4.4 *Summary*

Results from the present study suggest that experiential avoidance is an important variable for understanding how NAI and CT affect the tendency to engage in MBs. These finding add to research that has similarly established the avoidance of unwanted internal experiences as a key variable for understanding the relationship between aversive situational antecedents (e.g., parental criticism, sexual victimisation, adverse life event), risky trait-like predispositions (e.g., heightened emotionality, impulsivity, anxiety sensitivity), and a range of behavioural problems (see section 2.1.5). These behavioural problems have included, for example, BPD symptoms (Cheavens et al., 2005), global psychiatric distress (Marx & Sloan, 2002; Polusny et al., 2004), coping-

motivated drinking (Stewart et al., 2002), PTSD (Marx & Sloan, 2005), and suicidal ideation (Lynch et al., 2004). Together, these findings suggest that experiential avoidance is an important variable for understanding several psychological problems. It thus seems reasonable to propose that ACT may be a useful treatment for a heterogeneous group of patients with complex and entrenched disorders (i.e., treatment resistant patients). This is by virtue of the fact that excessive experiential avoidance should, in theory, underpin the many different psychological difficulties that this group present with. A logical next step, based on the 4-stage methodological guidelines discussed in chapter 3, could thus be to pilot test ACT (Hayes et al., 1999) for this group. This was the aim of study 3.

CHAPTER VI

Study 3. A Pre-Post Pilot Uncontrolled Trial Investigating ACT for a Heterogeneous Group of Treatment Resistant Patients

6.1 Introduction

Experiential avoidance has been the primary focus of the previous two studies. These studies, in conjunction with existing research (see section 2.1.5 and 6.1.1), suggest that excessive levels of experiential avoidance may maintain several topographically dissimilar psychological problems. It thus logically follows that ACT, which aims to reduce experiential avoidance, should be able to produce good outcomes for patients with complex, entrenched and co-morbid psychological problems. This should be the case even when the topography of those problems differs across participants (e.g., the group is symptomatically heterogeneous). This is because the heterogeneous symptoms of this group should, according to ACT-theory, be commonly maintained by high levels of experiential avoidance (e.g., Hayes et al., 1996). Based on the methodological guidelines discussed in chapter 3, the aim of this study was to pilot test the novel application of ACT to a heterogeneous group of treatment resistant patients. That is, patients who failed to benefit from, or relapsed following, previous psychological treatment.

6.1.1 *ACT for Treatment Resistant Patients*

The link between ACT and treatment resistance is not simply a logical one. Rather, several studies have implicated an important connection between experiential avoidance and treatment resistance. For example, studies on BPD, arguably the most treatment resistant of diagnostic groups (e.g., see Lieb et al., 2004), have suggested that experiential avoidance may play a primary role in entrenched and hard-to-treat symptoms. For example, Rosenthal et al. (2005) reported that the tendency to suppress thoughts fully mediated the relationship between NAI and BPD symptoms. Similarly, in a BPD sample, Gratz, Tull, and Gunderson (2008) found that controlling for the effect of impulsivity and NAI, experiential avoidance was the only significant predictor of BPD symptoms. The results of study 1 and 2 add to this literature, implicating experiential avoidance as an important variable for understanding MBs, which are

known to commonly co-occur in treatment resistant patients (see chapter 1). Complementary of these findings, research has also found that excessive avoidance of internal experiences is associated with chronic and/or co-morbid symptoms (e.g., Begotka, Woods, & Wetterneck, 2004; Forsyth et al., 2003; Roemer, Salters, Raffa, & Orsillo, 2005), poor treatment outcomes (e.g., Hayes, Beevers, Feldman, Laurenceau, & Perlman, 2005; Roemer Litz, Orsillo, & Wagner, 2001) and vulnerability to relapse (e.g., Moos & Moos, 2006; Salkovskis & Reynolds, 1994). For example, investigating the effect of experiential avoidance on relapse in a sample of alcoholics, Westrup (1999; cited in Chawla & Ostafin, 2007) reported that the use of avoidance strategies during stressful life events was uniquely predictive of relapse, above and beyond the effect of the stressor itself. Together, these findings implicate a link between experiential avoidance, symptom chronicity, treatment resistance, and relapse vulnerability.

In keeping with these implied links, a few trials suggest that ACT and ACT-like techniques may be effective for treatment resistant patients. Firstly, in a treatment resistant group of polysubstance abusers, Hayes, Wilson, et al. (2004) found that, compared to methadone maintenance and a 12-step facilitation programme, ACT was associated with lowest drug use 6-months after therapy ended. Similarly, Gratz & Gunderson (2006) conducted a pilot RCT (N = 24), which delivered a hybrid of ACT-DBT to self-harming BPD patients. They reported that, relative to TAU, ACT-DBT+TAU produced significant reductions in DSH, psychological distress and BPD symptoms. These positive findings were found even though the intervention was group-based *and* time limited (14 weeks). Thirdly, Dimidjian et al. (2006) conducted an RCT comparing CBT to an ACT-like intervention (Behavioural Activation (BA), see section 2.3.3). Although treatment effects were comparable for patients with mild depression, patients with more chronic symptoms (N = 61) demonstrated significant improvements following BA and medication *but not* CBT. Ma and Teasdale (2004) have similarly reported that the effects of MBCT are moderated by baseline chronicity, finding that MBCT obtained better effects for participants with a chronic history of recurrent depression compared to those with less chronic histories.

To summarise: theoretically-orientated analyses of experiential avoidance and a small number of acceptance-based outcome trials converge to suggest that ACT could be a promising treatment for treatment resistant patients, even when delivered as a time-limited and group-based intervention. It can thus be predicted that ACT will

significantly improve the global psychological functioning of this group, as evidenced by improvements in psychological symptoms, quality of life, and the tendency to engage in MBs (see section 6.1.2). Furthermore, based on the ACT model, it can be predicted that these effects will be mediated by reductions in experiential avoidance and cognitive fusion, and increased mindfulness and commitment to valued action (see section 2.1). Changes in the mere frequency of unwanted private experience should, however, be *un*related to outcome. The methodological considerations for exploring these predictions are discussed below.

6.1.2 *Methodological Considerations*

The mere fact that ACT has not previously been evaluated for treatment resistant patients informs many of the decisions regarding the design of such a trial. Chapter 3 discussed pilot trials as a recommended method for evaluating novel treatments. Pilot trials deliver treatment to a small clinical sample, aiming to establish whether it holds promise as a plausible treatment for the target group. This approach is more ethical and economic than piloting a treatment on large samples using powered and controlled trials. It also provides an opportunity to refine techniques and to develop treatment manuals. A variety of designs can be used (see section 3.3.2), of which the pre-post uncontrolled trial is particularly popular (e.g., Bohus, et al., 2000, N = 24; Dimeff, Rizvi, Brown, & Linehan, 2000, N = 12; Telch, Argas, & Linehan, 2000, N = 10). This prospective experimental design enables one to explore the effects of an integrated treatment package on a number of patients concurrently. It is therefore suited to pilot testing group-based treatments. Thus, in keeping with previous research, a pre-post uncontrolled design was used.

Pilot trials typically evaluate treatment outcomes using self-report measures. These are favourable because they are unobtrusive, inexpensive, and easy to administer. They also enable direct comparisons with other published trials. Furthermore, the use of well-validated diagnostic measures helps others to replicate and extend the research (see Ost, 2008). A particular challenge in piloting ACT for a heterogeneous group of patients is obtaining outcome measures that are applicable and sensitive to the variety of symptoms this group present with. Furthermore, because ACT aims to construct a new repertoire of valued action, and not merely to reduce symptoms, measures sensitive to both these

domains are necessary for obtaining a detailed account of treatment effects. Research on ACT mechanisms of change has tended to exclusively focus on experiential avoidance. This is partly because no validated measures have been developed for the measurement of self-as-context, valued living, and cognitive fusion. The present study aimed to explore these other mechanisms of change, but thus necessarily relied on un-validated measures.

Piloting a new treatment raises two further methodological issues. Firstly, treatment guides have usually not been developed for the patient group in question. This was overcome in the present study by adapting an existing self-help manual so as to fit a group-based and generic treatment approach (see section 6.2.4.2). Secondly, by definition, pilot trials are conducted before data exists to suggest that the treatment will be effective for the target group. It is therefore prudent to exclude the most 'at-risk' of patients from pilot investigations (see section 6.2.2).

6.2 Method

6.2.1 *Design*

A pre-post, uncontrolled, pilot trial was used. The independent variable was a 16-week, group-based ACT intervention, which all participants attended. The dependent variables (outcome measures) included a measure of global symptom severity, quality of life, PD symptoms, depression, and alcohol misuse. Mediator variables included a measure of experiential avoidance, thought frequency and believability, valued living and mindfulness. Outcome and process variables were measures at baseline (T1), post-treatment (T2), 6-month follow-up (T3) and 12-month follow-up (T4) and repeated measures, within group comparisons were made.

6.2.2 *Participants*

Ethical approval was obtained from the Local Research Ethics Committee (LREC) before any participants were recruited to the trial. The inclusion criterion was: "patients who had already received *at least* one previous episode of psychological treatment, lasting for at least 8 sessions, and who were currently being re-referred to the adult mental health services with significant residual mental health complaints". This

definition was consistent with current research (e.g., Amsterdam et al., 2001; Kenny & Williams, 2007). The exclusion criteria were: (a) a current drug or alcohol dependency problem (defined using the DSM-IV dependence criteria), (b) schizophrenia or other psychotic disorder, (c) Anorexia Nervosa *and* a BMI of < 16, (d) a learning disability, or (e) DSH in the previous 6 months (defined using Kreitman's (1977) criteria[19]).

Treatment resistant patients were recruited from GP referrals to the General Adult Mental Health Services (The Chines) and from the waiting list for treatment at The Chines and the Intensive Psychological Therapies Service (IPTS). The IPTS is a specialised tertiary service for patients with entrenched problems reaching diagnostic criteria for a PD[20]. Figure 6.1 depicts the flow of participants through recruitment and testing stages of the trial. The details of 118 treatment seeking patients were reviewed (see section 6.2.4), of which 24 met the inclusion criteria and 14 consented to the trial. Four participants dropped out of the trial leaving a total N = 10 (see Table 6.1 for demographic information, Table 6.3 for baseline means and standard deviations and Appendix B for more detailed information on each participant). Patient records showed that two patients had a history of chronic DSH, three of Anorexia Nervosa, one of Bulimia Nervosa, two of illicit drug use, and six reported high levels of alcohol use (not reaching dependency criteria). Eight participants described at least one of their previous treatments as "Cognitive Therapy" or "Cognitive Behaviour Therapy" in nature.

6.2.3 *Materials*

Ethical approval for this study was obtained prior to the completion of study 1 and 2. Because the LREC required that all measures used in this trial had been piloted and validated, the MBQ (see study 1) could not be used. Patient records were thus used to obtain an account of the patient's tendency to engage in MBs (see section 6.2.2) and the Alcohol Dependency subscale of the Millon Mutliaxial Clinical Inventory III (MMCI-III; Millon, 1994) was used as an outcome measure (described below)[21].

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[19] "(1) Nonfatal, intentional self-injurious behaviours resulting in actual tissue damage, illness, or risk of death; or (2) any ingestion of drugs or other substances not prescribed or in excess of prescription with clear intent to cause bodily harm or death."

[20] It is usual practice for The Chines to refer more chronic patients to the IPTS.

[21] The MCMI-III was used to index alcohol use because it is standard practice at the IPTS for all patients to complete this measure as part of clinical audit.

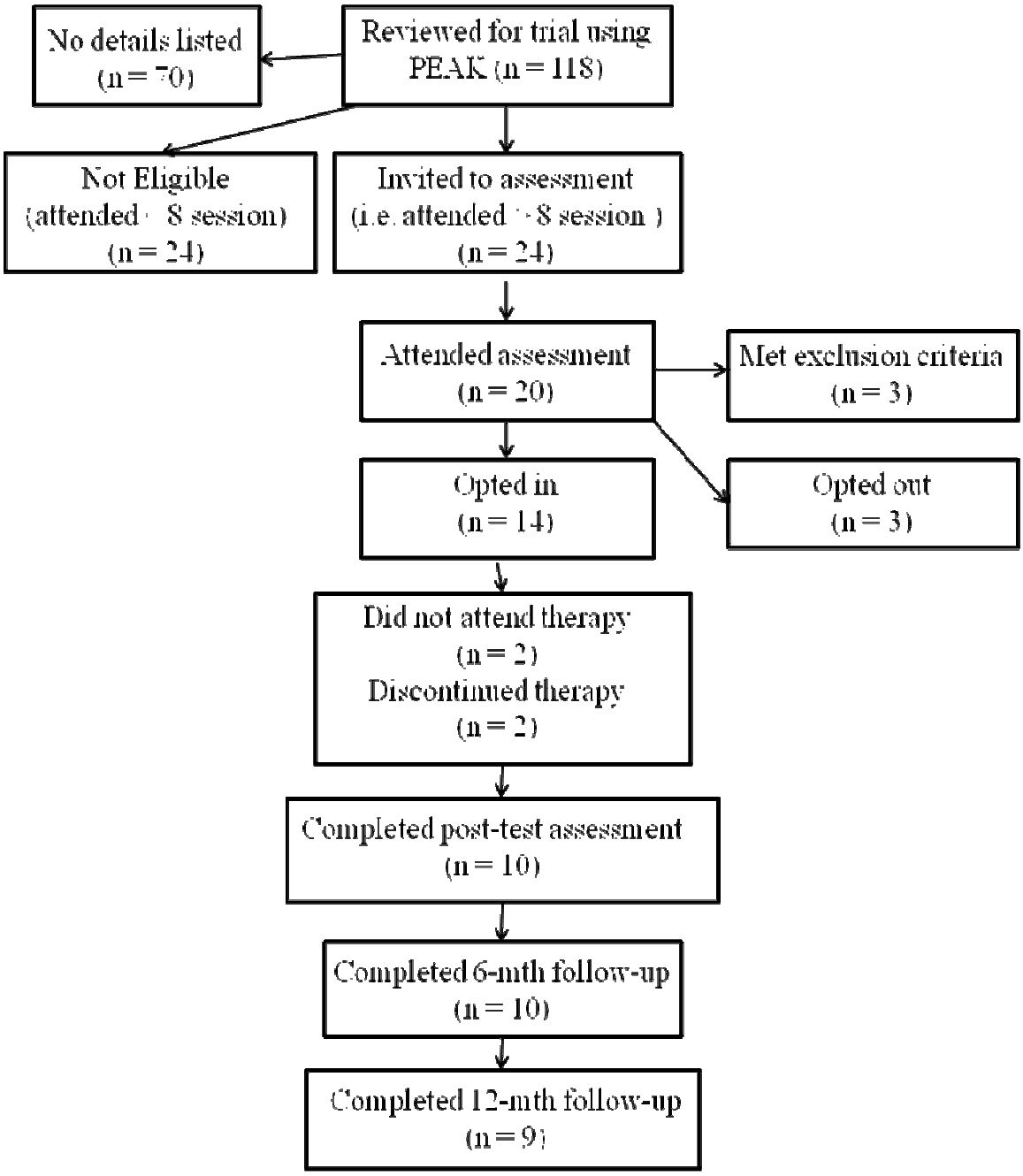


Figure 6.1 Flow Chart of Patient Recruitment to the Trial.

Note: PEAK is a DHFT database used to review the patients' history of psychological treatment (see section 6.2.4.1)

Table 6.1

Demographic and Baseline Statistics of Treatment Completers

Demographics/Baseline Symptoms	N = 10
Mean Age (years)	41
Gender (% female)	90%
Currently working or in education*	40%
Medication (% yes)	80%
Mean no. previosu therapeutic episodes (range)	3.00 (2-6)
Mean no. previous sessions (range)	78 (14-300)
Median no. months since last treatment (range) **	24 (2-84)
Clinical range for depression (%)	90%
Clinical range for GSI (%):	90%
Clinical range for 1 SCL-90 domain	30%
Clinical range in up to five SCL-90 domains	60%
Clinical range in six or more SCL-90 domains	10%
PD Lifetime Criteria (%)	50%

*At start of ACT groups. **Calculated from end of last treatment to start of ACT group.

6.2.3.1 *Primary Outcome Measures.* The following outcome and process measures were completed at all assessment periods (i.e., T1, T2, T3, and T4) except the SCID-II, which was administered at T1 and T3 only.

The *Revised Symptom Check List -90* (*SCL-90;* Derogatis, 1993). This 90-item, self-report inventory measures nine acute psychiatric symptoms (Somatisation, Obsessive-Compulsive Disorder, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychotocism). Items, rated on a 5-point scale (0 – not at all to 4 – extremely), measure levels of distress in each domain over the past 7 days (e.g., "How distressed were you by having urges to beat, injure, or harm someone?" for Hostility). The Global Severity Index (GSI), used here as a primary outcome measure, is the mean score across acute disorders. The psychiatric outpatients' GSI Mean = 1.26; SD = 0.68, and the non-psychiatric Mean = 0.31; SD = 0.31 (Detogatis, 1993). The GSI has been well validated and shows good stability over time (r = .91; Derogatis, 1993).

The *World Health Organisation Quality of Life (WHOQOL;* Skevington, Lofty, & O'Connell, 2004). This measure is a 26-item, cross-culturally comparable device for assessing four domains of quality of life: Physical Health, Psychological Health, Social Relationships, and Environment (e.g., "How satisfied are you with yourself" for Psychological Health). Each domain includes 3-8 items, rated on a scale ranging from 1 (not at all/very poor) to 5 (an extreme amount/very good). Scores are transformed for compatibility with WHOQOL-100, and high scores indicate good quality of life. Respectable psychometric properties have been reported in both clinical and non-clinical samples (e.g., α = .97; Skevington, Carse, & Williams, 2001). Hawthorne, Herrman, and Murphy (2006) reported the following non-clinical norms: Physical Health (Mean = 73.5; SD = 18.1), Psychological Health (Mean = 70.6; SD = 14), Social Relationships (Mean = 71.5; SD = 18.2), Environment (Mean = 74.83; SD = 13.0), and total WHOQOL (Mean = 72.61; SD = 15.83). Clinical norms are not yet available. Test retest reliability values indicate acceptable stability over a 4-week delay (r = .71 to r = .92; Taylor, Myers, Simpson, McPherson, & Weatherall, 2004; Naumann, & Byrne, 2004, respectively).

The *Beck Depression Inventory-II* (*BDI-II*; Beck, Brown, & Steer, 1996). The BDI-II is a 21-item self-report inventory measuring attitudes and symptoms characteristic of depression. Each item has four different phrasings of increasing intensity (e.g., "I don't feel I'm being punished" (0) /"I feel I may be punished" (1) /"I expect to be punished" (2) /"I feel I am being punished" (3)). Scores thus range from 0-63. Authors reported good internal consistency (α = .86) and split-half reliability values (α = .93). Dozois, Dobson, and Ahnbery (1998) reported a non-psychiatric Mean = 8.9; SD = 12.36, and Beck et al., (1996) reported a psychiatric Mean = 22.45; SD = 12.75. High test retest values have been reported: r = .93 (Beck et al., 1996), r = .96 (Sprinkle et al., 2002).

The *MCMI-III Alcohol Dependency Subscale* (Millon, 1994). The MCMI-II is a 175-item questionnaire that measures 14 personality disorders and 10 Axis I syndromes based on DSM-IV classification system. The Alcohol Dependency subscale (ADS) was used in this trial as an indicator of alcohol misuse. This subscale is measured using 15-items (e.g., "I have a great deal of trouble trying to control my impulse to drink in excess") that are rated using a 'true/false' response format. Scores of greater than 85 indicate clinical levels of symptomatology and scores greater than 75 indicate a trait tendency. Good psychometric properties have been reported by the authors.

The *Structured Clinical Interview for DSM-IV Axis II Disorders* (*SCID-II*: First, Spitzer, Gibbon, & Williams, 1996). The SCID-II is a 140-item, semi-structured interview organised by DSM-IV PD diagnosis. It thus measures: Avoidant, Dependent, Obsessive-Compulsive, Passive-Aggressive, Depressive, Paranoid, Schizotypal, Schizoid, Histrionic, Narcissistic, Borderline, and Antisocial PD. Items are rated as '1' – symptom not present, '2' – threshold, and '3' - symptom present. The number of '3' ratings per PD indicates its presence or absence. Clinical cut-offs differ across subscales. The SCID-II has shown adequate internal consistency (mean α across subscales = .82, range .61 to .97; see Maffei, et al., 1997) and inter-rater reliability values have varied from kappa .53 to .80 (see First et al., 1996). I was trained by reviewing the SCID-II manual (Spitzer, Wiliam, Gibbon, & First, 1989), reviewing and rating SCID-IIs previously administered by IPTS clinicians, and role-play. I was trained to a level of 80% concordance with IPTS clinicians' ratings. Moreover, 20% of all SCID-IIs administered in the course of this research were second rated by two independent assessors (using audio recordings). Inter-rater reliability for 'PD present versus absent' was kappa = .72 (92% agreement). Inter-rator reliability for '3' ratings (i.e., symptom present versus threshold or absent) was kappa = .61 (82% agreement). These values are acceptable relative to published research (see First et al., 1996).

6.2.3.2 *Process Measures.* Four process measures were administered.

The *Mindful Attention Awareness Scale* (*MAAS;* Brown & Ryan, 2003). This 15-item, self-report inventory measures the frequency of daily mindful states (e.g., "I find myself doing things without paying attention"). Items are rated on a scale ranging from 1 (never) to 6 (always) and high scores indicate high mindful awareness. The MAAS has shown adequate psychometric properties (α = .86, 4-week test retest reliability *r* = .81) and has been validated in clinical and non-clinical samples (Brown & Ryan). Authors reported a non-clinical Mean = 59.6 (SD = 9.6), and in a mixed group of psychiatric outpatients, Ree and Craigie (2007) reported Mean = 49.2 (SD = 17.1).

The *Thought Frequency and Believability Questionnaire* (TFQ/TBQ). This instrument, adapted from Bach and Hayes (2002), measures the frequency and believability of unwanted/intrusive thoughts and feelings. Respondents rated: (a) How *frequently* they have experienced unwanted/intrusive thoughts/feelings in the past week

(1 - never to 7 - almost constantly), and (b) How *believable* and *meaningful* those thoughts/feelings were (0 - not at all real and/or meaningful to 10 - very real and/or meaningful; see Appendix C). No norms are available.

The *AAQ* (Hayes, Stroshal, et al., 2004). The AAQ (described fully in study 1) is a 9 item measure of experiential avoidance. Norms for non-clinical populations are reported to range from Mean = 3.57 to Mean = 3.90; SD = 0.82 (Hayes, Stroshal, et al., 2004). Authors also reported M = 4.66 as an upper quartile score for clinical populations.

The *Valued Living Questionnaire* (*VLQ;* K. Wilson, *unpublished*). The VLQ is a 10 item questionnaire which measures how consistent a participant's behaviour is with 10 pre-defined valued domains; family, marriage, parenting, friends, work, educational training, recreation, spirituality, citizenship, and physical well-being. Participants' rate how important each domain is to them (0-not at all important to 2-very important) and how frequently, in the last week, they have acted in a way that is consistent with that value (0-not at all to 3-more than four times; see Appendix C). Scores are computed by subtracting value consistency from value importance, producing a discrepancy score. No psychometric data were available for this measure.

6.2.3.2 *Get out of Your Mind and into Your Life* (Hayes & Smith, 2005). The design of the intervention was based on the chapters of this self-help work book (see section 6.2.4.3). Homework activities were also drawn from this resource.

6.2.4 *Procedure*

6.2.4.1 *Recruitment.* A timeline of the study procedure is depicted in Figure 6.2. On a weekly basis, over two 4-month periods, I reviewed GP referral letters directed to The Chines, followed up referrals from The Chines and IPTS, and reviewed waiting lists from both sites. The PEAK database[22] was used to establish whether potential participants met the inclusion criterion. Patients meeting this criterion were sent an information pack (see Appendix D) that described the aims of the study and invited them to an individual assessment at the IPTS. This was conducted by an ACT-trained

[22] PEAK is a DHFT database that stores patients' involvement with the psychological services. PEAK shows whether a patient has had previous therapy and, if so, when, with whom, and number of sessions attended.

clinician and was designed to establish whether the patient fulfilled any exclusion criteria. Patients meeting the necessary criteria were invited to join the trial and offered a week to consider whether they would like to take part. Consenting participants either completed a consent form at this session or at the pre-intervention assessment (see section 6.2.4.2). The GPs of consenting participants were sent information about the trial via post (see Appendix D).

6.2.4.2 *Pre-Intervention Assessment.* Consenting patients received questionnaire packs by post and were booked in for a formal, pre-intervention interview (with myself). This interview began by obtaining information on the patient's treatment history, and this information was supplemented by details from their file and the PEAK database. The SCID-II was then administered to assess (a) the patient's lifetime experiences of PD symptoms (diagnostic criterion) and (b) symptoms for the 12-months immediately preceding treatment (symptomatic criterion). This distinguished between lifetime history and recent PD symptoms, thus providing a baseline against which post-treatment interviews could be compared.

6.2.4.3 *Intervention.* Two ACT treatment groups were run sequentially (see Figure 6.2). The first group (N = 6) was run by two ACT trained Consultant Clinical Psychologists (therapist one and therapist two) [23] and the second (N = 4) by an ACT trained Consultant Clinical Psychologist (therapist one) and an ACT-trained Occupational Psychologist (therapist three). Clinicians received regular telephone supervision from an author of the original treatment manual (Dr. Kelly Wilson), who provided feedback on audio recordings of group sessions and verified that the content was ACT.

ACT groups consisted of 16, 2.5 hour group sessions with a 20 minute break. The content of weekly/bi-weekly sessions was informed by the self-help work book 'Get out of Your Mind and into Your Life' (Hayes & Smith, 2005). A synopsis of the content of weekly sessions is depicted in Table 6.2. For a more detailed account and in-session examples, refer to Appendix E.

[23] Therapist one was Professor Susan Clarke (co-supervisor of the PhD).

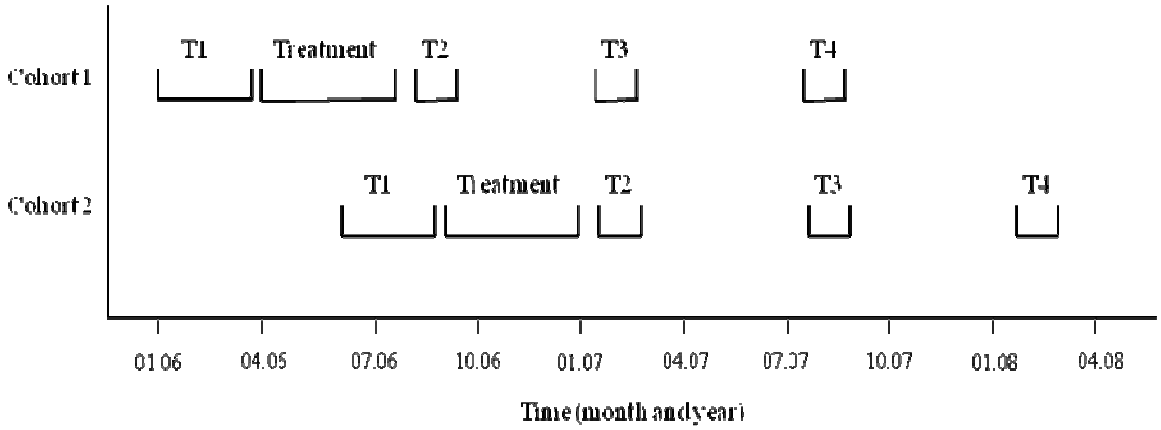


Figure 6.2 Study Timeline

Note: T1 = pre-treatment, T2 = post-treatment, T3 = 6-month follow-up, T4 = 12 month follow-up.

The first half of all session began with a mindfulness activity followed by a review of the exercise and a review of the previous homework tasks. Mindfulness activities differed each week. Early sessions aimed to develop mindful awareness of internal sensations such as mindfulness of the breath, of sights, and of touch. Latter sessions aimed to develop mindful awareness of thoughts, emotions, and memories. Early sessions were fully guided by clinicians, but latter sessions were more prompted than guided. In reviewing these exercises, participants offered to discuss their experience rather than be addressed in turn. The second half of each therapy session focused on a weekly theme, involved didactic and experiential learning, and closed with a reflective review and homework setting.

The first stage of treatment was concerned with *Creative Hopefulness* (see Figure 2.3, section 2.2.1). The clinicians' focus in this stage was to explore the patients' psychological difficulties and to help them recognise that they have been relating to these difficulties as if they can, and must, be controlled and eliminated (Hayes et al., 1999). Through a process of Socratic questioning, requiring the participant to reflect on the short and long term effectiveness of this approach, this stage was designed to undermine the participants' faith in this 'control and eliminate' strategy. For example, "suffering inventories" were used to (a) elicit the participants' key psychological difficulties, (b) to explore the strategies they have used to manage those difficulties, (c) to identify how much energy and time they have invested in managing them, and (d) to consider how effective those strategies have been. Group discussions also aimed to

convey the idea that human suffering is ubiquitous and a natural consequence of language processes. By socialising participants to a simplified version of the RFT mode, clinicians aimed to demonstrate that attempts to problem solve, avoid, or control psychological pain is logical, but may not be effective in the long term.

Stage one led naturally into stage two, which explicitly identified *Control as the Problem*. This stage aimed to expose experiential avoidance as a logical, but counterproductive means of dealing with psychological pain. Experiential avoidance was explored as a phenomenon that may (a) amplify psychological difficulties and (b) inhibit valued living. For example, exercises were used to demonstrate the paradoxical effects that can arise from trying to control thoughts and feelings. Group discussions, with the aid of suffering inventories, were used to explore how these ideas related to participants' own experiences. This stage of treatment also introduced the idea of "letting go" of struggles against private experience and accepting that experience for what it is, rather than entanglement with the functions it has acquired.

Stage three focused exclusively on *acceptance* and *defusion*. These sessions aimed to help participants to observe their thoughts and feelings as events of the mind that can influence, but that do not control, behaviour. Participants were guided through the process of treating their thoughts as thoughts, their emotions as emotions, and their memories as memories; rather than events to be feared and avoided. In-session exercises explored how individuals find it difficult to separate themselves from their thoughts and feelings. Acceptance and de-fusion techniques were used to undermine the perceived literality of thoughts and their apparent correspondence with reality. Exercises using metaphors such as 'The Passengers on the Bus' (see Appendix E) were also deployed to convey the possibility that patients could determine the direction of their life, even in the presence of psychologically difficult private events.

Table 6.2

Breakdown of Treatment Session Content

Treatment Stage	Week	Session name	First Half	Second Half
1. Creative Hopelessness	1	*The Ubiquity of Human Suffering* (p. 9)	Introductions, ground rules, group commitment	Focus: Drawing out the "control and eliminate" agenda. **Suffering Inventories** (p. 14): What is psychologically painful for the patient? What strategies have they tried to manage this pain? Based on their experience, how well have these strategies worked in the short and long term? Does it help them to live the life they most want?
	2	*Why Language Leads to Suffering* (p. 17)	Mindful awareness of breathing	Focus: Demonstrate that psychological pain is 'normal' not 'abnormal'; a product of natural language processes. Discuss how attempts to avoid, eliminate, or control psychological pain makes sense logically but may not be effective in the long term. **Coping Strategies Inventory**
2. Control is the Problem	3	*The Pull of Avoidance* (p. 33)	Mindful awareness of sight	Focus: Undermining investment in avoidance and logic. Demonstrate that experiential avoidance paradoxically amplifies psychological pain and inhibits valued living. Use the participants experience (e.g., suffering inventories) to explore this concept. Describe and discuss different 'rules' for external and internal events: "if you don't like it get rid of it" versus "If you don't want it you've got it." Consult with the patients' experiences. **Riding the Mind Train**, **Thought Controlling**
	4	*Letting Go* (p. 43)	Mindful awareness of sound	Focus: Introduce 'acceptance' as an alternative to experiential avoidance. Discuss what acceptance is and is not. Create exercises to test acceptance versus EA (e.g., **To Be Willingly Out Of Breath)**. Prompt participants' awareness to consult with their direct experience. What does their *experience* say and what does their *mind* say**. The Tug-of-War metaphor** (Appendix E).

3. Acceptance and Defusion	5	*The Trouble with Thoughts* (p. 53)	Mindful awareness of touch	Focus: Describe cognitive fusion; when thoughts and the events that they refer to are treated literally, as if they were the same. Guide participants to noticing the act of thinking; catching the process in flight. Guide participants in feeling memories as memories, thoughts as thoughts etc. Prompt participants to begin to notice the flow of their thoughts and to notice those they fuse with. **Watching the Mind Train.**
	6-7	*Having a Thought versus Buying a Thought* (p. 69)	Mindful awareness of taste	Focus: Looking *at* our thoughts rather than *from* our thoughts. Develop experiential awareness of cognitive fusion and defusion. **Milk, milk, milk. Virtues of Saliva. Labelling Your Thoughts:** "I'm having the thought that_____" "I am noticing the urge to____". Physicalising pain. Must pain be the enemy? Create experiential awareness of the longstanding costs of fusion. **Passengers on the Bus. Buying Thoughts.**
4. Defining the Self	8	*If I'm Not My Thoughts Then Who Am I?* (p. 87)	Mindful awareness of thoughts	Focus: Elicit the key self-conceptualisations that participants' typically fuse with: "I am the type of person who____". Discuss how these might constrain personal development**.** Provide metaphors that allow participants to distinguish between 'self-as-content' and 'self-as-context': the self as the context in which thoughts occur. Practise defusion. Describe other senses of self. Provide exercises that allow participants to experience all three aspects of the self: the self as an on-going process, the observing self, and the verbal self. **The Chess Metaphor, The Rock Metaphor**. Practise defusion from self-as-content.
	9	*Mindfulness* (p. 105)	The Breathing Space and Unguided Mindfulness	Focus: Discuss what mindfulness is and is not and the value of being awake to one's experiences, non-judgementally. Discuss the ways that the participants could integrate mindfulness into everyday living. **Self-judgements:** List your ten favourite judgements – mindfully notice what shows up psychologically for you.

	10	*Willingness* (p. 121)	Unguided Mindfulness	Focus: Learning to say 'Yes' even if your mind says 'No'. Discuss the possibility of saying 'Yes' to a universe of internal experience when that takes you in a valued direction. Help participants identify what there is to be accepted? Discuss what willingness is and is not.
5. Values	11	*Learning to Jump* (p. 133)	A Brief Body Scan	Focus: The Willingness Question: Are you willing to feel, think, sense, and remember private experiences, fully and without defence, as you directly experience them to be, not as what your mind says they are *and* do whatever it takes to move you in the direction that you truly value? Discuss: the choice to say yes and to say no. Consider the pro's and con's of saying yes versus no. What does the participants' mind have to say about willingness? **The Willingness Scale**
	12	*What are Values?* (p. 153)	Mindful awareness of Movement	Focus: Core values, what does the participant want their life to be about? Discuss values as chosen life directions, a compass to guide action. **Passengers on the Bus** metaphor**.** Explore differences between choices based on values and choices based on reasoned judgements.
	13	*Choosing Your Values* (p. 165)	The 3 minute Breathing Space	Focus: Arriving at core values. **Attending your own funeral** exercise- What do you want your life to have meant? The ten valued domains.
6. Committed Action	14	Committing to it (p. 177)	The 3 minute Breathing Space	Focus: Being willing to accept whatever private experiences show up in the service of valued action. Provide exercises to practise committed action. Help the participants to set goals. Identify and discuss possible barriers.
	15	*The Choice to Live a Vital Life* (p. 195).	Unguided mindfulness	Focus: Actively choosing between the familiar, logical, avoidant path and a new, accepting, defused, and valued path. Is the vulnerability and risk in the service of treading a new path something the participant is willing to experience? Discuss taking responsibility for action and change.
	16	*Commitments*	Focus: Making a public commitment to life changes in the service of a valued life.	

Note: Text in bold denotes example exercises (see Appendix E). Page numbers refer to chapters in *Get out of Your Mind and Into Your Life* (Hayes & Smith, 2005).

Stage four developed this work into *defining the self*. These sessions focused on developing and strengthening a sense of self that exists alongside, but independent from, private internal events. Sessions explored the idea that the 'I' that people most often verbalise is only one aspect of the self (i.e., self-as-content). Clinicians explored with the participants two other aspects of the self; a self that is grounded in the present moment (self-as-process) and a self that is constant and stable over time and context (self-as-context; see section 2.2.1). For example, patients were invited to recall events from their past and identify a self that was consistent throughout those experiences, regardless of age or context (self-as-context). Similarly, metaphors such as 'The Chess Board' (Appendix E) were used to convey the idea of a stable and consistent sense of self that may be witness to many battles or storms (a metaphor for periods of emotional distress), but that is not defined by them. Self-as-process exercises, on the other hand, were used to heighten the participants' awareness of an ongoing sense of self that can consistently observe behaviour in the present moment.

The fifth stage of treatment was concerned with helping the participant to identify their core life *values*. These were explored using experiential exercises and group discussions that distinguished core values from tangible goals. These sessions also discussed the willingness to act in valued ways despite the short term discomfort that such actions may bring, grounding willingness for change in the possibility of moving towards a fuller and more meaningful life. For example, a socially anxious patient could explore his willingness to feel highly anxious if doing so had the potential to bring him closer to the value of intimacy. Stage six naturally extended this work into goal setting and committed action. Participants were helped to define behavioural actions (goals) that would bring them closer to core values and group discussions were used to address barriers to change. Group discussions also considered the difference between pliance and meaningful commitment to change and the final session involved public commitment to a valued life direction.

Although the broad structure depicted in Table 6.2 was followed (see also Appendix E), it is important to note that clinicians sought to achieve a balance between following this structure and being flexible to participants' needs as they occurred during group sessions. For example, if participants showed strong attachment to the "control and eliminate agenda" and were not receptive to considering control as the problem, therapists were free to extend this stage so as to address it more comprehensively (and

vice versa). It is also important to note that, throughout the 16 weeks, clinicians continually engaged in a process of re-consenting; that is, they asked for a participant's permission before engaging in any one-to-one piece of work. This was done to avoid coercion that might otherwise have occurred given the vulnerability of the group and the evocative nature of ACT.

6.2.4.4 *Post-Intervention Assessment.* After the treatment phase, all participants completed a second questionnaire pack in their own time. They also revisited the IPTS for a post-intervention interview, which asked about their experiences of the group (these data are not presented here). After the interview, patients received a copy of "Get out of your Mind and into your Life" (Hayes & Smith, 2005) on 6 month loan and a mindfulness meditation CD. Six months after attending the group, participants received a third set of questionnaires and were invited to IPTS for a post-intervention SCID-II. In this second administration of the SCID-II, I asked participants to report PD symptoms in the last 12-months only. After the 6-month follow-up assessment, participants were invited to a 2.5 hour refresher session. Finally, a fourth questionnaire pack was completed 12-months following treatment.

6.2.5 *Analysis Strategy*

After the distribution of all study variables had been considered, the first stage of analysis focused on change in outcome measures across testing periods (i.e., treatment effects). Because current literature advocates the evaluation of group change (e.g., using ANOVA) and individual change (e.g., clinical significance), both strategies were used.

Firstly, to assess group change, a series of within subject, repeated measures ANOVA were computed for each outcome and process measure. These tested for a significant main effect of Time (T1, T2, T3 and T4). Additional post-hoc, paired samples *t*-tests were used to follow up main effects. Because the probability of Type II error is high in small samples, alphas were not adjusted for multiple comparisons. To quantify the magnitude of within group change in a manner comparable with published trials, Cohen's *d* effect sizes (ES) were also calculated.

Secondly, to assess individual change, the clinical significance change criteria proposed by Jacobson and Truax (1991; see section 3.4.2) were used. Reliable change was measured by calculating a reliable change index (RCI). Clinical cut-off was

determined using the least arbitrary of Jacobson & Truax's proposed methods; calculating the value half-way between the mean score of the clinical and non-clinical population (criterion c; also see Thomas & Truax, 2008 and section 3.4.2). Categories of change proposed by Thomas & Truax (2008) and Jacobson et al. (1999) were used. These are as follows: *recovered* (reliable change and crosses cut-off), *improved* (reliable change without crossing the cut-off), *same* (no change), and *deteriorated* (reliable worsening of symptoms). These analyses were computed for GSI and BDI-II scores only. This is because WHOQOL clinical norms were not yet available and, owing to exclusion criteria, no participants scored in the ADS clinical range.

Finally, although the sample size prohibited formal mediation analyses, Spearman's Rho correlations were computed to test for lagged associations between process and outcome measures. Firstly, correlations were obtained to assess the association between T1 to T2 change in process measures (e.g., AAQ) and T3 and T4 outcome measures (e.g., GSI, BDI-II). These correlations thus assessed whether change in process measures during treatment were associated with follow-up outcomes. Based on the recommendations of Steketee & Chambless (1992), change in process measures were transformed into residual gain (RG) scores to adjust for variance dependent on repeat testing[24]. Following this, a second set of correlations was computed to assess the association between process scores at T2 and T3 and outcomes at T3 and T4 (respectively). These correlations were thus used to identify whether any post-treatment processes predicted follow-up outcomes.

6.3 Results

6.3.1 *Preliminary Analysis and Participant Characteristics*

Preliminary analyses indicated that all study variables were normally distributed. Most participants scored in the clinical range for depression and global symptom severity (see Table 6.1 and Table 6.3 for means and standard deviations). Half met formal diagnostic criteria for at least one personality disorder (two of more PDs, N = 3) and most presented with co-occurring mood disorders (i.e., were in the clinical range for five or

[24] This procedure standardises pre- and post-treatment means and subtracts the T1 score, multiplied by the correlation between T1 and T2 scores, from T2 (i.e., $RG = Z_{T2} - (Z_{T1} * r_{T1,T2})$).

more SCL-90 domains, see Table 6.1). Completers attended an average of 14 therapy sessions (range 12-16).

Observing raw baseline data, it was apparent that one participant ('Elaine') presented with baseline scores that were lower than expected given her pre-treatment interview and correspondence with her previous therapist. At interview, Elaine appeared to be experiencing entrenched difficulties. She met lifetime diagnostic criteria for avoidant, depressive and borderline personality disorder and symptomatic criteria for avoidant PD (threshold for depressive and borderline PD). In marked contradiction, however, Elaine's baseline questionnaire data suggested that she was experiencing very low levels of psychiatric distress. In fact, her GSI score was below the mean of the non-clinical population. Similarly, her MCMI-III scores were 'invalid' on the basis of insufficient disclosure[25]. These observations suggested that her baseline data were falsely low. Rather than excluding Elaine's scores, analysis of group change are reported both with and without her data.

6.3.2 *Statistical Significance of Change*

6.3.2.1 *Full Sample Data.* Changes in outcome measures across the four testing periods are depicted in Figure 6.3 (full sample). These graphs suggest that anticipated improvements occurred across all outcome measures. Furthermore, trends suggest that improvements occurred both from T1 to T2 *and* during follow-up periods (i.e., continued gains).

[25] No other participants had "invalid" MCMI-III data or showed marked discrepancies between questionnaire and interview data. Furthermore, Elaine's post-treatment MCMI-III scores were not "invalid", neither were her questionnaire and interview data discordant at any time other than at baseline (see section 6.4.2 for a discussion).

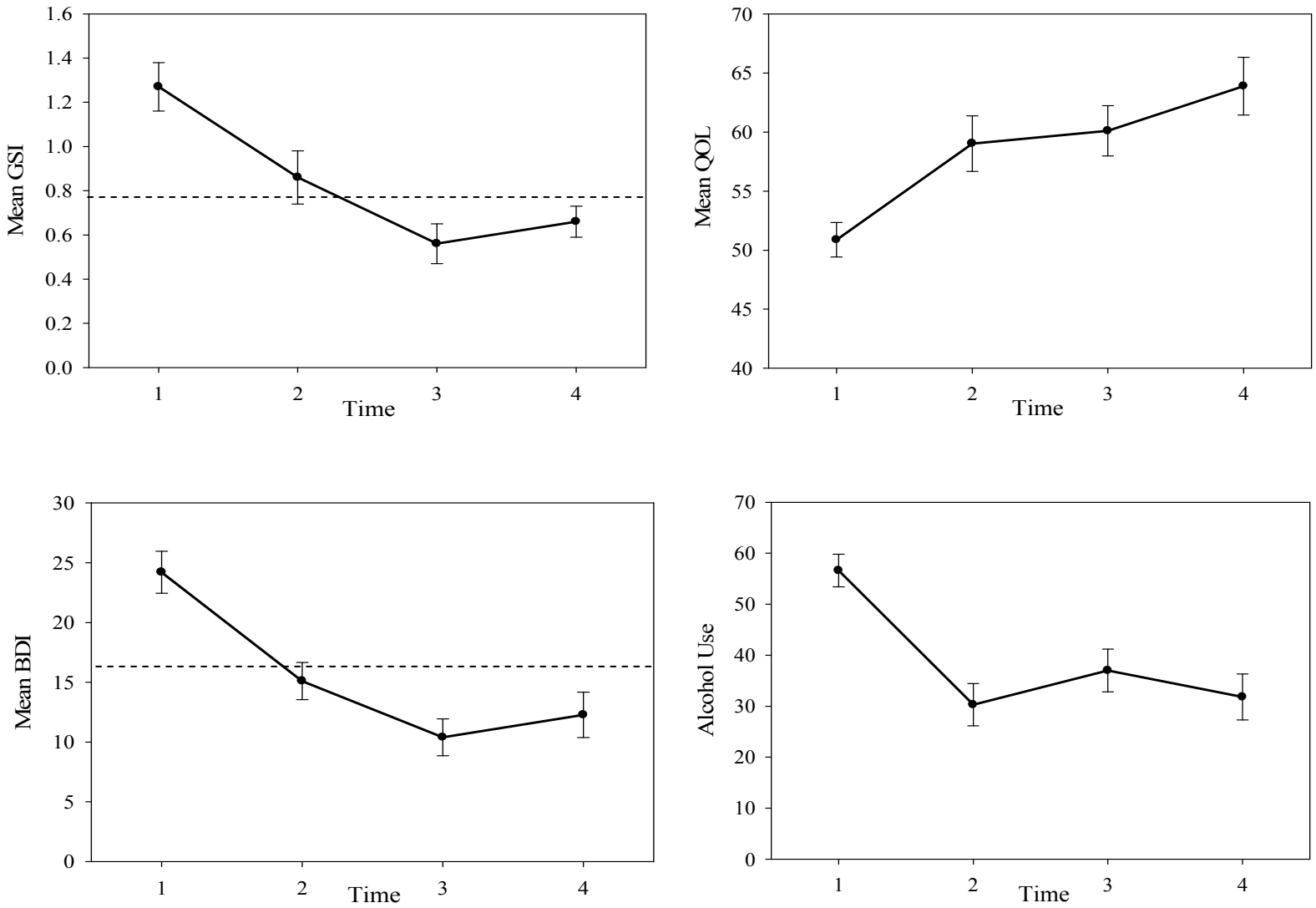


Figure 6.3 Group Mean T1, T2, T3, and T4 Outcome Measures with Standard Error bars (dashed lines denote clinical cut-offs).

Results of the repeated measures ANOVA and post-hoc *t*-tests are reported in Table 6.3. (SCID-II data are presented separately (section 6.3.2) because the SCID-II is a categorical variable). These analyses showed a significant main effect of Time for all outcome measures. Post-hoc *t*-tests comparing T1 and T2 scores showed a significant reduction in BDI-II ($t_{(1, 9)}$ = 2.49, p < .05) and ADS ($t_{(1, 9)}$ =3.03, p < .05), and a marginally significant reduction in GSI ($t_{(1, 9)}$ = 1.91, p =.09). From T1 to T3 significant improvements in GSI ($t_{(1, 9)}$ = 4.05, p < .01), BDI-II ($t_{(1, 9)}$ = 3.14, p = .01), ADS ($t_{(1, 9)}$ = 2.63, p < .05), and WHOQOL ($t_{(1, 9)}$ = 3.10, p < .01) were observed. From T1 to T4, marginally significant change occurred for GSI ($t_{(1, 8)}$ =1.98, p = .08), ADS ($t_{(1, 8)}$ = 2.23, p = .06), and WHOQOL ($t_{(1, 8)}$= 1.81, p = .10) scores. Testing for change in process measures revealed a marginally significant Time effect for AAQ and MAAS scores. Post-hoc *t*-tests comparing T1 and T2 showed a significant reduction in the AAQ ($t_{(1, 9)}$ = 2.63, p < .05), and a marginally significant increase in MAAS ($t_{(1, 9)}$ = 1.94, p = .08). From T1 to T3, change was marginally significant for both (AAQ: $t_{(1, 9)}$ = 1.83, p = .10; MAAS; $t_{(1, 9)}$ = 2.11, p = .06). Neither was significant when comparing T1 and T4, however.

Cohen's *d* ES statistic was computed to quantify the magnitude of within group change ('uncontrolled effect size'; Feske & Chambless, 1995). This was computed by subtracting the group's T2, T3 and T4 mean from the T1 mean, divided by the pooled standard deviation (e.g., $M_{T1} - M_{T2} / (\sqrt{[(\sigma_1^2 + \sigma_2^2) / 2]})$ (Cohen, 1988)). Using Cohen's (1988) guidelines of 'small' (d = 0.2), 'medium' (d = 0.5) and 'large' (d = 0.8), results indicated medium to large effects for all outcomes (Table 6.3).

6.3.2.2 *Data Excluding 'Elaine'.* Table 6.4 reports change in outcome and process measures with Elaine's data excluded. It is clear from these analyses that the effects of ACT were greater when her data were discounted. For example, significant reductions in key outcome measures were found at each time point and all improvements were large in terms of effect size. Furthermore, significant reductions in process measures were also found. These findings suggest that Elaine's data were anomalous in comparison to the other participants' data. However, because the exclusion of her data from subsequent analyses would be pro-hypothesis, it was considered prudent to report the findings with her included and to discuss her case in detail in the discussion (see section 6.4.2).

Table 6.3

Means (SD), Repeated Measures ANOVA and Post-Hoc T-tests for Outcome and Process Measures Comparing T1 to T2, T3 and T4 Testing in the Full Sample.

	Mean (SD)				ANOVA (*F*) / ES (*d*)						
	T1 (N = 10)	T2 (N = 10)	T3 (N = 10)	T4 (N = 9)	Time	T1-T2		T1-T3		T1-T4	
Measures					$F_{(1, 9)}$	$t_{(1, 9)}$	*d*	$t_{(1, 9)}$	*d*	$t_{(1, 8)}$	*d*
Primary											
ADS	56.36 (23.1)	22.00 (22.0)	35.75 (28.8)	31.88 (25.3)	5.25**	3.03*	1.52	2.63*	0.79	2.23†	1.00
GSI	1.27 (0.5)	0.86 (0.7)	0.56 (0.6)	0.58 (0.41)	4.92**	1.91†	0.67	4.05*	1.29	1.98†	1.50
BDI-II	24.23 (11.2)	15.12 (10.6)	10.40 (10.2)	11.00 (12.2)	4.28*	2.49*	0.83	3.14*	1.30	1.71	1.10
WHOQOL	50.68 (9.0)	55.76 (12.5)	65.29 (15.0)	66.15 (11.4)	3.63*	1.68	0.47	3.10*	1.18	1.81†	1.50
Process											
AAQ	4.72 (0.5)	4.00 (1.0)	4.00 (1.3)	4.11 (1.2)	2.60†	2.63*	1.00	1.83†	0.80	1.10	0.72
MAAS	59.55 (16.5)	62.85 (13.5)	66.75 (13.5)	73.50 (13.5)	2.67†	1.94†	0.22	2.11†	0.48	1.16	0.93
TBQ	4.50 (3.5)	2.62 (2.1)	3.25 (2.8)	4.00 (3.6)	0.77	-	-	-	-	-	-
TFQ	4.63 (2.1)	4.12 (1.6)	3.25 (1.8)	3.25 (1.5)	1.26	-	-	-	-	-	-
VLQ	6.33 (4.30)	3.67 (3.30)	3.77 (3.76)	3.88 (5.15)	2.00	-	-	-	-	-	-

† $p < .10$ *$p < .05$. ** $p < .01$. Note: BL = Baseline; M = mean; SD = Standard Deviation; T1 = pre-treatment; T2 = post-treatment; T3 = 6-month follow-up; T4 = 12-month follow-up; GSI = Global Severity Index; QOL = World Health Organisation Quality Of Life; BDI-II = Beck's Depression Inventory II; ADS = Alcohol Dependency Subscale; AAQ = Acceptance and Action Questionnaire; MAAS = Mindfulness and Awareness Scale; TFQ = Thought Frequency Questionnaire; TBQ = Thought Believability Questionnaire. VLQ = Valued Living Questionnaire. *d* = Cohen's *d* Effect Size. Note high VQL scores indicate *un*valued living.

Table 6.4

Means (SD), Repeated Measures ANOVA and Post-Hoc T-tests for Outcome and Process Measures Comparing T1 to T2, T3 and T4 Testing Excluding Elaine's Data.

	Mean (SD)				ANOVA (*F*) / ES (*d*)						
	T1 (N = 9)	T2 (N = 9)	T3 (N = 9)	T4 (N = 8)	Time	T1-T2		T1-T3		T1-T4	
Measures					$F_{(1, 8)}$	$t_{(1, 8)}$	*d*	$t_{(1, 8)}$	*d*	$t_{(1, 7)}$	*d*
Primary											
GSI	1.37 (0.6)	0.87 (0.8)	0.55 (0.6)	0.62 (0.7)	7.26**	2.36*	0.82	5.50**	1.33	2.84*	1.09
BDI-II	25.11 (11.5)	14.55 (11.0)	8.78 (9.9)	10.50 (9.6)	6.47**	2.81*	0.92	4.07**	1.56	2.30*	1.39
WHOQOL	48.98 (7.8)	57.96 (11.1)	67.03 (14.9)	67.10 (16.3)	6.55**	3.51**	0.95	5.00**	1.59	2.78*	1.51
ADS	55.44 (21.2)	30.88 (27.7)	38.33 (28.0)	27.86 (24.4)	4.57*	5.58*	1.00	2.18†	0.69	2.18†	1.21
Process											
AAQ	4.66 (0.5)	3.83 (0.9)	3.80 (1.2)	4.00 (1.1)	3.11*	2.81*	0.84	7.82*	1.00	1.35	0.83
MAAS	3.52 (0.7)	4.13 (1.5)	4.45 (1.0)	4.37 (1.0)	3.97*	2.25†	0.54	2.59*	1.08	1.61	0.97
TBQ	5.78 (3.2)	3.33 (1.2)	3.22 (2.5)	3.28 (3.2)	2.34†	2.81*	1.12	2.45*	0.90	1.18	0.79
TFQ	5.33 (1.5)	4.11 (1.5)	3.22 (1.5)	2.86 (1.1)	5.01*	4.40*	0.80	3.22*	1.42	3.20*	1.93
VLQ	6.37 (4.6)	3.50 (3.5)	3.25 (3.7)	3.13 (4.9)	3.63†	3.65*	0.70	2.73*	.76	2.01†	0.68

† $p < .10$ *$p < .05$. ** $p < .01$. Note: BL = Baseline; M = mean; SD = Standard Deviation; T1 = pre-treatment; T2 = post-treatment; T3 = 6-month follow-up; T4 = 12-month follow-up; GSI = Global Severity Index; QOL = World Health Organisation Quality Of Life; BDI-II = Beck's Depression Inventory II; ADS = Alcohol Dependency Subscale; AAQ = Acceptance and Action Questionnaire; MAAS = Mindfulness and Awareness Scale; TFQ = Thought Frequency Questionnaire; TBQ = Thought Believability Questionnaire. VLQ = Valued Living Questionnaire. *d* = Cohen's *d* Effect Size. Note high VQL scores indicate *un*valued living.

6.3.3 *Change in Personality Disorder Symptomatology*

At baseline assessment, half of the group (N = 5) met formal SCID-II diagnostic criteria for at least one PD. This was rated (a) over the participant's lifetime (diagnostic criterion) *and* (b) in the immediately preceding 12-months (symptomatic criterion). At T3 (i.e., one year later), only two of the five participants continued to meet the symptomatic criterion.

6.3.4 *Clinical Significance of Change*

The clinical significance of change was computed for GSI and BDI-II scores only (see section 6.2.5). The values used for change calculations were as follows: GSI non-clinical Mean = 0.31 (SD = 0.31), clinical Mean = 1.26 (SD = 0.68), test retest reliability r = .91 (Derogatis 1993); BDI-II non-clinical Mean = 8.9 (SD = 12.36; Dozois et al., 1998), clinical Mean = 22.45 (SD = 12.75; Beck et al., 1996), and test retest reliability r = .93 (Beck et al., 1996). Results (Table 6.5) showed that at T2, 50% of participants had either improved or recovered from depression, which rose to 70% at T3, and fell back to 50% at T4. For GSI scores, 60% of participants could be classified as improved or recovered at T2, 60% as recovered at T3, and 50% as recovered or improved at T4.

6.3.5 *Re-referral*

At 12 month follow-up, none of the participants were awaiting, or receiving, treatment for a psychological problem from the psychological services.

Table 6.5

Clinical Significance of Change from T1 to T2, T3, and T4 Testing

ID	GSI			BDI-II		
	T2	T3	T4	T2	T3	T4
1	R	R	R	R	R	R
2	R	R	R	R	R	R
3	R	R	R	R	R	R
4	R	R	I	I	R	I
5	I	R	S	R	R	S
6	S	I	I	I	R	R
7	S	S	S	S	S	S
8	S	I	-	S	S	-
9	S	S	S	S	S	S
10*	S	S	D	S	S	D
Totals:						
Recovered	4	5	3	4	6	4
Improved	1	2	2	2	0	1
Same	5	3	3	4	4	3
Deteriorated	0	0	1	0	0	1

Note: 'ID' = participant identification number (this corresponds with information on each participant reported in Appendix B. 'D' = deteriorated; S = 'same'; I = 'improved'; R = 'recovered'; GSI = Global Severity Index; BDI-II = Beck's Depression inventory. * = Elaine's data.

6.3.6 *Exploratory Mechanisms of Change*

Although change in process measures did not reach significance (see Table 6.3), exploratory analysis were used to examine the relationship between the observed changes and outcome measures. These analyses began by assessing whether changes in process measures from T1 to T2 (i.e., change scores) were associated with outcomes at T3 and T4. These analyses were conducted using Spearman Rho correlations (owing to the small sample) with residual gain (RG) scores indexing T1 to T2 change in process measures. Results are reported in Table 6.6.

Table 6.6

Correlations (Spearman Rho) Between T1-T2 Change in Process Measures and T3, T4 Outcome Measures

Process (T1-T2 change)	Outcomes							
	GSI		BDI-II		WHOQOL		ADS	
	T3	T4	T3	T4	T3	T4	T3	T4
RG AAQ	.74*	.60*	.74*	.58	.66*	.48	.19	.49
RG MAAS	.63*	.70*	.43	.59	.43	.68*	.09	.66*
RG VLQ	.16	.71*	.33	.67*	.06	.65*	.32	.50
RG TBQ	.50	.47	.62*	.51	.46	.50	.44	.61
RG TFQ	.15	.10	.39	.22	.21	.10	.51	.22

*$p < .01$. Note: RG = Residual Gain; AAQ = Acceptance and Action Questionnaire (AAQ); MAAS = Mindfulness Attention and Awareness Scale; VLQ = Valued living Questionnaire. TBQ = Thought Believability Questionnaire; TFQ = Thought Frequency Questionnaire.

Results from this analysis showed that T1 to T2 changes in the AAQ were significantly associated with T3 GSI, BDI-II, and WHOQOL and T4 GSI and WHOQOL scores. Change in MAAS was significantly associated with T3 and T4 GSI and T4 WHOQOL and ADS scores. Change in the VLQ was significantly associated with T4 GSI, BDI-II, and WHOQOL scores. Change in TBQ was significantly associated with T3 BDI-II scores and change in TFQ was not significantly related to any outcomes.

To assess whether post-treatment process scores (rather than change) were associated with future outcomes, a second series of correlations were run. These showed that T2 AAQ scores were significantly associated with T3 and T4 GSI ($r = .61$, $p < .10$; $r = .81$, $p < .01$ respectively), BDI-II ($r = .83$, $p < .01$; $r = .87$, $p < .01$, respectively), and WHOQOL ($r = .71$, $p < .05$; $r = .82$, $p < .05$ respectively). T3 MAAS scores were significantly associated with T4 measures of GSI ($r = .70$, $p < .01$) and BDI-II ($r = .71$, $p < .01$). T3 TBQ scores were significantly associated with T4 BDI-II ($r = .73$, $p < .01$) and WHOQOL ($r = .78$, $p < .01$). Again, consistent with expectation, TFQ was not related to any outcomes.

6.4. Discussion

6.4.1 *Study Findings*

Previous research has shown that ACT can obtain meaningful clinical effects when delivered to patients experiencing acute distress, but little was known about its applicability to treatment resistant patients. To develop an application to patients meeting this criterion, the present study was designed to ascertain whether ACT had possible clinical benefits when delivered to them in a 16-week, group-based format. Because this application was novel, in keeping with previous research on similarly vulnerable groups (e.g., Telch et al., 2000), a pre-post, uncontrolled pilot trial was used.

Overall, results were supportive of the utility of ACT for this group. This occurred despite a number of factors that might have mitigated against it. For example, the sample size was small and the variability of symptoms was high. Both these factors reduce the power of detecting significant effects. The recruitment of participants whose symptoms have been resistant to, or relapsed following, previous psychological treatment further challenged the probability of obtaining clinically meaningful change. Moreover, the group-base mode of delivery meant that none of the participants received one-to-one care. Furthermore, the intervention did not explicitly aim to reduce symptoms; rather, it dealt more specifically with increasing acceptance of them and creating a new repertoire of value consistent action.

Despite these many factors, clinically meaningful effects were found. Group analysis showed significantly fewer self-reported psychiatric symptoms (GSI, BDI-II), a significant reduction in self-reported alcohol use, and significant improvements in quality of life 6-months following the ACT group as compared to at baseline. Moreover, judged against well respected criteria (Cohen 1988), the magnitude of these within group effects were found to be medium to large (compared to baseline). Additionally, on the individual level, clinically significant and reliable improvements were found in 50% - 70% of the participants (depending on the measure and time of testing). Furthermore, only two of five participants continued to meet criteria for PD symptoms at 6-months and 0% of the participants had received or were awaiting further psychological treatment at 12-months.

The fact that effects were most noticeable at 6-month follow-up is interesting and replicates a trend that is often reported in ACT outcome trials; that is, *continued gains*

following treatment termination (e.g., Hayes, Wilson, et al., 2004, see section 2.2.2). This pattern may reflect the fact that ACT does not focus on symptom removal per se; rather, it aims to help patients construct a new repertoire for behaviour that, when successful, allows increasing access to positive reinforcement. Furthermore, this behaviour should be guided by personal values and should not be contingent on the reinforcement of the therapist. These factors are likely to support long term and durable changes. Indeed, in keeping with these speculations, preliminary evidence suggested that valued living was related to long term outcomes.

Using criteria defined by Hollon, Stewart, and Strunk (2006), the continued gains that have been found may suggest that ACT does not simply have palliative effects ("suppress the expression of the disorder so long as they are applied" Hollon et al., p. 287); but rather, for some patients, it may have the capacity to produce enduring effects ("effects that reverse processes that would otherwise lead to the continuation of the disorder"). In support of this proposition, theory-consistent changes in process measures were found to be associated with follow-up outcomes. Similarly, post-treatment scores on the AAQ, MAAS, and TBQ were significantly associated with outcomes at 6-month and 12-months following treatment. Although these trends were based on correlational analyses of a small sample, they nevertheless are consistent with the ACT-based prediction that changing the way a person interacts with unwanted private events precedes and facilitates mental health improvements. Findings were not, however, in keeping with the CBT-based prediction that changes in the frequency of certain cognitions affect a change in symptoms. Although changes in thought frequency occurred, these changes were unrelated to outcome. This tentative finding suggests future work could focus on comparing the effects of these different processes on treatment outcomes. This could be more thoroughly explored by, for example, comparing the use or ACT versus CBT for this group.

6.4.2 *Elaine's Response to Treatment.* Despite the promising findings reported, Elaine's data were anomalous. As previously discussed, she provided low but invalid baseline data. Her MCMI-III scores were "invalid" on the basis of insufficient disclosure. Moreover, there was poor concordance between her baseline questionnaire and interview data. This was not observed in any other participants, nor was it observed in relation to Elaine at any other testing period. Thus, although Elaine showed a reliable

deterioration of symptoms across testing periods, she remarked at interview "from my point of view nothing seems to have changed ... I am at the point that I was at before ACT... this is my life, this is the way it is, and I am resigned to it".

At best, therefore, ACT appeared to have had no effect on Elaine's psychological well being. One factor that may have been responsible for the lack of change was the recurrence of episodes of dissociation during treatment. Dissociation occurred at least once during most sessions and was usually precipitated by exchanges relating to issues of interpersonal intimacy. This occurred despite the fact that she reported that her desire for intimacy was a key reason for attending the group. The clinicians observed that dissociation rendered her psychologically absent from much of the treatment. Indeed, Elaine remarked that after attending ACT she had begun to read the self-help book and found that most of its content was unfamiliar to her: "a lot of it ... I can't remember what we did in the sessions ... a lot of it was like completely new ... so I don't think I was ... I found it very hard to get involved in sessions". Other factors that could have contributed to change resistance included other well developed and deeply engrained patterns of avoidance (i.e., in addition to dissociation) and strong attachment to the self-as-content. For example, she remarked that the thought of abandoning avoidance strategies was "too scary" to contemplate ("I thought oh my God I can't do this") and showed strong attachment to self-conceptualisations ("I get the feeling of being unsafe when people get the idea of who I am or what I am ... of not being good enough"). Although it is particularly difficult to speculate about the effect of treatment on Elaine's well-being, a possible interpretation of the improved concordance between questionnaire and interview data following treatment, and her valid MCMI-III scores, is that ACT increased her ability or willingness to report honestly on her distress.

6.4.3 *Methodological Limitations*

Although, overall, the present findings have been promising, this study was designed as a pilot trial. Because of this, the outcomes are tentative and require replication. For example, in the design deployed, the lack of a control group means that the effect of non-specific factors (e.g., therapeutic alliance and expectancy bias) is unknown. It is most likely that these factors account for some of the effects observed, but there are several reasons to suspect that these factors were not the main agents of change. For

example, by definition, this group had already had several exposures to similar non-treatment specific variables (i.e., therapist contact) to which their symptoms had been resistant or subsequently relapsed following. Furthermore, the absence of these non-therapeutic factors at post-test makes it unlikely that they would account for the enduring changes that were observed for some participants. Also, theory consistent associations between experiential avoidance, thought believability, and mindfulness support the interpretation that treatment-specific variables played some role in treatment outcomes.

The second main limitation of this study is its reliance on self-report data. Obvious problems with this include demand bias, self-report accuracy and increasing familiarity with the measures. Attempts to minimise these effects were made. Participants completed questionnaires in their own time at home and used ID numbers as identifiers rather than names. Additionally, a third party liaised with them regarding the completion and return of their data. Furthermore, carry over effects should have been minimised by the long delay between testing periods. The third main limitation concerns the use of a small sample from which the most at-risk of patients were excluded. This makes it difficult to establish whether the effects seen will generalise to other clinical groups. Nevertheless, the use of broad inclusion criteria is a strength in terms of external validity (see chapter 3).

6.4.4 *Implications*

The results from this trial tentatively suggest that ACT can be delivered to a group of patients who failed to benefit from, or have relapsed following, previous psychological treatment. ACT was associated with meaningful effects for many of the participants and, overall, their gains were maintained at follow-up. Moreover, the current findings, albeit tentative, were in keeping with the prediction that ACT-derived processes were related to outcomes. The main implication of this study, therefore, is that ACT may prove to be a useful intervention for this patient group. Given the limitations that have been discussed, however, no firm conclusions about cause-and-effect, or generalisability, can be made. Furthermore, although most patients had already received cognitive-behavioural treatments in the past, it cannot be inferred that a CBT-based intervention would not have obtained equally beneficial outcomes. In keeping with

Ost's (2008) suggestion, future research should extend this work, ideally exploring the effects of ACT relative to an active comparison group such as a CBT-based approach. To this end, study 4 used a more scientifically rigorous method to compare the effects of ACT and CBT-based treatment as usual (CBT-TAU) for treatment resistant patients.

CHAPTER VII

Study 4. A Pilot Randomised Control Trial of Acceptance and Commitment Therapy (ACT) versus Cognitive Behaviour Therapy-Based Treatment as Usual (CBT-TAU) for a Heterogeneous Group of Treatment Resistant Patients

7.1 Introduction

Study 3 tentatively supported the use of ACT for treatment resistant patients. As predicted, significant improvements in psychological functioning, quality of life, and alcohol use were found and these tended to be sustained at 6- and 12-month follow-up. Furthermore, preliminary data on mechanisms of change appeared to be consistent with the ACT model; changes in experiential avoidance, thought believability, mindfulness, and valued living were associated with outcome, but changes in thought frequency were not. Although these findings are promising they are nonetheless preliminary. Because study 3 was an uncontrolled trial, one of its main limitations was the lack of a control group. For example, although many of the patients had previously received CBT, it is impossible to say with confidence that ACT was more efficacious than this currently available alternative. The present study aimed to address this issue more directly, using a randomised control trial (RCT) to compare the effect of ACT relative to Cognitive Behaviour Therapy-based treatment as usual (CBT-TAU) for treatment resistant patients. Mechanisms of change were also explored.

7.1.1 *ACT versus CBT for Treatment Resistant Patients*

The main distinction between ACT and CBT is their reliance on different processes of change. ACT's primary focus is on altering a patient's relationship with his/her private experiences, whereas CBT's is on altering the form and/or frequency of those experiences (see sections 1.2 and 2.2). Few trials have directly compared these treatments. Of those that have, data tentatively suggest that ACT is at least as effective as CBT (e.g., Forman et al., 2007; Lappalainen et al., 2007; see section 2.2.3). Both treatments have also obtained some empirical evidence to suggest that they can achieve effects in theory-consistent ways. Nevertheless, perhaps because the CBT literature is more extensive than ACT's, most CBT trials have reported mediation-based analyses that are inconsistent with predictions of the cognitive model (see section 1.2.3). Some of this work further

suggests that CBT could achieve treatment effects by indirectly cultivating mindful awareness of symptoms (e.g., Forman et al., 2007; Teasdale et al., 2002).

No study to date has compared ACT and CBT for treatment resistant patients. For this group, it is reasonable to suspect that different outcomes may occur. This is because, as discussed in chapter 1, the effectiveness of cognitive interventions appears to be more limited when delivered to patients' with chronic, co-occurring and/or PD symptoms (see section 1.2.4). Conversely, some research tentatively suggests that ACT and ACT-like techniques may hold promise with this type of patient (study 3; Dimidjian et al., 2006; Gratz & Gunderson, 2006; Hayes, Wilson, et al., 2004). Based on ACT theorising, there are at least two possible reasons why ACT may hold more promise than CBT for treatment resistant patients.

Firstly, different outcomes can be predicted based on the different approaches of the two treatments. ACT is designed to alter higher order generic processes that do not differ across diagnostic categories (e.g., experiential avoidance). As such, ACT can be more easily administered to a heterogeneous group of patients. Although CBT also targets underlying processes, such as overgeneralisations and catastophising, this approach arguably gives greater credence to processes regarded as specific to particular diagnostic groups. That is to say, CBT has relatively fixed methods for modifying schemas that are specific to, for example, depression or anxiety. This has at times rendered CBT difficult to deliver to dual diagnosis patients (e.g., see Conrad & Stewart, 2005).

Secondly, ACT theorising proposes that symptoms of psychological distress arise from associative networks that cannot be unlearned (section 2.1). By virtue of the persistence and pervasiveness of entrenched disorders, it is reasonable to suspect that these networks are especially elaborated in treatment resistant patients. They may, therefore, be especially resistant to techniques designed to dismantle or overlay them (e.g., cognitive restructuring). Indeed, working with BPD patients, Linehan (1993) observed that direct attempts to change thoughts did not work well with this chronic patient group, often resulting in early drop-out and change resistance. Linehan has subsequently presented good evidence to suggest that an acceptance-based and principle-driven approach (i.e., Dialectical Behaviour Therapy) can be effective in treating the entrenched and co-morbid symptoms of this group (e.g., Linehan et al., 2006). These findings are in keeping with the successful use of acceptance-based

treatments for other entrenched disorders (e.g., Dimidjian et al., 2006; Kenny & Williams, 2008). Ironically, some CBT research is not inconsistent with ACT's theoretical predictions, suggesting that changing the occurrence of thoughts is not actually an active treatment process, but rather that CBT obtains effects through indirectly cultivating a mindful perspective to unchanging thoughts (Teasdale et al., 2002). If this is indeed the case, ACT could be more effective than CBT because its techniques are specifically designed to use this change mechanism.

7.1.2 *Methodological Considerations*

The most scientifically rigorous method of comparing ACT and CBT would be to conduct a powered RCT. As discussed in chapter 3, however, powered RCTs are seldom conducted during the pilot phase of investigating a new treatment. Instead, pilot RCTs typically precede powered trials, providing vital information that can be used to justify and inform them (Campbell et al., 2001, see section 3.3.2). The present trial thus used a pilot RCT. Many design features of this RCT concurred with Ost's (2008) recent recommendations for improving the internal validity of ACT outcome research. Other aspects, however, were specifically designed to maximise external validity.

The first way in which the present trial was in keeping with Ost's (2008) recommendations was by comparing ACT to an ecologically valid, well matched, active comparison group: CBT-TAU. Several procedures were implemented to maximise internal validity. For example, in the patient information sheets (see Appendix F), the order of information regarding the two treatments was randomised so as to prevent implicit bias. Furthermore, both groups received treatment of equal duration, therapists had comparable levels of experience delivering the given intervention (i.e., ACT or CBT-TAU), and all therapists were offered fortnightly supervision. Moreover, to control for non-specific effects of mindfulness training in the ACT group (i.e., relaxation, breathing skills), the CBT-TAU group began with a 10 minute deep breathing exercise. Finally, the possible confounds of expectancy for change and treatment credibility were monitored during the treatment phase.

Because of the concerns regarding the effectiveness of CBT for this group (see section 1.2.4), this active comparison was ethically defended on the following grounds: (a) patients were offered treatment imminently (in 1-3 months) as opposed to in 8-12

months (waiting list duration); (b) CBT, either group-based or individual, was the most probable treatment after the 8-12 month wait; (c) both CBT and ACT were longer in duration than standard care (in DHFT, CBT is a 12-week course); and (d) the group-based delivery, which has therapeutic effects in and of itself, was novel for some participants. Moreover, the most at-risk of the potential patient group were again excluded from the trial because of its exploratory nature.

The second way in which this trial concurred with Ost's recommendations was by employing an independent researcher to use an unobjectionable randomisation procedure, and to conceal the details of this procedure from individuals involved in the study (see section 7.2.3). Thirdly, also in keeping with Ost's recommendations, well-recognised and validated diagnostic, outcome, and process measures were deployed. This study continued to use the outcome measures piloted in study 3, but refined some of the process measures. Specifically, a well-validated CBT-derived measure was used to index the frequency and believability of automatic negative thoughts, and a newly validated questionnaire was used to measure valued living (see section 7.2.5). Finally, also consistent with Ost's suggestions, data were analysed using three analytic procedures: analysis by treatment administered, intention to treat analyses, and clinical significance of change.

Most of the procedures that have been described were designed to maximise internal validity. As discussed in chapter 3, however, external validity is an equally important goal of outcome research. To maximise external validity, therefore, some aspects of this trial were designed to approximate treatment delivery in a real-world clinical setting. Firstly, samples were heterogeneous and the exclusion criteria were unrestrictive. Indeed, patients recruited to this trial would be those typically excluded from most RCTs because of the threat they pose to internal validity (Westen et al., 2001). Secondly, interventions were not fully manualised. Both followed a broad protocol that guided the content of weekly or bi-weekly sessions, but that was flexible, self-correcting and reactive to the participants' specific in-session needs. Related to this, therapists delivering both treatments were permitted to see participants on an individual basis if deemed essential for ethical reasons. Thirdly, because it was not feasible to obtain 'blind' assessors, allocation concealment was used to ensure non-biased assessment prior to randomisation (see Schulz, 2000). Although these design aspects compromised

internal validity to some extent, they also reflected some important aspects of real life treatment settings.

In addition to issues of internal and external validity, the rate of participant recruitment was a concern for the present study. Although study 3 recruited a sample of patients with symptoms characteristic of the treatment resistant population (as indicated by the literature), this larger trial required a more rapid rate of recruitment. The trial was thus publicised to all The Chines and IPTS clinicians and referrals were taken from a larger participant pool than study 3 (section 7.4.2.1). This included patients who had no details listed on the PEAK database, but for whom it could be reliably established that they had received previous therapy from other NHS regions or privately.

7.1.3 *Synopsis of the Present Study*

The aim of this study was to pilot test the relative effects of ACT versus CBT-TAU for treatment resistant patients and to conduct a preliminary investigation into mechanisms of change. Based on previous literature, it was predicted that both interventions would obtain effects in the short term (i.e., neither would be inert), but that the degree and durability of change would be superior in the ACT group. Thus, between-group differences were expected to be most pronounced at follow-up. Exploratory analyses of mechanisms of change were also conducted. Based on the ACT model (see section 2.2), it was predicted that ACT would improve psychological functioning by reducing experiential avoidance and cognitive fusion, and by increasing mindfulness and valued living. Conversely, based on the CBT model (see section 1.2), it was predicted that CBT would achieve change through reducing the occurrence of automatic negative thoughts. Predictions regarding mindfulness were less clear for CBT. Although the traditional CBT model does not consider mindfulness to be critical for change, some research suggests that it may play an important role (e.g., Teasdale et al., 2002; Forman et al., 2007). The current study explored these possibilities.

7.2 Methodology

7.2.1 *Design*

This study used a RCT. The independent variable was treatment type: 16 weeks group-based ACT versus 16 weeks group-based CBT-TAU. The dependent variables (outcome measures) included a global measure of symptom severity, quality of life, PD symptoms, depression and the tendency to engage in MBs. Process variables included a measure of experiential avoidance, thought frequency and believability, valued living and mindfulness. Outcome and process variables were measures at baseline (T1), post-treatment (T2), and 6-month follow-up (T3) and repeated measures, between-group comparisons were made. The design was thus a 2 x 3 ANOVA in which the between subjects factor was group and the within subjects factor was time.

7.2.2 *Participants*

After obtaining LREC approval, 40 participants were recruited to the trial. The inclusion/exclusion criteria were the same as those described in study 3 (see section 6.2.2). The routes to recruitment were (a) GP referrals to The Chines, (b) waiting list for psychology at the IPTS or The Chines, (c) clinician referrals following an initial assessment at the IPTS or The Chines, or (d) clinician referrals following a course of therapy during which the patient's symptoms failed to remit. Participants were identified as potentially eligible if the PEAK database or the patient's file and/or previous therapist indicated that they met the inclusion criteria. Figure 7.1 shows the flow of participants through recruitment and testing stages of the trial. Sixty three patients were identified as meeting the inclusion criteria, of which 42 opted in (see section 7.2.6). Two patients met the exclusion criteria, resulting in 20 participants per condition. Table 7.1 shows baseline characteristics of the whole sample, split by group.

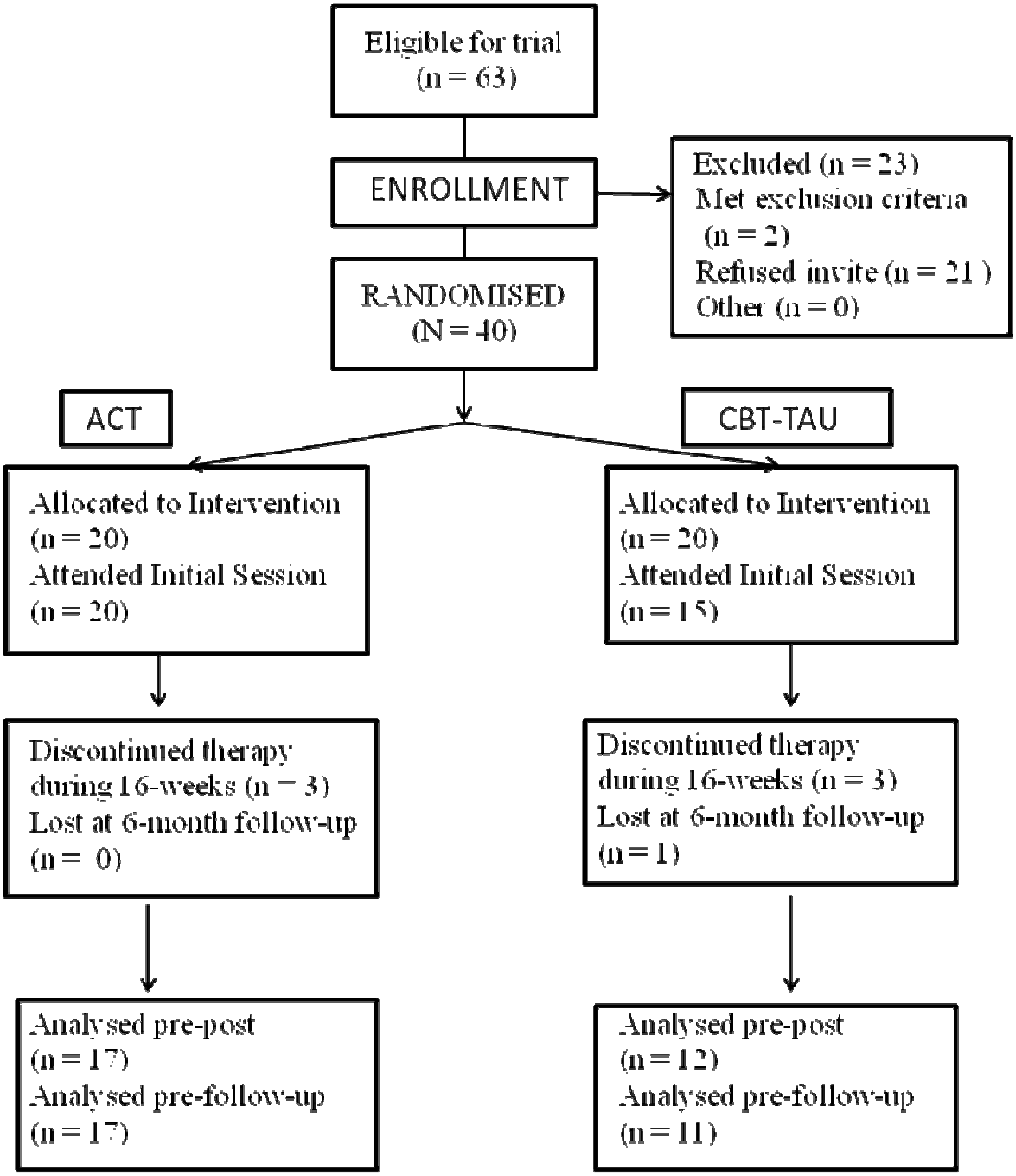


Figure 7.1 Flow Chart of Patient Recruitment to the Trial.

Table 7.1

Demographic and Baseline Statistics for Full Sample Split by Group

Demographics/Baseline Symptoms	ACT (N = 20)	CBT-TAU (N = 20)
Age (years)	44.50	44.35
Gender (% female)	65%	55%
Currently employed/ in education* (%)	45%	50%
Medication (% yes)	85%	80%
Previous no. therapeutic episodes (Mean)	2.75	2.25
Previous no. sessions	38.70	34.40
Median no. months since last therapy (range) **	12 (1-54)	24 (1-75)
Clinical range for depression (%)	95%	85%
Clinical range for GSI (%)	75%	75%
Clinical range for 1 SCL-90 domain	15%	5%
Clinical range in up to five SCL-90 domains	20%	25%
Clinical range in six or more SCL-90 domains	55%	55%
PD Lifetime Criteria (%)	40%	40%

*At start of ACT/CBT-TAU groups. **Calculated from end of last treatment to start of ACT/CBT-TAU.

7.2.3 *Randomisation Procedures*

Randomisation was organised by a third party who had no investment in the study. Details of this procedure were concealed from individuals involved in the study until *after* all participants had been randomised. To ensure random allocation, the individual used an on-line random number generator to block[26] randomise the 40 spaces on the trial (www.random.org). This randomisation was done in blocks of two and four. Firstly, a virtual dice roll was used to randomise the order of blocks with an odd number indicating a block of two and an even number indicating a block of four. Secondly, the blocks were numbered (block 1, 2, 3 etc) and half of the cases in each block were allocated to either ACT or CBT-TAU. Thirdly, to randomise the order of the cases

[26] Block randomisation ensures that the number of participants allocated to each intervention is closely balanced at any one time.

within the blocks, each case was allocated a randomly generated 2 digit number. The block number was applied as a prefix to the 2 digit number and the cases were ordered from smallest to largest. This randomised cases within each block but maintained block order. Fourthly, "CBT" or "ACT" was printed onto pieces of paper, which were folded and sealed (with staples) in a non-transparent envelope. The front of each envelope was marked with a number denoting the order in which envelopes should be opened. After a participant had consented to the trial and completed baseline assessment (see section 7.2.6) the next envelope in number sequence was opened. There was no specific order in which participants consented to the trial.

7.2.4 *Therapists*

ACT groups were run by two ACT-trained Consultant Clinical Psychologists ('therapist one' and 'therapist two'[27]) and CBT groups were run by a Consultant Clinical Psychologist ('therapist four') and a Consultant Counsellor ('therapist five'). Therapist one worked at the IPTS and the remaining therapists worked in primary care. Although both sets of clinicians had comparable experience delivering the given intervention, therapist one had greater experience in treating entrenched disorders than the remaining three. Each intervention was supervised by a clinical psychologist who was specialised in its delivery. ACT groups were supervised by Dr. Kelly Wilson and CBT by a lead CBT practitioner in DHFT (Ms. Debbie Lee). Supervision was available on a fortnightly basis during the treatment phase of the trial.

7.2.5 Materials

7.2.5.1 *Primary Outcome Measures.* Consistent with study 3, primary outcome measures included Global Severity Index (GSI; Derogatis, 1993), the World Health Organisation Quality of Life Scale (WHOQOL; Skevington et al., 2004), Beck's Depression Inventory II (BDI-II; Beck et al., 1996), and the Structured Clinical Interview for DSM-III Axis II Disorders (SCID-II; First et al., 1996). Rather than using the Millon Alcohol Dependency subscale, however, the MBQ (see study 1) was available for use in this trial.

[27] These names correspond with study 3.

7.2.5.2 *Process Measures*. Also consistent with study 3, the AAQ9 (Hayes, Stroshal, et al., 2004) and the MAAS (Brown & Ryan, 2003) were administered. Another two process measures were also used.

The *Automatic Thoughts Questionnaire* (*ATQ;* Hollon & Kendall, 1980). This is a CBT-based process measure which assesses the frequency (ATQ-TF) of 30 depressogenic, self-referent thoughts (e.g., "I'm a loser") using a scale ranging from (1) "not at all frequent" to (5) "all the time". Participants were also asked to rate how believable these automatic thoughts were when they occurred, using the scale of (1) "not at all believable" to (5) "very believable". Consistent with previous research (Zettle & Hayes, 1989), this was used to index cognitive fusion (ATQ-TB). The ATQ has evidenced good psychometric properties (e.g., $\alpha = .96$ and split-half reliability = .97; Kendall & Hollon, 1980).

The *Revised Valued Living Questionnaire* (*VLQ-R;* Wilson, Sandoz, Kitchens, & Roberts, *in press*). This instrument was used to measure (a) how important 10 pre-defined valued domains (family, marriage, parenting, friends, work, educational training, recreation, spirituality, citizenship, and physical well-being) were to the participant and (b) how consistent, in the past week, their behaviour has been with each valued domain. Items were measured using a scale ranging from 1 ("not at all important/consistent") to 10 ("extremely important/consistent"). VLQ-R scores are computed by calculating the discrepancy between values and valued living (i.e. b – a). Authors reported adequate internal consistency ($\alpha = .77$) and test retest reliability over a two week delay ($r = .75$).

7.2.5.3 *Credibility, Expectancy and Therapeutic Alliance.* To measure the potential confounds of intervention credibility, expectancy for change and therapeutic alliance, an additional two questionnaires were used.

The *Credibility/Expectancy Questionnaire* (*CEQ*; Devilly & Borkovec, 2000). This is a 6-item measure assessing how credible an intervention appears to a participant (e.g., "At this point, how logical does this therapy seem to you?") and the participants expectancy for change (e.g., "How much do you feel that this therapy will help you reduce distress in your daily life?). Items were z-scored and summed for an overall credibility/expectancy score. This measure has shown acceptable internal consistency ($\alpha = .85$, Devilly & Borkovec).

The *Helping Alliance Questionnaire-II Patient Form* (*HAQ-II;* Luborsky et al., 1996). This 19-item instrument measures the strength of the therapist-patient relationship by asking participants to indicate their agreement with 19 statements (e.g., "I feel I can depend on the therapist"). Items are rated using a scale that ranges from (1) strongly disagree to (6) strongly agree. Authors reported that the scale showed good internal consistency (α = .79).

7.2.6 *Procedure*

7.2.6.1 *Recruitment.* A time line of the study is depicted in Figure 7.2. Potentially eligible participants were identified using the routes described in section 7.2.2. These patients received an information pack by post (see Appendix F), which introduced them to the study and described its aims, objectives, and the methods involved. This pack also explained why they were being contacted, reviewed the potential costs and benefits of participation, explained that the final selection for the trial would be based on inclusion/exclusion criteria, and invited them to the initial assessment. Participants opted in to the assessment by contacting the IPTS. Before attending the assessment, patients were required to complete a questionnaire pack which they received by post. At the assessment, I reviewed the patient's suitability to the trial with regard to the inclusion/exclusion criteria. Those unsuitable for the trial (N = 2)[28] were informed that they would remain on the waiting list without their original position having been compromised[29]. Eligible patients were asked to complete a study consent form, to provide a verbal account of previous treatments, and to complete the SCID-II interview. Randomisation occurred after participants had left the clinic (see section 7.2.2) and they were informed of group allocation via post. Similarly, GPs were informed of the patients' involvement in the trial up to 2 weeks before the trial began (see Appendix F).

[28] One had attempted suicide within the last 6-months and the other had a history of Substance Dependency and DSH in the last 6-months

[29] The date that the patient joined the waiting list remained the same regardless of the invitation to participate in the trial.

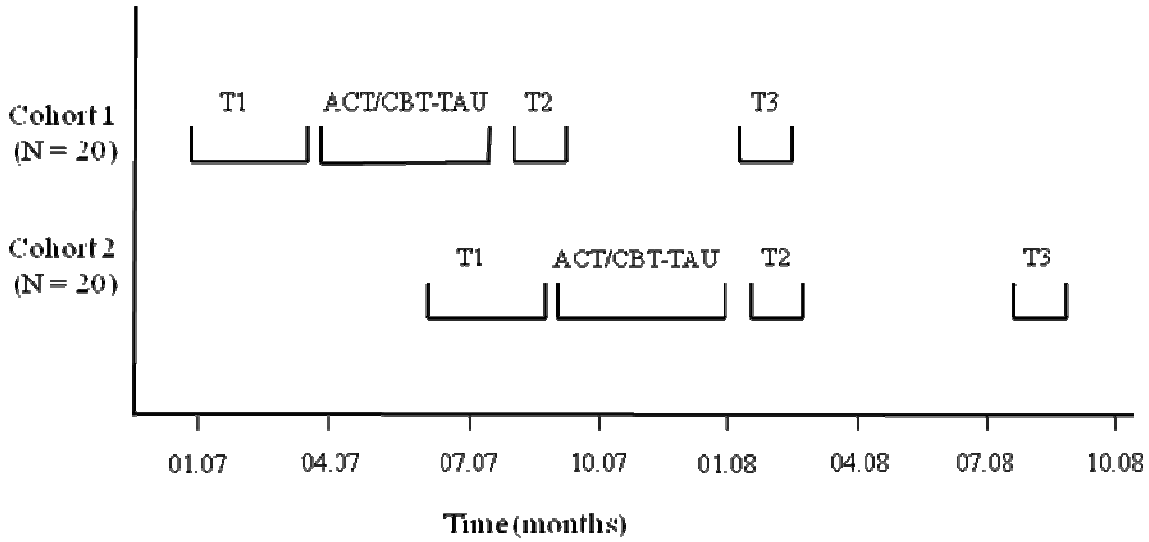


Figure 7.2 Study Timeline

7.2.6.2 *Interventions.* Interventions were held weekly and lasted 2 hours with a 10 minute break. Two groups were run per condition with a maximum of 10 participants invited to each group. ACT and CBT-TAU groups ran concurrently, but on different days of the week (ACT – Monday morning, CBT-TAU Tuesday afternoon). Cohort one treatments (Figure 7.2) were held at different venues (CBT-TAU at a GP surgery; ACT at the IPTS), but cohort two treatments were both held at IPTS. Treatment content was informed by published books on the respective interventions but not fully manualised (see below). Before describing each intervention, their shared and unique aspects are reviewed.

Non-specific Treatment Elements. Common, non-specific elements of both ACT and CBT-TAU treatments included (non-exhaustively) group work, group cohesion, therapist contact, active listening, empathy, and feedback. Both interventions also necessarily involved theory-driven socialisation to the treatment model, in-session behavioural tasks/exercises, and homework setting and reviewing. Furthermore, to match for the non-specific relaxation effects of mindfulness training in ACT, CBT-TAU sessions began with a 10 minute 'relaxation' exercise (e.g., deep breathing, light yoga). Both the ACT and CBT-TAU groups drew resources from self-help books: the CBT-TAU group used "Mind over Mood: Change How You Feel by Changing the Way You Think" (Greenberg & Padesky, 1995), and the ACT intervention used "Get Out of Your

Mind and into Your Life" (Hayes & Smith, 2005). Each group received handouts at the end of treatment sessions to supplement learning and aid homework tasks. Therapists also recommended the target book to aid learning. ACT patients were offered a copy on 6-month loan and CBT-TAU members were offered a library loan.

Specific Treatment Elements. CBT-TAU sessions divided time up so as to address each patient in turn, whereas ACT sessions tended to address the group more globally, at times working with one or two members of the group with others observing. Other techniques considered unique to CBT-TAU included (a) CBT-TAU conceptualisation of psychological difficulties, (b) elicitation and discussions of automatic negative thoughts, schemas, early life experiences, core beliefs, thinking errors and distortions, (c) disputation, challenging and reality hypothesis testing, and (d) patient-to-patient, unguided discussions. Techniques considered unique to ACT included (a) ACT conceptualisation of psychological difficulties (e.g., language based problems, creative hopelessness), (b) acceptance, willingness and de-fusion, (c) mindfulness, and (d) elicitation of, and commitment to, core values. More specific details for the CBT-TAU group are described below (see section 6.2.4.3 and Appendix E for a review of the ACT groups).

Cognitive Behaviour Therapy-Based Treatment-as-Usual. CBT-TAU groups were permitted to run as they would in usual clinical practice, but were requested not to include any mindfulness or acceptance components. Sessions adopted the following broad structure. Early sessions began by socialising patients to the CBT model, proposing that antecedent situations (A) activate beliefs or interpretations about an event (B), which in turn cause certain consequences (C). Pre-determined examples (from the book) were used to convey that the way in which people think and feel about events (their cognitions) determines how they react to those events. Guided discussions were used to explore how different thoughts and feelings may result in different outcomes. This model was extended to in-group guided analysis, using participants' current psychological concerns to explore how the model applied to them. This teaching was supplemented with information regarding cognitive processes such as over-generalised autobiographical recall and selective attention, which were used to explain how cognitions may serve to maintain psychological distress. Homework diary cards were used to aid the identification of idiosyncratic automatic thoughts and beliefs and the role that these had on subsequent behaviour. Where necessary, this was extended

into a discussion of core schema, helping patients to recognise how early experiences may have formed long standing beliefs that everyday life, in conjunction with cognitive biases, might serve to confirm and maintain.

Later sessions focused on teaching CBT-based skills. For example, patients were taught to catch automatic dysfunctional thoughts, to write them down, to evaluate the objective evidence for and against each thought, and to consider possible alternative interpretations. Similarly, behavioural tests and graded task assignments were used both in session and as homework as forms of exposure to feared events. For example, using reality hypothesis testing, participants were helped to devise experiments that would allow then to assess whether anticipated outcomes of feared events were grounded in reality, or whether there was any objective evidence for key over-generalisations. Socratic questioning was used to work through examples of reality hypothesis testing, encouraging participants to notice aspects of their experience that may have been overlooked, and to discuss why these may have gone unnoticed (i.e., selective attention). The last three sessions focused on relapse prevention.

Acceptance and Commitment Therapy. Although the ACT intervention was 0.5 hours shorter than in study 3[30], the content of each session followed the same guidelines described in Table 6.2. The first half of every ACT session began with a mindfulness exercise followed by a review and a discussion of homework. Unlike CBT-TAU, ACT participants were invited to volunteer information rather than being addressed in turn. The second half of each session were concerned with the following stages: (1) Creative Hopelessness, (2) Control as the Problem, (3) Acceptance and Defusion, (4) Defining the Self, (5) Values, and (6) Committed Action (Figure 2.3, Table 6.2, and Appendix E).

7.2.6.3 *Mid-Therapy Assessment.* During treatment I monitored patient well-being using the BDI-II at 4-weekly intervals (data not presented here). After the first, eighth, and last session participants completed the CEQ. After the mid and last session participants completed the HAQ-II. All questionnaires were sealed in envelopes and distributed to participants at the end of a treatment session by the clinicians. To ensure anonymity,

[30] It was not possible for the CBT-TAU groups to run for longer than 2-hours per session. ACT sessions were thus also run for 2-hours so as to match conditions across treatment duration.

participants marked each questionnaire with a memorable ID number before returning the data to the IPTS in the stamped addressed envelope provided.

7.2.6.4 *Post-Therapy Assessment.* Therapists distributed questionnaire packs to participants at the end of the last therapy session and all participants completed them again. The completed packs were brought to the post-intervention interview (conducted by author), during which they described their experiences in the group and the ways in which they felt that the group had or had not been helpful (data not presented here). Those who had dropped out of therapy were not invited to an interview (because the interview was designed to cover group experiences), but they were sent questionnaire packs by post. Six months after attending the last therapy session, all participants were sent the final questionnaire pack to complete before attending a second SCID-II. After all T3 measures had been completed, participants were invited to a 2-hour ACT or CBT-TAU top-up session conducted by the respective clinicians.

7.2.7 *Analysis Strategy*

Preliminary analyses tested for the normal distribution of study variables and for between-group comparability on demographic variables (e.g., age), baseline variables (e.g., GSI), and possible confounding variables (e.g., therapeutic alliance). The main analyses then compared ACT and CBT-TAU on an *analysis by treatment administered* basis (see section 3.4.1). Treatment impact was assessed using a series of repeated-measures ANOVA (one per outcome measure) to test for the effects of Time, Group and Time x Group interactions. Significant effects were further analysed with post-hoc ANOVAs. The magnitude of the difference between the two groups at T2 and T3 was also quantified by calculating Cohen's *d* effect size (ES) per outcome measure.

Owing to the small sample, and the fact that both interventions were expected to produce post-treatment improvements, it was reasoned that analysis of Time x Group interactions might not detect anticipated differential group effects at T3 (i.e., Type II error). Thus, for cases where medium or large ES values were reported at T3, but the Time x Group interaction did not reach conventional levels of significance, post-hoc ANOVAs were nevertheless used to test for within-group differences. Although this

method of analysis would not be acceptable in powered RCTs, it has been employed in other pilot trials to obtain a detailed account of the preliminary findings, which may prove helpful for informing powered trials (e.g., Lynch, Morse, Mendelson, & Robins, 2003). To reduce the probability of committing Type I errors, significant differences in post-hoc analyses were determined using $\alpha = 0.01$[31]. Marginal *p*-values ($.01 < p < .10$) are stated where found.

Three secondary analyses were conducted. Firstly, a series of repeated-measures ANOVAs were computed on an *intention-to-treat* basis (ITT; see section 3.4.1). ITT is a useful analysis strategy because, unlike analysis by treatment administered, it upholds randomisation. However, it simultaneously introduces the problem of missing data. A number of missing value analysis methods were considered (see section 3.4.1), of which LOCF was deemed most appropriate for this trial. LOCF is based on the assumption that participants who discontinue treatment experienced no change from the point of dropping out to post-treatment assessment. Owing to the treatment resistant nature of this group, the assumption of no change was considered to be conservative. Furthermore, most of those dropping out of treatment were re-referred to another form of treatment, most often individual care (see section 7.3.4.5).

Secondly, the clinical significance of change was calculated for each participant and chi-square was used to test whether clinical change differed between groups. Thirdly, mechanisms of change were explored. Owing to the small sample size, formal mediational analyses were not computed. However, exploratory analyses of mechanisms of change were conducted by testing whether theory-driven changes in process measures occurred in each group and whether these changes preceded and predicted outcomes. These mediation-based questions have been recommended by researchers when full mediation cannot be adequately tested (e.g., Hollon, Evans, & DeRubis, 1990). It was predicted that ACT would reduce experiential avoidance and the believability of automatic negative thoughts, and increase valued living and mindfulness. These changes were hypothesised to predict T3 outcomes in this condition. CBT-TAU was predicted to significantly reduce the frequency of automatic negative thoughts, but not significantly alter experiential avoidance, thought believability or

[31] Alpha adjustments, such as Bonferroni, protect against Type I error. When samples are small, however, it is equally important not over-adjust, because this inflates the probability of Type II error. Owing to this, $\alpha = .01$ was considered to be the most appropriate adjustment.

valued living. No firm prediction was made regarding mindfulness. Change in the frequency of automatic negative thoughts was hypothesised to predict treatment gains in CBT-TAU. Finally, simultaneous regression was used to test whether any post-treatment process measures predicted T3 outcomes. Owing to the exploratory nature of these analyses, an adjusted alpha was again used.

7.3. Results

7.3.1 *Preliminary Analysis*

7.3.1.1 *Missing Data and the Distribution of Variables.* For each participant, questionnaire responses were considered valid if they had less than 10% missing data. On the seven occasions that this occurred, participants' mean scores for that scale were substituted for the missing value. For cases where more than 10% was missing, the participants' scores were not included in analysis *for that variable* (i.e., they were included in all other analyses). Four participants had more than 10% data missing for a measure. Sample size thus varied slightly across analyses. All study variables were normally distributed.

7.3.1.2 *Baseline Characteristics and Group Comparability*. As indicated in Table 7.1, most participants scored in the clinical range for global symptom severity and depression and more than a third met the diagnostic and symptomatic criteria for at least one PD. Furthermore, most participants scored within the clinical range for six or more of the psychiatric domains measured by the SCL-90. Between-group comparisons suggested groups were comparable at baseline. There were no significant between-group differences for gender ($\chi^2_{(1, 39)} = .417$, *ns*), age ($t_{(1, 39)} = .034$, *ns*) or number of previous treatments ($\chi^2_{(1, 39)} = 6.80$, *ns*). Similarly, a series of independent *t*-tests indicated that there were no significant between-group differences on baseline indices of psychological distress (GSI (ACT M = 1.63, CBT-TAU M = 1.66) $t_{(1, 39)} = 0.10$, *ns*; BDI (ACT M = 29.65, CBT-TAU M = 28.65) $t_{(1, 39)} = 0.25$, *ns;* WHOQOL (ACT M = 48.89, CBT-TAU M = 45.79) $t_{(1, 39)} = 0.78$, *ns*; and MBQ (ACT M = 2.46, CBT-TAU M = 2.34) $t_{(1, 39)} = 0.84$, *ns*) or process measures (AAQ (ACT M = 5.02, CBT-TAU M = 4.66) $t_{(1, 39)} = 1.44$, *ns;* MAAS (ACT M = 49.95, CBT-TAU M = 53.00) $t_{(1, 39)} = 0.69$, *ns;* ATQ-TF (ACT M = 96.45, CBT-TAU M = 96.70) $t_{(1, 39)} = 0.13$, *ns*; ATQ-TB (ACT

M = 95.45, CBT-TAU M = 96.70) $t_{(1, 39)} = 0.55$, *ns*). The CBT-TAU group did, however, show a greater trend towards *un*valued living (VLQ discrepancy (ACT M = 8.7, CBT-TAU M = 23.50) $t_{(1, 39)} = 1.89$, $p > .05$). No between-group differences were found for treatment credibility/expectancy for change following the first session ($t_{(1, 30)} = 0.12$, *ns*), at 8-weeks ($t_{(1, 22)} = 1.00$, *ns*), or at T2 ($t_{(1, 25)} = 1.01$, *ns*). Similarly, there were no significant differences in therapeutic alliance at 8-weeks ($t_{(1, 27)} = 0.26$, *ns*), or at T2 ($t_{(1, 26)} = 0.49$, *ns*).

7.3.1.3 *Attrition.* Between T1 and T2, eight participants (40%) dropped out of the CBT-TAU group and three from the ACT group (15%). A further CBT-TAU participant dropped out from T2 to T3 assessment. No participants sought external treatment during the 6-months consolidation period. However, an ACT participant who was considered high risk for suicide received 14 individual ACT treatment sessions following the 16-weeks of group treatment. Because this contingency plan was written into the protocol for both conditions (i.e., this was not a deviation from the protocol), his data were included in the analyses[32].

Comparison of completers versus non-completers indicated that the latter had higher baseline GSI ($t_{(1, 39)}$ 2.02, $p = .05$) and AAQ scores ($t_{(1, 39)} = 2.19$, $p < .05$), and lower WHOQOL scores ($t_{(1, 39)} = 2.18$, $p < .05$). Furthermore, 64% (N = 7) of those discontinuing treatment met diagnostic and symptomatic criteria for a PD. Because attrition was greater in the CBT-TAU group, the above analyses were re-run on completers only. This indicated that those completing ACT had a tendency towards greater AAQ scores than those completing CBT-TAU ($t_{(1, 27)} = 1.90$, $p = .07$). The proportion of participants falling into the 'moderate-severe' depression category (BDI > 20; Dozois, et al., 1998) was also marginally higher in the ACT than CBT-TAU group (N = 15/17 versus N = 7/12, respectively; $\chi^2_{(1, 27)} = 3.44$, $p = .06$), and the ACT group had a greater number of participants meeting PD diagnosis (N = 6 versus N = 3).

[32] The findings are comparable both with and without this participant's data in the analyses.

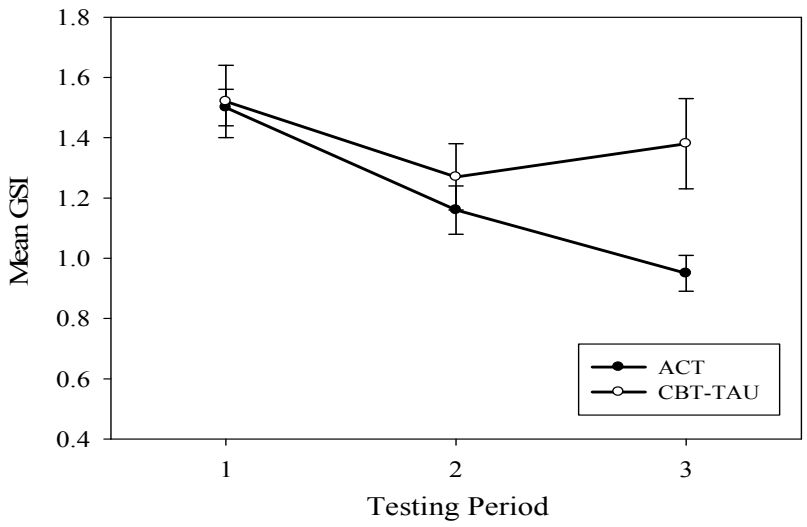
7.3.2 *Main Analysis*

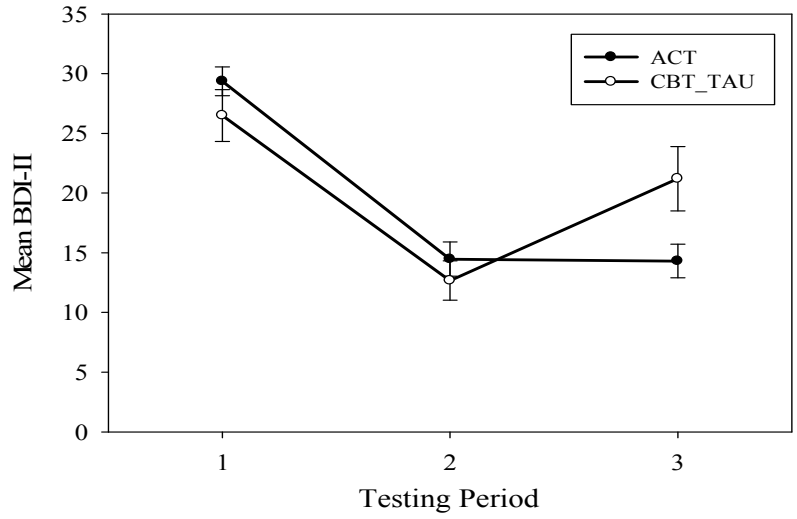
7.3.2.1. *Statistical Significance of Change.* Figure 7.3 visually depicts change in outcome measures, across testing periods, for all participants completing treatment. (SCID-II data are presented separately (section 7.3.2.2) because the SCID-II is a categorical variable). Inspection of these graphs suggests that both ACT and CBT-TAU obtained comparable T2 gains, but differential effects were implied at T3. Specifically, trends suggested that while the symptoms of CBT-TAU participants tended to relapse from T2 to T3, ACT participants either maintained gains (BDI-II), or continued to improve (GSI and WHOQOL).

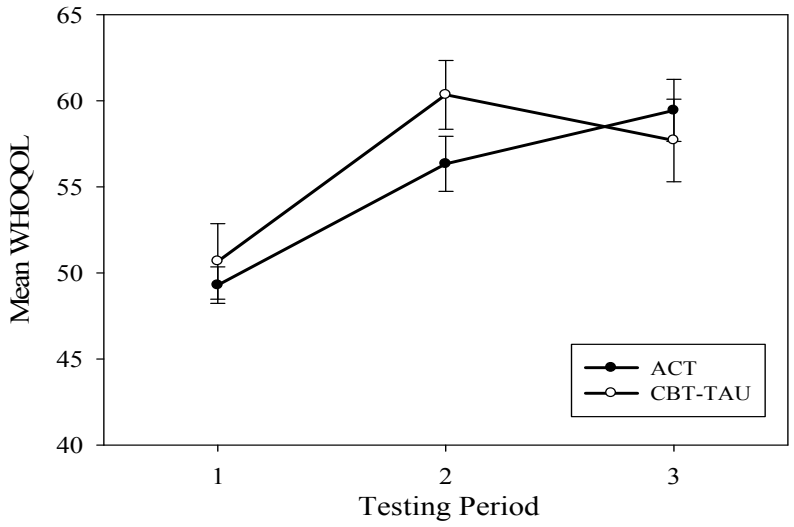
A series of repeated-measures ANOVA (Table 7.2) tested these trends. A significant Time effect was found for all four outcome measures. Post-hoc repeated measures ANOVA (adjusted $p < .01$) revealed a significant reduction in BDI-II scores from T1 to T2 ($F_{(1, 28)} = 43.47$, $p < .001$) and T1 to T3 ($F_{(1, 27)} = 18.31$, $p = .001$) and a significant increase in WHOQOL scores from T1 to T2 ($F_{(1,28)} = 19.31$, $p < .001$) and T1 to T3 ($F_{(1,27)} = 12.25$, $p = .01$). GSI was marginally reduced from T1 to T2 ($F_{(1,28)} = 5.51$, $p = .03$) and significantly reduced from T1 to T3 ($F_{(1,27)} = 7.37$, $p = .01$). For MBQ scores, only a marginally significant reduction was found from T1 to T3 ($F_{(1, 26)} = 4.97$ $p < .05$).

No Group effects were found and only the BDI-II Group x Time interaction approached significance ($p = .06$). Post-hoc analysis of this marginally significant interaction showed significant T1 to T2 reductions in BDI-II for both groups (ACT ($F_{(1, 16)} = 25.21$, $p < .001$), CBT-TAU ($F_{(1, 11)} = 16.82$, $p < .01$)). Change from T1 to T3 was significant only for the ACT group: ACT ($F_{(1, 16)} = 22.67$ $p < .001$); CBT-TAU ($F_{(1, 10)} = 1.56$ $p > .05$). In the CBT-TAU group, a marginally significant *increase* in the BDI-II occurred from T2 to T3 ($F_{(1, 10)} = 3.55$ $p = .09$).

Between-group Cohen's *d* ES values were computed for each outcome measure at T2 and T3 (see Feske & Chambless, 1995). These were calculated by dividing the differences of the group means ($M_{ACT} - M_{CBT}$) by the pooled standard deviation ($\sigma_{pooled} = \sqrt{[(\sigma_1^2 + \sigma_2^2) / 2]}$). Using Cohen's (1988) guide (see section 6.3.1), T2 differences showed small ES values in favour of CBT-TAU for WHOQOL ($d = 0.21$), BDI-II ($d = 0.16$), and MBQ ($d = 0.28$) and in favour of ACT for GSI ($d = 0.14$). At T3, however, ES values were found in favour of ACT for GSI ($d = 0.48$), BDI-II ($d = 0.50$), and WHOQOL ($d = 0.10$). MBQ scores were comparable.







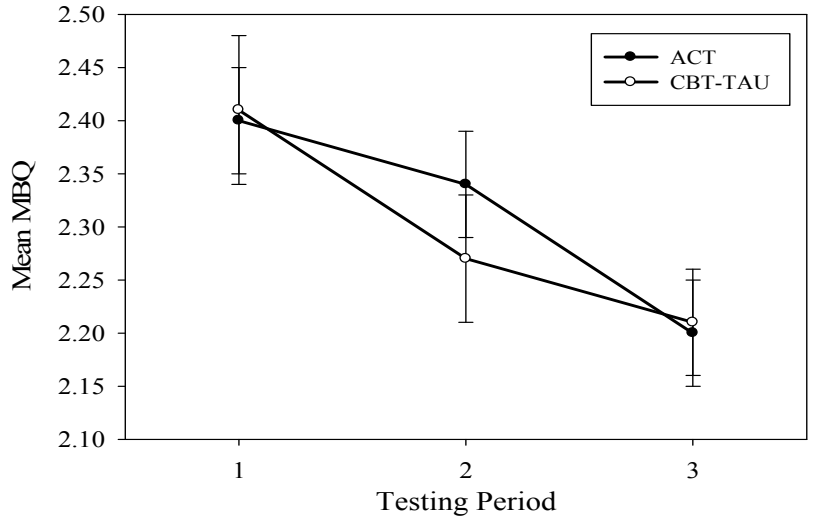


Figure 7.3 Line Graphs (with SE bars) to Show Mean Value on Primary and Secondary Ouctome Measures for Both Groups across Testing Periods.

Table 7.2

Mean (SD) and Repeated Measures ANOVA for Outcome and Process Measures Comparing T1, T2 and T3 Testing in Treatment Completers

	Completing Participants (N = 28)						Repeated-Measures ANOVA		
	ACT (N = 17)			CBT-TAU (N = 11)			Time	Group	Time x Group
Measure	T1	T2	T3	T1	T2	T3			
	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	M (SD)	$F_{(1, 26)}$	$F_{(1, 26)}$	$F_{(1, 26)}$
Outcome									
BDI-II	29.35 (9.98)	14.47 (11.84)	13.71 (11.58)	26.50 (15.16)	12.67 (11.36)	21.20 (17.97)	17.61***	.01	2.97†
WHOQOL	49.28 (8.80)	56.34 (13.48)	59.44 (14.91)	52.76 (16.13)	59.25 (13.36)	57.58 (16.22)	7.04**	.15	.92
GSI	1.50 (0.48)	1.16 (0.66)	0.90 (0.51)	1.52 (0.86)	1.27 (0.86)	1.30 (1.04)	4.23*	.55	1.80
MBQ	2.40 (0.37)	2.34 (0.37)	2.20 (0.38)	2.41 (0.46)	2.27 (0.13)	2.21 (0.31)	4.26*	.13	.41
Process									
ATQ-TF	92.20 (29.39)	67.73 (27.30)	62.00 (31.14)	90.63 (34.51)	64.81 (22.84)	70.63 (30.48)	11.47***	.02	.54
AAQ	4.90 (0.73)	4.10 (0.84)	3.91 (0.72)	4.33 (0.82)	4.30 (0.79)	4.20 (0.83)	8.88**	.02	5.22**
VLQ-R	-10.57 (25.22)	-2.00 (21.26)	0.64 (23.67)	-15.22 (20.47)	2.78 (22.74)	2.88 (28.87)	4.08*	.01	.37
ATQ-TB	92.46 (28.27)	74.80 (30.64)	62.67 (32.40)	91.18 (34.58)	73.63 (32.25)	83.36 (33.57)	3.53*	.49	1.27
MAAS	51.38 (11.15)	57.43 (8.05)	57.81 (13.27)	54.23 (17.80)	57.63 (12.49)	60.27 (15.59)	2.93†	.26	.17

† $p < .10$, * $p < .05$, ** $p < .01$ *** $p < .001$. Note: GSI = Global Severity Index; WHOQOL = Quality of Life; BDI-II = Beck's Depression Inventory; MBQ = Maladaptive Behaviours Questionnaire; ATQ-TF = Automatic Thoughts Questionnaire - Thought Frequency; AAQ = Acceptance and Action Questionnaire; VLQ = Valued Living Questionnaire; AQT-TB = Automatic Thoughts Questionnaire - Thought believability; MAAS = Mindfulness Attention and Awareness Scale. T1 = baseline; T2 = post-treatment; T3 = 6-month follow-up

Although the Time x Group interaction for GSI scores was not significant, the medium ES in favour of ACT at T3 suggested meaningful between-group differences. Post-hoc analyses were therefore computed for the non-significant Time x Group effect. For ACT, this revealed a marginally significant reduction from T1 to T2 ($F_{(1,16)} = 4.16$, $p = .05$), a significant reduction from T1 to T3 ($F_{(1,16)} = 14.07$, $p < .01$), and a marginally significant reduction from T2 to T3 ($F_{(1,16)} = 4.78$, $p = .04$). Within group changes for CBT-TAU were non-significant, however.

7.3.2.2 *Change in Personality Disorder Symptomatology.* Six of the participants completing the ACT group met SCID-II diagnostic and symptomatic criteria for at least one personality disorder at baseline. Of these, 2 remained symptomatic at 6-month follow-up. Three participants completing the CBT-TAU group met criteria for a personality disorder at baseline. Of these, one no longer met the symptomatic criterion at follow-up, one continued to meet the symptomatic criterion and one dropped out of follow-up assessment.

7.3.3 *Secondary Analysis*

7.3.3.1 *ITT Analysis.* Because attrition was selective, ANOVAs were re-run on an ITT basis. The results from these analyses were comparable to those in section 7.2.1. A significant Time effect was found for all outcome measures (GSI ($F_{(1,39)} = 6.50$, $p < .01$), WHOQOL ($F_{(1,39)} = 9.45$, $p < .001$), BDI-II ($F_{(1, 39)} = 20.87$, $p < .001$) and MBQ ($F_{(1, 39)} = 4.61$, $p < .05$). Post-hoc analysis revealed a marginally significant reduction in symptom severity (GSI) from T1 to T2 ($F_{(1,39)} = 5.28$, $p < .05$) and a significant reduction from T1 to T3 ($F_{(1,39)} = 8.20$, $p < .01$). WHOQOL scores significantly improved from T1 to T2 ($F_{(1,39)} = 16.07$, $p < .01$) and T1 to T3 ($F_{(1,39)} = 12.55$, $p < .01$), and BDI-II scores significantly improved from T1 to T2 ($F_{(1,39)} = 33.58$, $p < .001$) and T1 to T3 ($F_{(1,39)} = 18.75$, $p < .001$). Furthermore, there was a significant reduction in MBQ scores from T1 to T3 ($F_{(1,39)} = 6.40$, $p = .01$). No Group effects were found, and only the BDI-II Group x Time interaction neared significance ($F_{(1,39)} = 2.63$, $p = .08$). Post-hoc analysis were used to explore this effect, revealing a significant reduction in depression from T1 to T2 for both groups (ACT ($F_{(1,19)} = 21.03$, $p < .001$) CBT-TAU ($F_{(1,19)} = 12.77$, $p < .01$)). Only the ACT group, however, showed a significant reduction

from T1 to T3 ($F_{(1,19)} = 19.26$ $p < .001$). The CBT-TAU group, on the other hand, showed a marginally significant *increase* in depression from T2 to T3 ($F_{(1, 19)} = 3.47$, $p = .09$).

7.3.3.2 *Clinical Significance of Change.* Jacobson and Truax's (1991) criteria for clinically meaningful and reliable change were computed for GSI and BDI-II scores (treatment completers only). This analysis compared T1 to T2 and T1 to T3 scores using the same classification system outlined in section 3.4.2 and 6.2.5. Results are shown in Table 7.3 and Figure 7.4. GSI scores at T2 showed that a greater proportion of ACT than CBT-TAU participants had recovered or improved following treatment (41% (N = 7) and 24% (N = 3) respectively). Similarly, fewer ACT than CBT-TAU participants stayed the same or deteriorated (59% (N = 10) and 76% (N = 9) respectively). The distribution of change across conditions comparing 'same or deteriorated' and 'recovered or improved' was not, however, significant ($\chi^2_{(1,28)} = 1.10$, $p > .05$, see Figure 7.4). At T3, a greater proportion of ACT than CBT-TAU participants had recovered or improved (59% (N = 10) and 18% (N = 2) respectively), and fewer ACT than CBT-TAU participants stayed the same or deteriorated (41% (N = 7) and 82% (N = 9) respectively). These between-group differences were significant ($\chi^2_{(1,27)} = 3.84$, $p < .05$, see Figure 7.4).

The same analysis, conducted on BDI-II scores, showed that at T2 most ACT participants had recovered (65%, N = 11); five stayed the same and one worsened. Conversely, for CBT-TAU, a minority had recovered or improved (33%, N = 4) and the rest stayed the same (67%, N = 8). The distribution of change was not significantly different between conditions ($\chi^2_{(1, 28)} = 4.22$, $p > .05$). At T3 most ACT participants had recovered (65%; N = 11), five stayed the same and one deteriorated, whereas most CBT-TAU participants stayed the same or had deteriorated (82%, N = 10). Analysis showed a significant between-group difference in the distribution of change when comparing 'same or deteriorated' with 'recovered or improved' ($\chi^2_{(1, 27)} = 8.59$, $p = .01$, see Figure 7.4). These analyses thus indicate that at T3, a greater number of ACT than CBT-TAU participants had recovered or improved on GSI and BDI-II measures.

Table 7.3

Percentage of Clinically Significant Change Split by Group

	GSI				BDI-II			
	T2		T3		T2		T3	
	ACT	CBT-TAU	ACT	CBT-TAU	ACT	CBT-TAU	ACT	CBT-TAU
Recovered (%)	35	8	41	18	65	25	65	9
Improved (%)	6	16	17	0	0	8	0	0
Same (%)	47	68	35	72	29	67	29	81
Deteriorated (%)	12	8	6	8	6	0	6	9

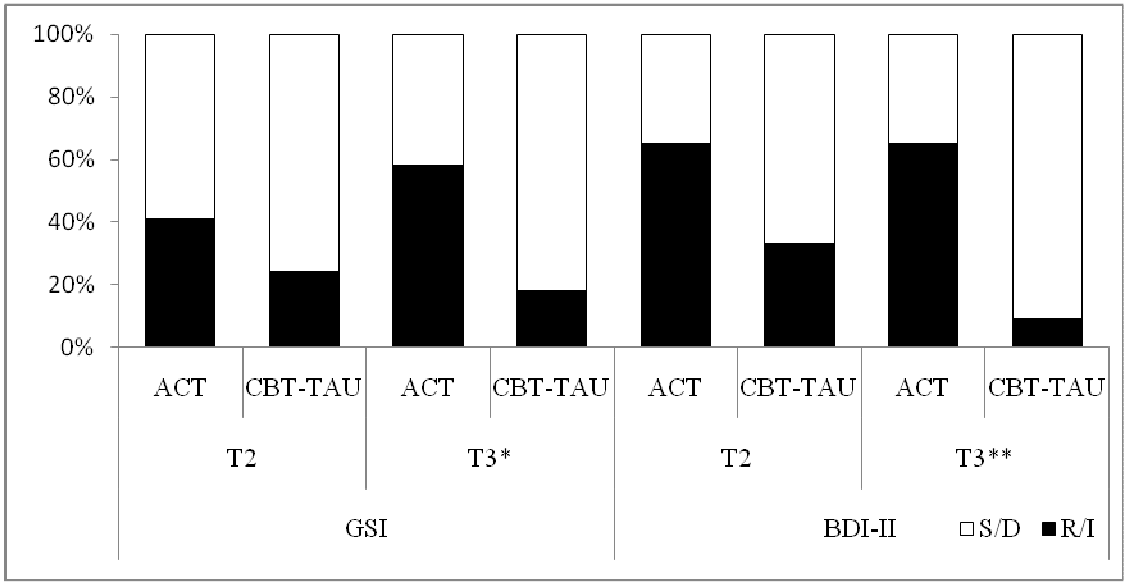


Figure 7.4 Percentage of Participants 'Recovered/Improved' (R/I) and 'Same/Detereorated' (S/D) for ACT vs. CBT-TAU on GSI and BDI-II scores at T2 and T3.

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

7.3.3.3 *Exploratory Mechanisms of Change.* Based on ACT theory (see section 2.2), it was predicted that ACT would reduce AAQ and ATQ-TB and increase MAAS and VLQ-R scores. For CBT-TAU, only a reduction in ATQ-TF was expected (see section 1.2). As reported in Table 7.2, a significant Time effect was found for all process measures. Further analysis of Time effects showed a significant T1 to T2 reduction in AAQ ($F_{(1, 28)} = 9.19$, $p < .01$), and ATQ-TF ($F_{(1,28)} = 26.29$, $p < .001$), a marginally significant reduction in ATQ-TB ($F_{(1,28)} = 5.23$, $p = .03$) and MAAS ($F_{(1,28)} = 3.37$, $p = .07$) and no change for VLQ-R. Comparing T1 to T3 scores, a significant reduction occurred in AAQ ($F_{(1,27)} = 16.04$, $p < .001$), ATQ-TF ($F_{(1,26)} = 15.37$, $p = .001$) and ATQ-TB ($F_{(1,26)} = 8.63$, $p < .01$), and a marginally significant reduction (adjusted $p \leq .01$) was observed for VLQ-R ($F_{(1,26)} = 5.56$, $p = .03$) and MAAS ($F_{(1,27)} = 4.73$, $p = .04$). The only significant Time x Group interaction observed was for the AAQ. Post hoc comparisons indicated significant reductions in AAQ scores only for the ACT group: T1 to T2 ($F_{(1, 16)} = 12.32$, $p < .01$), T1 to T3 ($F_{(1,16)} = 22.48$, $p < .001$).

To assess whether change in processes measures during treatment predicted T3 outcomes, multiple regressions were computed for those measures showing significant T1 to T2 change (i.e., AAQ and ATQ-TF). These analyses controlled for baseline measures of the outcome variable in block 1 of the regression (i.e., T1 GSI, QOL, BDI-II, MBQ), and regressed T3 values of the outcome variable (i.e., T3 GSI, QOL, BDI-II, MBQ) on to the residual gain (RG)[33] of AAQ/ATQ-TF in block 2. Because differential treatment mechanisms were predicted, separate regressions were computed for each condition. For ACT, regression analyses used AAQ RG and ATQ-TF RG as the predictors (one regression per predictor); for CBT-TAU, ATQ-TF RG was the predictor (because AAQ scores did not change in the CBT-TAU condition).

Results for the ACT group showed that AAQ RG scores were significantly predictive of T3 GSI ($\beta = .787$, $p = .001$), T3 BDI-II ($\beta = .662$, $p < .01$) and marginally predictive of T3 WHOQOL ($\beta = .467$, $p = .03$), and MBQ ($\beta = .386$, $p = .09$) scores. ATQ-TF RG scores were predictive of T3 GSI only ($\beta = .686$, $p < .01$). Because AAQ and ATQ-TF RG scores both significantly predicted T3 GSI, a simultaneous multiple regression was computed in which T3 GSI was regressed on to both predictors simultaneously in block 2 of the analysis. Results showed that AAQ RG scores were marginally predictive of T3

[33] Recall that RG is the difference score from T1 to T2, adjusted for dependent imposed by repeat testing.

GSI (β = .629, p = .03), but ATQ-TF RG scores were not (β = .253, p > .05). In the CBT-TAU group, ATQ-TF RG scores did not significantly predict any T3 outcomes (GSI (β = .140), QOL (β = .124), MBQ (β = .04), or BDI-II (β = .318; all p > .10). These findings thus suggest that changes in the AAQ were predictive of T3 outcomes for the ACT condition, but changes in ATQ-TF were not predictive of T3 outcomes in the CBT-TAU condition.

Finally, to test whether any T2 process measures could predict T3 outcomes, a series of simultaneous regression analyses were computed, one per outcome. These analyses simultaneously regressed each T3 outcome measure on to all T2 process measures. Using the data of both groups, results (Table 7.4) showed that only T2 AAQ scores predicted T3 outcomes.

7.3.3.5. *Reduced Re-referrals?* The treatment of individuals with a long history of re-referral was central to this trial. For this reason, the proportion of patients either awaiting or receiving psychological treatment at the end of the trial (i.e., after T3 assessment) was obtained. This indicated that of the 17 participants completing ACT, one continued to receive individual ACT sessions from 'therapist one' after treatment had ended. Of the three that discontinued treatment, one was awaiting further psychological treatment from DHFT. Of the 12 participants completing CBT-TAU, three were receiving individual psychology 1 month after T3 assessment. Another one was awaiting treatment. Of the eight who discontinued treatment, five were awaiting or already receiving alternative treatment. Thus, 2/20 ACT allocated participants and 9/20 CBT-TAU allocated participants were awaiting or receiving treatment following this trial. This difference was significant ($\chi^2_{(1,39)} = 6.14$, p < .05).

Table 7.4

Simultaneous Regressions Predicting T3 Outcomes from T2 Process Measures

Outcome	T3 GSI			T3 WHOQOL			T3 MBQ			T3 BDI-II		
Predictor	*B*	*SE B*	β	*B*	*SE B*	β	*B*	*SE B*	β	*B*	*SE B*	β
T2 AAQ	.044	.024	.425†	1.18	.554	.538*	.002	.015	.036	1.18	.396	.663**
T2 ATQ-TF	.007	.007	.256	.097	.151	.165	.001	.004	.074	.035	.108	.072
T2 ATQ-TB	.003	.005	.120	.039	.105	.079	.000	.003	.006	.027	.075	.081
T2 MAAS	.002	.015	.021	.149	.343	.094	.010	.009	.280	.044	.245	.181
T2 VLQ-R	.009	.007	.279	.031	.136	.044	.000	.004	.023	.097	.097	.172
Overall R^2		.460*			.362			.107			.501*	

† $p < .10$; * $p < .05$ ** $p < .001$

Note: T2 = Post-test; T3 = Follow-up; GSI = Global Severity Index; QOL = Quality of Life; MBQ = Maladaptive Behaviours Questionnaire; BDI = Beck's Depression Inventory; AAQ = Acceptance and Action Questionnaire; ATQ-TF = Automatic Thought Questionnaire - Thought Frequency; AQT-TB = Automatic Thought Questionnaire -Thought Believability; MAAS = Mindfulness Attention and Awareness scale; VLQ-R = Revised Valued Living Questionnaire (value discrepancy).

7.4 Discussion

7.4.1 *Study Findings*

Previous research suggests that cognitive interventions are not an effective means of treating all adult mental health patients, with approximately 30-50% either showing no change or relapsing after treatment (see section 1.2.4). It is important, therefore, to refine existing techniques and to establish new ones for this diagnostically heterogeneous group. With this in mind, the primary aim of this study was to pilot test ACT versus CBT-TAU for patients whose symptoms have been resistant to, or re-occurred following, previous psychological treatment. Based on previous literature, it was predicted that ACT would obtain larger and more durable effects than CBT-TAU. The secondary aim of this study was to test whether theory-driven mechanisms of change could differentially account for treatment gains in each group. Based on the theoretical models discussed in chapter 1 and 2, it was predicted that experiential avoidance, mindfulness, cognitive defusion, and valued living would predict change in the ACT group. Conversely, reductions in thought frequency were expected to predict change in the CBT-TAU group.

These predictions were tested using a methodology that was sensitive to issues of both internal and external validity. For example, the sample presented with a range of psychological problems and many reported co-comorbid and/or PD symptoms. It can thus be argued that, compared to the 'pure' homogeneous samples typically recruited to RCTs (Westen et al., 2004), this heterogeneous group better approximated the characteristics of patients usually seen in clinical settings. Treatments were also delivered in a manner that aimed to reflect real life treatment conditions. Sessions were guided by broad treatment protocols, retaining some flexibility to meet individual patient needs. Similarly, clinicians were able to provide participants with additional one-to-one treatment if deemed necessary. Although these features of the trial tend to challenge internal validity, the design included important features to increase confidence in the findings. For example, participants were randomised to treatments and many confounds were controlled or monitored, such as treatment duration, therapeutic alliance, and treatment credibility.

The main findings of this trial suggested that both interventions reduced mean levels of psychological distress, reduced the tendency to engage in MBs, and improved quality

of life scores. These Time effects remained significant even when evaluated using a more conservative ITT analysis strategy. This suggested, somewhat contrary to expectation, that *both* interventions produced significant treatment effects. Trends in the data, however, suggested that the effects of these two interventions were not fully comparable over time. Although the power of the study was not sufficient to detect any significant Time x Group interactions, group means strongly suggested that change was more durable for the ACT condition. This implication was supported by several findings. Firstly, follow-up scores indicated medium effect sizes in key outcome measures that were in favour of ACT. Secondly, a greater proportion of ACT than CBT-TAU participants had follow-up scores that were within the 'normal' range and that were reliably better than at baseline. Thirdly, within group analyses showed that ACT obtained significant baseline to follow-up changes that were not observed in the CBT-TAU condition.

Together, these findings suggest that while the symptoms of the CBT-TAU group tended to relapse following treatment, ACT members either maintained gains (BDI-II) or continued to improve (GSI, WHOQOL). This occurred despite the fact that, compared to CBT-TAU, ACT clinicians treated larger groups that tended to be more symptomatic at baseline. Owing to the design of the study, these differences were unlikely to be attributable to expectancy for change, intervention credibility, and/or therapeutic alliance. These findings support the conclusion that both interventions produced short term benefits, but that ACT achieved more enduring change seemingly by preventing relapse. MBQ scores were an exception to this trend, however. Although the MBQ proved sensitive to detecting changes over time, no between-group differences were found. This finding was anomalous in relation to the other outcome measures and requires further investigation. It is possible that the null effect observed was related to the fact that patients selected for this trial were not engaging in high levels of risk behaviours. This is speculative, however, and should be addressed in future work.

In addition to investigating symptom change, exploratory analyses of mechanisms of change were also carried out. Consistent with prediction, ACT produced significant T1 to T2 reductions in the AAQ that were significantly predictive of T3 outcomes. Because change in experiential avoidance during treatment preceded and predicted outcomes at T3, the associations observed are supportive of a cause-and-effect model. Furthermore, post-treatment AAQ scores were uniquely predictive of follow-up outcomes (GSI,

QOL, BDI-II), suggesting that participants with a high AAQ score at post-treatment were more symptomatic at 6-months. An unexpected finding was that ACT also significantly reduced thought frequency and this reduction appeared predictive of symptom severity at follow-up. However, further analysis indicated that a change in the frequency of negative thoughts was not predictive of symptom severity after accounting for the effect of change in the AAQ. Consistent with ACT theorising, therefore, these findings suggested that accepting unwanted private events, rather than merely experiencing or not experiencing those events, was the main agent of change. Exploratory analysis of change in the CBT-TAU group showed that although anticipated reductions in thought frequency occurred, this change was unrelated to outcome.

To summarise: these results suggest that in keeping with study 3, ACT had the capacity to achieve enduring change in patients who might otherwise had been classed as *treatment resistant*. Although some participants did not experience significant mental health improvements, ACT appeared to have greater potential for change than CBT-TAU. Furthermore, preliminary data suggests that ACT achieved these gains in theory consistent ways. In contrast, CBT-TAU tended to obtain short term gains that were not sustained and that were not predicted by reductions in the frequency of negative thoughts.

7.4.2 *Methodological Limitations*

Although the present study produced promising results it must be evaluated in the light of some limitations. Firstly, it is most probable that the sample size prohibited the detection of anticipated Group x Time interactions, especially given the high within group variability. Although within group analyses provided some insight into the differential effects of the two treatments, these analyses were based on non-significant interactions. These findings should thus be considered as tentative; providing a firm basis for replication and extension. Power calculations based on the present data suggest that the anticipated Group x Time interaction could be detected with a sample of 65 participants per group (calculations based on a medium between-group ES, 2 group comparison, 5% significance level, and 95% power).

The second limitation concerns the fidelity of the two treatments to their respective models and techniques. Although all sessions were recorded and a collection were informally checked for treatment integrity, it was simply not feasible for a non-biased member of the clinical team to rate the content of these recordings. (For ethical reasons, only members of this team were allowed access to the tapes). No evidence can therefore be offered to prove that treatments were delivered in keeping with ACT and CBT models. Internal evidence suggesting treatment fidelity was, however, implied by (a) theory consistent shifts in process measures and (b) the use of resources drawn from ACT and CBT treatment books.

Another concern regarding the treatments is their comparability across factors that were not measured. One ACT therapist, but neither CBT-TAU therapist, had personal involvement in the trial. Similarly, CBT was TAU, but ACT was a novel treatment. Non-measured, non-treatment specific factors, such as greater enthusiasm or organisation on the part of ACT therapists, may thus have contributed to change. Counter to this rival hypothesis, however, is the fact that groups were comparable in their expectancy for change and differential effects arose long after the end point of treatment.

7.4.3 *Implications*

This trial has provided evidence to suggest that ACT could be an acceptable and beneficial intervention for treatment resistant patients. This was shown by its ability to obtain more durable and clinically significant effects than its ecologically valid counterpart within the DHFT. Moreover, in keeping with the ACT model, results suggested that failure to address experiential avoidance during treatment could be prognostic of poor future outcomes. Although it is necessary to test whether these findings hold in a trial that addresses some of the limitations raised (see section 8.2), the present study suggests that the treatment of repeat service users could be greatly improved by using ACT. From the perspective of the service provider (i.e., DHFT), ACT appears to have made a greater contribution to the reduction of re-referral rates than CBT-TAU. From the perspective of the consumer, low attrition in the ACT group suggested this treatment was a more acceptable option than CBT-TAU, especially for those patients with high experiential avoidance and meeting PD diagnostic and

symptomatic criteria (characteristics of drop-out participants). Furthermore, ACT achieved more durable and clinically meaningful change than CBT-TAU. ACT thus presents itself as an economically feasible and promising treatment for this group. Ideas for future research are discussed in chapter 8.

CHAPTER VIII
GENERAL DISCUSSION

For many decades, psychologists have worked to understand how clinical disorders are established and maintained, with the ultimate aim of developing effective treatment programmes. Traditionally, this has involved discriminating disorders topographically (e.g., DSM-IV, 2000) and developing disorder-specific treatments. This has been based on the understanding that disorder-specific treatments are integral to successful outcomes. I began this thesis by identifying a trans-diagnostic group of patients whose resistance to standard care treatment continues to challenge the strained resources of the National Health System (NHS). The main aim of this thesis, therefore, was to develop and test a novel and economically feasible treatment for this 'treatment resistant' cohort. Based on existing literature, ACT was proposed as a promising candidate therapy. This was by virtue of the fact that, according to ACT theorising, the formally dissimilar symptoms that this group present with are commonly maintained by excessive levels of experiential avoidance. Because this application was novel, the work presented in this thesis focused on the first two stages of treatment evaluation described in chapter 3. First, based on the observation that treatment resistant patients often engage in maladaptive behaviours, analogue research was designed to test the ACT assumption of their common experiential avoidance function. Second, pilot trials were carried out to test run ACT for treatment resistant patients. This chapter aims to summarise the main findings, discuss possible implications, and make suggestions for future research.

8.1 Main Findings

8.1.1 *Analogue Research*

Although theorists have for many years discussed the negative reinforcement properties of addictive substances (e.g., Baker et al., 2004; Wikler, 1948), few have provided a wide ranging account of maladaptive behaviours. ACT theorising offers a parsimonious and integrated model, which proposes that these behaviours co-vary because of a shared experiential avoidance function. As reviewed in chapter 2 and 4, however, this is a long held assumption that was in need of investigation. In fact, although this is a key tenet of the ACT model, assumptions regarding the link between maladaptive behaviours and

experiential avoidance have mostly been inferred using data on experiential avoidance-like constructs, such as avoidant coping and thought suppression (see section 2.1.4).

Using the AAQ to index experiential avoidance, some evidence was presented to support the 'common experiential avoidance hypothesis' of maladaptive behaviours (studies 1 and 2). Extending previous research (e.g., Cooper et al., 2003), study 1 used structural equation modelling techniques to show that a wide range of clinically relevant behaviours could be conceptualised as sharing a common cause or function of some kind. Aiming to elucidate the nature of this shared commonality, it was found that the AAQ accounted for a significant proportion of maladaptive behaviour covariance. This finding, which was replicated in study 2, suggested that one of the reasons maladaptive behaviours co-vary is because of their shared experiential avoidance function.

After the isolated bivariate relationship between experiential avoidance and maladaptive behaviours had been identified, research designed to understand this relationship more fully could be developed. Specifically, study 2 tested whether experiential avoidance was one of the reasons why negative affect intensity and childhood trauma increase the probability of engaging in maladaptive behaviours. Overall, results were supportive of the hypothesised mediational models; experiential avoidance reduced a substantial proportion of the effect of negative affect intensity, and a slightly more moderate proportion of the effect of childhood trauma, on the tendency to engage in these behaviours. These findings were consistent with the hypothesis that people who experience negative affect intensely and/or who have experienced childhood trauma are more likely to engage in maladaptive behaviours in an attempt to escape, avoid, or reduce contact with unwanted private experiences. Owing to the cross-sectional design that was used, however, rival hypotheses regarding the direction of causation could not be ruled out.

In addition to these main findings, both studies also suggested a differential association between experiential avoidance and maladaptive behaviours in the self-declared clinical as compared to the non-clinical group. In particular, both studies suggested that experiential avoidance was related to a greater range of behaviours in the clinical sample. Furthermore, associations tended to be of greater magnitude in this subgroup. This suggests that experiential avoidance is especially implicated in maladaptive behaviours exhibited by treatment seeking samples. It is possible that this occurs because this group present with higher levels of experiential avoidance. This

could not be ascertained with certainty, however, because other non-measured between group differences could have produced similar findings.

Overall, this theory-testing phase of research supported three main conclusions: (1) experiential avoidance is one of the reasons why dissimilar maladaptive behaviours co-vary; (2) individuals who are high in emotionality and/or who have experienced childhood trauma are more likely to engage in problem behaviours, in part, because of heightened levels of experiential avoidance; (3) maladaptive behaviours are more likely to serve an experiential avoidance function in treatment seeking samples. I argued that the findings of these two studies, in conjunction with existing research (e.g., Gratz & Gunderson, 2007; Gratz et al., 2008; Hayes, Wilson, et al., 2004), provided sufficiently compelling evidence to embark on the novel application of ACT to treatment resistant patients.

8.1.2 *Applied Research*

Although previous research had shown promising effects for ACT with acute psychiatric disorders, little was known about its applicability to treatment resistant patients. A pilot phase of investigation was thus considered prudent.

Study 3 used a pre-post uncontrolled trial to test run ACT for this group. Despite many factors that might have mitigated against it, significant effects were found. ACT was associated with medium to large effect sizes, and significant improvements were reported for several indices of psychological distress, the use of alcohol, and quality of life. Furthermore, 50%-70% of participants achieved significant improvements on key outcome measures and none were awaiting or receiving further psychological treatment at 12-month follow-up. Although investigations into mechanisms of change could be no more than exploratory, they were nonetheless consistent with ACT-based predictions. Reductions in experiential avoidance and thought believability, and increments in mindfulness and valued living during treatment were associated with several 6-month and 12-month outcomes. Therefore, although no firm conclusions could be made regarding cause-and-effect relations or the generalisability of findings, study 3 provided good evidence to support the continuation of this research.

Study 4 was designed to evaluate ACT more rigorously by comparing it to an active comparison group (CBT-TAU). This trial indicated that although ACT and CBT-TAU

obtained comparable post-treatment effects, only ACT was associated with durable gains. For example, at follow-up, ACT was associated with significantly greater rates of clinical improvement than CBT-TAU. Additionally, medium effect sizes were found in favour of ACT. Moreover, ACT, but not CBT-TAU, obtained significant pre-treatment to follow-up changes. In an attempt to elucidate these differential effects, exploratory mediational analyses were conducted. These analyses suggested that reductions in experiential avoidance during ACT predicted follow-up outcomes. In the CBT-TAU group, however, reductions in the frequency of automatic negative thoughts were unrelated to outcome. Furthermore, regardless of group, post-treatment levels of experiential avoidance were uniquely predictive of follow-up outcomes, even when controlling for the frequency of automatic negative thoughts. This tentatively suggests that levels of experiential avoidance at post-test influenced psychological functioning at 6-months.

In addition to these main findings, both trials also indicated that ACT group attrition rates (23%) were substantially lower than average rates in published RCTs (50%; Hansen, Lamber, & Forman, 2002) and those of the comparison group in study 4 (CBT-TAU; 45%). Unlike CBT-TAU, which tended to lose participants with PD symptoms, higher baseline severity, and excessive experiential avoidance, ACT retained participants with these characteristics. Overall, therefore, a time-limited, group-based delivery of ACT appears to have been acceptable for treatment resistant patients.

Although these two pilot trials have some limitations, and the generalisability of their findings is unknown, they nonetheless support several tentative conclusions: (1) ACT can meaningfully improve the psychological functioning of treatment resistant patients; (2) ACT could offer a means of obtaining more long term change than standard care for this group; (3) ACT can achieve change in theory consistent ways; (4) experiential avoidance is implicated in the durability of treatment outcomes; and (5) a group-based, time-limited delivery of ACT is a viable and acceptable option for treatment resistant patients. Overall, the findings presented in this thesis have thus been largely supportive of the theoretical underpinnings of ACT and its applicability to treatment resistant patients. They must be considered in the light of several strengths and limitations, however.

8.1.3 *Main Strengths and Limitations*

8.1.3.1 *Strengths.* Reflecting on the programme of research as a whole, it appears to have had some strengths. Firstly, a number of gaps in current knowledge were identified and theoretically grounded principles were used to try to enhance understanding in those areas. Secondly, the research was conducted in a systematic manner, embedded within a recommended stage-based approach to evaluating novel treatments. Thirdly, the integration of analogue and applied research provided a broad assessment of ACT's theoretical applicability to diverse samples and different clinical phenomena. Fourthly, the multi-method approach (e.g., cross-sectional non-experimental design, prospective experimental design) provided convergent evidence in support of the ACT model. Because findings from different methods converged, support for this model is less likely to have been confounded by any one methodological weakness. Similarly, replicating the main findings across separate and diverse samples reduces the probability that the findings are attributable to idiosyncrasies of a certain sample, such as undergraduates. Finally, the use of advanced statistical techniques offered a modern and flexible means of testing ACT predictions. Although this type of hypothesis testing is in stark contrast to traditional behavioural approaches, it has provided a parsimonious means of testing ACT's broad predictions.

8.1.3.2 *Limitations.* These strengths are offset by some unavoidable limitations. Firstly, although the research was designed to flow neatly from analogue to applied investigations, this flow was partially disrupted by the need to exclude patients who were engaging in 'at-risk' behaviours (studies 3 and 4). Because maladaptive behaviours are an important element of treatment resistance, this limits the generalisability of findings to the treatment resistant population. Nevertheless, the work has provided a broad analysis of the common experiential avoidance hypothesis using a range of dependent variables that have specific relevance to this group.

A second main limitation was that all work relied on self-report measures. Although this was ethically defendable, self-report is subject to several sources of inaccuracy, such as demand bias and memory distortions (see section 3.2.1). Several efforts were made to reduce the effect of known confounds. For example, in each study, participants completed measures in their own time and anonymously. Also, each study randomised

the order of questionnaires so as to prevent systematic carry over effects. Furthermore, associations between variables were consistently based on statistical analyses rather than self-reported associations. Also, efforts were made to reduce the possibility of artificially inflated associations. For example, the MBQ was designed to measure the tendency to engage in maladaptive behaviours in a manner not confounded by motivations to engage.

A third limitation is that, although all studies had broad and unrestrictive inclusion criteria, some groups were nevertheless underrepresented. Specifically, the findings of studies 1 and 2 may not generalise to males and the findings of studies 3 and 4 may not generalise to patients with more entrenched disorders (e.g., Borderline Personality Disorder, see section 8.3.1.3). Finally, by virtue of investigating the novel application of a modern treatment, the sample sizes of the applied work have been small. The findings in this thesis thus, quite naturally, only tell part of an unfolding story.

8.2 Implications and Suggestions for Future Research

Having reviewed the main findings of this thesis, the second aim of this chapter is to consider their possible implications and to discuss fruitful areas for future investigation. This will begin by discussing maladaptive behaviours, followed by the treatment of treatment resistant patients, and will finish by considering the broader field of ACT literature as a whole.

8.2.1 *Understanding Maladaptive Behaviours*

Studies 1 and 2 tested, and found some evidence to support, the common experiential avoidance hypothesis of maladaptive behaviours. Despite the formal dissimilarity of the many behaviours investigated, the higher order factor model provided a parsimonious account of behaviour covariances. Furthermore, experiential avoidance accounted for a significant proportion of that covariance. The possible implications of these findings are discussed below.

8.2.1.1 *Implications of Commonality of Functions for Symptomatic Treatments and Syndromal Classifications.* One implication of the functional equivalence of maladaptive behaviours is that they could be treated using principle-based approaches designed to undermine common causes or functions, such as experiential avoidance. This can be contrasted to the more syndrome-specific and protocol-based treatment approach, which focuses on understanding the topographical form of the core presentation and aims to effect a change in that syndrome directly. The functional approach has several strengths. It provides key principles that can efficiently guide the treatment of co-occurring behaviour problems, focusing on their functional similarities (e.g., experiential avoidance, rule-governed behaviour) rather than formal dissimilarities (e.g., binge eating, substance abuse, and so on). Indeed, the co-occurrence of problem behaviours has traditionally challenged syndrome-specific treatment programmes, because it is often unclear which behaviour to treat and when (see Conrad & Stewart, 2005; Westen et al., 2004). Although the functional approach seems to be particularly suited to the treatment of co-occurring behaviour problems, it could also be useful for individuals who present with a singular or core maladaptive behaviour. This is because reducing the occurrence of that core behaviour can increase the probability of engaging in others (i.e., behaviour switching; Donovan, 1988). In theory, functional treatment approaches could have the capacity to prevent this phenomenon from occurring.

Commonality of function also has implications for how maladaptive behaviours are conceptualised. Contemporary psychiatry, which strongly influences psychological treatments and their evaluation, understands clinical phenomena in terms of formal symptoms. Consequently, maladaptive behaviours are defined mainly on the basis of features that make them dissimilar from one another. Although this approach is particularly useful for communicating and generalising findings across settings, it has several weaknesses. Most importantly, at least from the perspective of this thesis, it de-emphasises their commonalities and their high co-occurrence (see section 4.1). Studies 1 and 2 suggested that a functional diagnostic system, such as understanding dissimilar clinical phenomena in terms of their common experiential avoidance function (Hayes et al., 1996), could offer a more parsimonious approach. Nevertheless, this model requires a more fine-grained level of detail than is currently available. For example, further research is required to understand more fully the learning histories or predispositional factors that determine the specific behaviour topography that an individual adopts.

Similarly, research could be designed to elucidate additional factors that account for unexplained variance that maladaptive behaviours share.

8.2.1.2 *Additional Mediators and Moderators of Maladaptive Behaviours.* Although the higher order factor model adequately accounted for maladaptive behaviour covariation, unique factors also appeared to play an important role for many of the behaviours under investigation. Similarly, a large proportion of covariance was not accounted for by experiential avoidance. Impulsivity is one factor that could help to unpack the relationship between experiential avoidance and maladaptive behaviours more fully.

Impulsivity is a multi-component concept that refers to the tendency to give in to urges and impulses, to act hastily without forward planning, and to show poor task perseverance (see Whiteside & Lynam, 2001). Impulsivity is reliably related to risk taking (e.g., Grano et al., 2004), but its relation to experiential avoidance is not well understood. Cooper et al. (2003) cast some interesting light upon the possible ways in which these two variables might interact. Investigating avoidant coping, they reported that individuals high in both avoidance *and* impulsivity were particularly vulnerable to problem behaviours. This finding makes intuitive sense; individuals predisposed to act without forethought who are also motivated to alleviate distress may be less likely than others to consider adaptive, low-risk means of affect regulation. It is also possible that interactions between experiential avoidance and impulsivity could determine which of the range of behaviours becomes predominant. For example, high experiential avoidance plus high impulsivity could be predictive of behaviours that, through some action, provide immediate relief or gratification (e.g., drug or alcohol use). Conversely, high experiential avoidance coupled with low impulsivity could lead to more passive forms of risk taking such as restrictive eating. Exploring multivariate relations between experiential avoidance and other variables, such as impulsivity, could help to understand maladaptive behaviours and to develop a functional diagnostic system more broadly.

8.2.1.3 *Treating Maladaptive Behaviours.* Although experiential avoidance only accounted for a proportion of maladaptive behaviours covariance, the present findings suggest that acceptance-based and mindfulness-based interventions could be valuable

for treating these behaviours. Although this suggestion seems to be at odds with study 4, which showed that the AAQ was unrelated to MBQ scores, it is possible that this occurred because patients who were actively engaging in high risk behaviours were excluded from the trial. Certainly the theoretical implications of studies 1 and 2 are consistent with recent research on the efficacy of acceptance-based and mindfulness-based techniques for several risk behaviours. These have included, for example, recovery from polysubstance abuse (Hayes, Wilson, et al., 2004), nicotine addiction (Gifford et al., 2004), DSH (Gratz & Gunderson, 2006; Low, Jones Duggan, Power, & MacLeod, 2001), binge eating (e.g., Telch et al., 2000) and alcoholism (Marlatt et al., 2004).

A possible extension of the present findings into clinical practice could involve using acceptance-based and mindfulness-based techniques in a preventative, early intervention scheme, designed for individuals at-risk for developing behaviour problems. This could include, for example, individuals who have experienced childhood trauma, individuals who experience negative affect intensely, or perhaps those with low social economic status (another known risk factor). Such an intervention could be designed to target these individuals before experiential avoidance manifests in maladaptive behaviour patterns. This could involve, for example, helping individuals to make undefended contact with unwanted private experiences and helping them to identify valued domains. Acceptance-based and mindfulness-based techniques could also prove useful for achieving and maintaining abstinence in patients who have already developed entrenched patterns of maladaptive behaviour. For example, ACT could help patients to mindfully observe stimuli that cue self-destructive patterns of behaviour; rather than mindlessly reacting to them. Heightened awareness of these stimuli, and mindful exposure to them, should in principle help to disrupt the link that has been established between their occurrence and engaging in maladaptive behaviours. Furthermore, the values component of ACT could be especially important in leveraging, supporting, and augmenting change.

8.2.2 *Understanding and Treating Treatment Resistant Patients*

The current findings could also hold substantial and direct implications for understanding and treating patients who have been unresponsive to, or relapsed

following, previous standard care treatments. The following section discusses the care of treatment resistant patients and possible directions for future work.

8.2.2.1 *Understanding Treatment Resistance.* Although resistance to treatment is common, and the symptoms that predict it are well documented, little was known about the processes that might maintain it. Based on ACT theorising, it was predicted that excessive experiential avoidance would commonly maintain the dissimilar symptoms that treatment resistant patients tend to display. In support of this prediction, the samples who participated in studies 3 and 4 presented with high levels of experiential avoidance and, on the whole, showed successful long term gains after this process had been effectively targeted. Furthermore, findings also indicated that reductions in experiential avoidance during treatment were significantly predictive of outcomes above and beyond the effect of other plausible maintenance factors, such as the frequency of negative thoughts. Thus, although it is impossible to identify factors that predicted these samples' resistance to treatment in the past, it is clear that experiential avoidance was uniquely implicated in terms of future psychological functioning. Although these findings are preliminary and in need of replication, they certainly converge with existing research, which has found that experiential avoidance, avoidant coping, and thought suppression precipitate relapse (e.g., Moos & Moos, 2006; Salkovskis & Reynolds, 1994; Westruff, 2001, cited in Chawla & Ostafin, 2008). Likewise, they are also consistent with Ma and Teasdale's (2004) research which has shown that mindfulness helps to maintain remission in patients with major depression (another highly relapse prone group). These findings tentatively suggest that ACT could be a useful treatment for treatment resistant patients and that experiential avoidance could be important for understanding relapse and relapse prevention. Possible directions for future research are discussed below.

8.3.1.2 *ACT for Treatment Resistant Patients.* Although the current findings have supported the use of ACT for treatment resistant patients, they are nonetheless preliminary and in need of replication. To establish ACT as an empirically supported treatment for this group (Chambless & Hollon, 1999), it must be evaluated using research designed to address some of the limitations that are inherent in the pilot phase

of testing. In the current trials, these limitations mainly included the use of small samples, reliance on self-report measures, and the use of a TAU comparison to compare ACT and CBT.

The most obvious extension of this work is to conduct RCTs that recruit samples sufficiently large to detect the Group x Time interactions that were expected in study 4 (cell sizes adjusted for attrition). This would not only help to confirm/disconfirm the effectiveness of ACT for this group, but could also provide a more thorough assessment of the implied link between experiential avoidance and relapse (see section 8.3.1.3). Future RCTs could also be improved by adding a wait-list group as an additional control condition (i.e., ACT, CBT, WLC). This would allow for formal mediation analyses and the estimation of treatment effects relative to a no-treatment control. The use of behavioural and psychophysiological measures in addition to self-report measures could also be valuable, offering more detailed and objective information regarding treatment effects. Additionally, the collaboration of ACT and CBT specialists would help to ensure that both interventions were appropriately represented and that equally suitable process and outcome measures were selected. Independent ratings of treatment content could not only help to ensure treatment fidelity, but could also provide a means of objectively quantifying the key similarities and differences of these two interventions in practice.

Heterogeneous sampling is uncommon in well-controlled RCTs because variability in treatment response increases the probability of Type II error and makes it difficult to replicate findings. This sampling approach is essential, however, if the aim is to obtain a representative sample of treatment resistant patients. To balance these tensions, the inclusion/exclusion criteria could be refined so as to access a representative sample that has less within-group variability than the samples in studies 3 and 4. For example, the inclusion criteria could be refined to patients with *at least* 2 previous treatment episodes and who had finished their previous treatment within a specific time frame, such as within the last two years. These refinements could help to target a more treatment resistant cohort. Inclusion criteria could also be more prescriptive about the type of treatments the patient has previously received and their responsiveness to them. Such information could be obtained, for example, from the patients' clinical files and/or contact with previous clinicians. Refinements such as these would help to reduce

statistical within-group variability without impairing the recruitment of a representative sample of symptomatically heterogeneous patients.

The RCT has been recommended primarily because tightly controlled experimental trials are necessary for establishing empirical support for a novel treatment (Chambless & Hollon, 1999). Nevertheless, these trials could also show some sensitivity to issues of external validity. For example, it would be possible to compare ACT and CBT delivered by standard care therapists, rather than specialist clinical teams. In this design, standard care therapists with a range of experience and preferred treatment orientations could be randomly allocated to either ACT or CBT training. Treatment session recordings could allow one to ascertain the degree of fidelity to the respective interventions and also to assess how successfully these techniques were taken up. Furthermore, a design of this kind could also include an assessment of training effects and cost-effectiveness analysis. Thus, although neither treatment would be optimally delivered, this approach would provide a good approximation to real life clinical settings and allow for ACT and CBT to be compared along many important dimensions. One limitation of this type of approach, however, is that the dominance of CBT within current clinical training and practice could introduce non-specific treatment confounds. For example, even newly trained clinicians would have had previous experience delivering CBT but not ACT.

A final recommendation is that future work is designed to understand the factors that predict poor outcomes following ACT. Indeed, although the current findings showed promising changes on the group level, a minority of patients either stayed the same or worsened following treatment. Because this cluster of patients was so small, it was impossible to statistically identify factors that were predictive of poor outcomes. Nevertheless, informal observations during treatment suggested that patients with a strong attachment to the self as defined by the content of verbal behaviour (i.e., self-as-content) were less likely to show change (e.g., Elaine: “I get the feeling of being unsafe when people get the idea of who I am or what I am ... of not being good enough”). These participants also tended to struggle with identifying and committing to core values (e.g., Elaine: “I have these ideas that I am going to be very different with people... but when it comes down to it I’m like “no, I am the same old person, it’s not going to work””). This could have occurred because their continued attachment to self-as-content provided them with good ‘reasons’ why they could not pursue valued domains. Identifying moderators of treatment response such as these objectively, could

greatly enhance our understanding of when ACT is most likely to produce beneficial effects. Furthermore, knowledge of moderators could also guide the development of treatment protocols for this group and help clinicians to adapt protocols given certain baseline profiles. For example, if excessive attachment to self-as-content does predict poor outcomes in treatment resistant patients, greater clinical time could be allocated to this component. Similarly, it could be helpful to expose patients to the concept of values earlier in treatment so as to gradually develop their ability to make undefended contact with them.

8.2.2.3 *Understanding and Preventing Relapse.* The clinical trials in this thesis could also hold some clues regarding relapse prevention. Specifically, in keeping with existing research (e.g., Marlatt et al., 2004; Teasdale et al., 2000), findings have suggested that cultivating awareness and acceptance of symptoms could help to foster resilience in this relapse prone group.

How might ACT and mindfulness-based techniques achieve this? One possibility is that they disrupt the link between residual or re-occurring symptoms and subsequent relapse. For example, many ACT techniques encourage exposure to unwanted internal experiences in conjunction with inaction, defusion, and/or mindful observation. These learning episodes could help to weaken the motivation to engage in negatively reinforced operant behaviour. In turn, this could provide a window of opportunity for new patterns of responding, such as value consistent responding, to be established. New patterns of behaviour could initially occur only when prompted by the therapist ("Would you be willing to have these feelings if it brought you closer to your value of intimacy?"), but if ACT is successful, value orientated action should begin to occur independent of social prompts (i.e., after treatment has ended). Effectively using one's values to guide behaviour could lead to long term changes in psychological well-being by enabling a new repertoire to emerge that allows increasing access to positive reinforcement. Indeed, something of this kind could account for the continued gains that were found on some of the key outcome measures. Although this account is in need of further development and at present lacks empirical support, it provides a direction for further ACT research.

To explore these implications further, future research could aim to pin down some of the factors that mediate or moderate relapse prevention and continued gains. For example, a longitudinal questionnaire-based design could be used to monitor patients at regular intervals following treatment. This could allow for an analysis of whether the relationship between re-occurring symptoms at 'time 1' and a relapse episode at 'time 2' is mediated by changes in experiential avoidance and/or increased valued living. Alternatively, interviews (before and after treatment) could be used to establish whether ACT trained participants report qualitative change in the way they relate to unwanted internal experiences and whether this impacts on valued action. For example, interviews could be used to obtain detailed retrospective accounts of the patients' most recent responses to a real life stressor or a dysphoric mood. Content analysis could then be used to test for change in response to that event both within-subjects and/or relative to a control group. A further option could be to use a laboratory-based design to model the way in which patients respond to emotional challenges before and after treatment. For example, a mood or stress induction task could be use to induce temporary emotional arousal. Psychophysiological measures could then be used to test whether ACT uniquely affects the patient's level of arousal and/or rate of recovery (e.g., using the measurement of cortisol). A method such as this could provide an objective assessment of whether ACT can affect a change in biological responses to distress and/or whether it affects the ability to regulate that distress when it is experienced.

8.2.2.4 *Borderline Personality Disorder.* Although the present thesis did not focus on Borderline Personality Disorder (BPD) specifically, there are several reasons to allocate attention to this diagnostic group. Firstly, BPD has long been recognised as one of the most treatment resistant of the clinical diagnoses; with patients consuming disproportionate amounts of clinical resources (see Lieb et al., 2004). Secondly, patients with BPD typically are involved in a wide range of maladaptive behaviours, experience intense and unstable levels of affect, and engage in excessive levels of emotional and cognitive avoidance (e.g., see Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006; Linehan, 1993). Furthermore, a reliable link has also been established between BPD and childhood maltreatment (e.g., Herman, Perry & Kolk, 1989). Thus, the variables under investigation in studies 1 and 2 also have particular and direct relevance to this group.

ACT-based theorising could prove helpful both in conceptualising and treating BPD. In terms of conceptualisation, ACT suggests that many of the symptoms constituting a BPD diagnosis can be understood as chronic forms of experiential avoidance (e.g., Hayes et al., 1996). Dissociation, for example, which is triggered by intense negative affect, provides immediate escape from unwanted internal events. Similarly, the co-occurrence of maladaptive behaviours that characterise this group, such as co-morbid substance abuse and DSH, were found to relate to BPD risk factors (childhood trauma and negative affect intensity), in part, through high experiential avoidance (study 2). Although this research did not use a BPD sample, the findings are complemented by the work of Gratz et al. (2008). In a BPD sample, these authors found that experiential avoidance was a large, significant and unique predictor of BPD symptoms even when controlling for the effect of emotional vulnerability, impulsivity, and anxiety sensitivity. Moreover, having accounted for the effect of experiential avoidance, none of the other variables were significantly predictive of BPD symptoms (see also Cheavens et al., 2005). Thus, although work in this area is limited, preliminary findings suggest that experiential avoidance could be useful for understanding BPD.

In terms of treatment, there are some very clear conceptual similarities between ACT and DBT (Linehan, 1993), which is currently the most empirically validated treatment for this diagnosis. For example, both treatments adopt a learning perspective that underscores the function of BPD symptoms. Both also aim to treat 'core' and 'co-morbid' disorders and use acceptance-based techniques to this end. Furthermore, both are based on a flexible and principle-based model of change (Miller & Rathus, 2000). Although it is too early to tell whether ACT could be valuable for BPD patients as a stand-alone intervention, especially considering its emotionally evocative nature, the theoretical continuity between these two treatments makes their synthesis a possibility. One approach already developed has been to synthesise components from both these treatments into a time-limited and group-based format (e.g., Gratz & Gunderson, 2006). Although the evidence for this approach is limited, it has nonetheless shown promising effects; significantly impacting on a rage of BPD-specific (e.g., DSH) and non-specific (e.g., global psychiatric functioning) outcomes.

Another possibility, especially for more entrenched BPD individuals, could be to integrate components of ACT into later stages of the DBT programme. Linehan (1993) proposed four stages of treatment in DBT and of these, stage one has received most

empirical attention (Dimeff & Linehan, 2001). Stage one is effective for stabilising self-destructive behaviours; however, at the end of this stage patients are often described as being in a state of "quiet desperation" (Dimeff & Linehan, p. 2). That is, their behaviours are typically under control, but they continue to feel unhappy, incomplete, and unfulfilled. Stage 2 therefore aims to increase appropriate experiencing of emotions. This stage, which is described as exposure-based in approach, helps patients to experience emotions non-traumatically (Dimeff & Linehan). It is possible that ACT techniques could usefully contribute at this point. For example, ACT could build on the patients' mindfulness skills acquired in stage 1 of DBT, helping them to experience their emotions from a defused observer perspective. Similarly, ACT could use values to motivate and augment change, helping to bring the patient's life into contact with natural reinforcers. Furthermore, ACT's focus on identifying and defining the self, which is not part of the DBT programme, could be especially useful for this group. This is because identity disturbance, such as incoherence and inconsistency in the sense of self, is a key component of this disorder (Wilkinson-Ryan & Westen, 2000).

Synthesising ACT and DBT would involve addressing some tensions, however. For example, DBT advocates distraction and avoidance of certain experiences as a way of down regulating emotional arousal. This could be made harmonious with ACT if patients could be successfully taught to discriminate situations where experiential avoidance is more or less likely to be effective. Specifically, patients could learn to use avoidance strategies flexibly and mindfully, rather than rigidly and excessively, and only when this does not lead to value inconsistent patterns of action.

So far, the discussions in this chapter have focused on the most direct implications of the work in this thesis. The final section, however, is designed to address some more global considerations for ACT research in general.

8.2.3 *Future Developments in the Field of ACT Research*

8.2.3.1 *Future Outcome Trials.* Although ACT outcome research has obtained many promising findings over the last ten years, I have argued that much of this research remains in the pilot stage of investigation. In fact, the limitations of the clinical trials in this thesis are also relevant to many of the published ACT outcome trials. Although there is a broad appreciation that pilot trials are a necessary stage of treatment

evaluation (Ost, 2008; see section 3.3), future research should begin to push forward into the next stage. This would involve evaluating ACT in well-controlled and powered clinical trials, with the aim of obtaining an objective assessment of its true impact by subjecting it to the same level of scrutiny that CBT has received over the years. These investigations would greatly profit from: (1) comparing ACT to active comparison conditions, (2) testing for mediation, (3) using different measurement methods to assess process and outcomes (e.g., self-report plus behavioural measures), and (4) conducting at least a 1 year follow-up. I agree with Ost's (2008) suggestion that empirically comparing ACT and CBT, both in terms of process and outcome, could be particularly insightful. This is for three main reasons. Firstly, it could help to discriminate which approach is most appropriate and when. Secondly, it could help to discover how these treatments differ in real life practice. Thirdly, it would address the fundamental question of whether ACT offers anything above-and-beyond already widely available techniques. This question is especially important when considering the treatment of acute psychiatric disorders, for which CBT has a good evidence base (see section 1.2).

8.3.2 *Understanding Mechanisms of Change.* Current knowledge could also be enhanced by testing the relative contribution, and differential effects, of ACT's many techniques. Dismantling research is a common method for investigating these types of questions. For example, holding extraneous variables constant, do different combinations of techniques (e.g., defusion, acceptance, and valued living versus acceptance and valued living) result in different outcomes? Similarly, does the utility of certain combinations differ according to group chronicity and/or the order of delivery? Although the dismantling approach is valuable, it has some limitations. For example, the full potential of some ACT techniques, such as self-as-context, may rest on the successful reception of other techniques, such as acceptance. Similarly, because ACT techniques affect a change in more than one process, it could prove difficult to divide them up unambiguously.

Another means of addressing similar questions, therefore, could be to monitor process and symptom changes regularly during treatment (e.g., using process measures). This approach could be used to assess how and when processes are affected, allowing a detailed assessment of how those changes correspond with symptom changes. Sample sizes permitting, structural equation models could be particularly useful for analysing

this type of data, allowing one to test whether each process has unique, interactive, or moderated effects on outcome. This could, for example, be used to assess whether the successful development of self-as-context facilitates valued living, or whether acceptance and defusion differentially affect outcome. Similarly, it could help to clarify whether defusion, acceptance, mindfulness, and self-as-context together constitute a higher order factor of 'undermining verbal governance' techniques (see Figure 2.2). These more fine-grained analyses could greatly enhance our understanding of precisely how ACT affects change. However, a current barrier to conducting analyses such as these is the availability of valid ACT process measures.

8.3.3 *Improving Process Measures.* To date, a noticeable area of weakness within the ACT literature is that measurement tools are simply not available and/or mature enough to assess its processes of change. For example, although ACT is described in terms of 6 processes, the AAQ is the only validated measurement tool currently available. Recently, even this measure has become the topic of debate, with authors retrospectively reconsidering whether it is a measure of experiential avoidance alone, experiential avoidance and psychological flexibility, or just psychological flexibility (see section 2.1; Hayes et al., 2006). An examination of the AAQ's content suggests that this measure is most concerned with (a) negative appraisal of internal events, (b) attempts to control and eliminate those events, and (c) inhibited action as a result of both (a) and (b). This is consistent with the early definitions of experiential avoidance (e.g., Hayes et al., 1996), but includes items that could arguably be described as measuring cognitive fusion (e.g., "When I evaluate something negatively, I usually recognize that this is just a reaction, not an objective fact") and the capacity for valued living action (e.g., "When I feel depressed or anxious, I am unable to take care of my responsibilities"), thus obscuring the boundaries between these processes. It is not hard to discern why this has occurred; ACT theory suggests that experiential avoidance is the result of excessive fusion and the antecedent of unvalued living.

A further complication with using the AAQ to measure experiential avoidance is that, by virtue of being a questionnaire, it measures this construct in a trait-based manner. As acknowledged by the authors (Hayes, Stroshal, et al., 2004, p. 572), this is not entirely harmonious with the idea that experiential avoidance is a contextually determined process. That is, experiential avoidance is understood to be a process that

occurs when fusion with private experiences is high and the motivation to escape those experiences predominate in the repertoire. Although this will occur more frequently for some individuals than others, from a theoretical perspective, experiential avoidance is regarded as a situational action and not an underlying trait. Thus, although in keeping with most of the work in this field, the reliance of this thesis on the AAQ to measure experiential avoidance is not without its problems. Developing process measure that are more sensitive to this contextual process is challenging, but some suggestions seem worthy of consideration.

In the laboratory, measures that have been developed to monitor physical engagement/disengagement from aversive experiences, such as exposure to emotionally evocative stimuli (e.g., International Affective Picture System (IAPS, Lang, Bradley & Cuthbert, 1999), could be used as pre and post-treatment measures of experiential avoidance. For example, eye tracking apparatus could be used to monitor the tendency to approach or avoid aversive visual stimuli (e.g., IAPS). Alternatively, an operant procedure could be used to measure the delay before participants terminate exposure to those stimuli (see Cochrane et al., 2006). The 'approach-avoidance' task is another possibility for measuring experiential avoidance. In this task, participants are presented with stimuli (e.g., words or pictures) of varied valance and are required to 'approach' or 'avoid' them by moving a joystick. Because instructions are either congruent or incongruent with valance (e.g., approach negative, avoid positive), delay latencies for cursor movement can be used as an index of pre-conscious approach or avoid tendencies (e.g., see Lange, Keijsers, Becker & Rinck, 2008). A strength of behavioural approaches of this kind is that they can be individualised by selecting stimuli that are salient to the individual participants. For example, the stimuli for a spider phobic could include pictures of spiders, whereas those for a patient with social phobia would include pictures of feared social events (e.g., Lange et al., 2008). Behavioural tasks such as these would help to recapture the essence of contextually determined process variables.

8.3.4 *Testing Theoretical Links with RGB and RFT.* A final recommendation is that future research begins to test the link between ACT techniques and theoretical accounts of those techniques. In chapter 2, I argued that the link between ACT and theoretical accounts of RFT and RGB is at present, tentative. The existing evidence for this link is weak and the conceptual associations often seem to be more analogous than direct. A

better understanding of these links could help to refine and develop ACT-based interventions. Below are two suggestions for addressing these links.

Firstly, the contingency sensitivity paradigm (see section 2.1) could be used to test whether ACT increases sensitivity to subtle changes in the environment. Early indications from unpublished work by F. Bond and colleagues (personal communication, 19th August 2006) certainly support the hypothesised relationship between experiential avoidance and contingency sensitivity. These authors reported that the AAQ was significantly predictive of the ability to detect unsignalled changes in reinforcement contingencies in a simple operant task. Individuals high in experiential avoidance were less sensitive to such changes than less avoidant counterparts. Extending this work to clinical samples, perhaps by using a behavioural pre-post measure, could help to elucidate the relationship between ACT and verbal governance.

A second approach, which arguably has greater ecological validity, could be to conduct logical functional analysis during treatment (LFA; see Ghaderi, 2007). LFA is an extension of functional analysis that uses a logical structure to identify the variables that maintain a disorder. Specifically, this approach guides the clinician to identify inadequate verbal and non-verbal stimulus control. An example of inadequate antecedent control, for example, is excessive or improper regulation of behaviour by verbal rules (see Ghaderi, 2007). Ghaderi (2006) has used this approach in patients with eating disorders, and identified excessive RGB as a defining characteristic of those patients who failed to improve following CBT. Extending this line of work could thus help to explain the link between RGB and treatment resistance more fully. One possibility could be to use a multiple baseline design across participants to establish whether excessive RGB diminishes when CBT is replaced with ACT.

In summary, this section has aimed to provide some suggestions for extending the work presented in this thesis, discussing the application of ACT to treatment resistant patients specifically and considering future ACT research more globally. The present research has provided some of the necessary foundations for investigating ACT for treatment resistant patients and strongly justifies the continuation of work in this area.

8.4 Concluding Comments

This thesis aimed to test the theoretical and clinical applicability of ACT for treatment resistant patients. Although its findings are tentative, and the story is still unfolding, ACT offers a promising and acceptable method of treatment for this group. Furthermore, it appears to have effected change in theory-consistent ways. To conclude, I would like to reiterate a few main points. Firstly, throughout the history of clinical research, the correspondence between theoretically-orientated and clinically-orientated research endeavours has been weaker than originally claimed. It remains important to narrow this gap. Treatments should evolve concurrently with theoretical models *and vice versa* so that each can inform the other. Secondly, despite their different theoretical allegiances, several modern psychological treatments appear to have many qualities in common. Most notable is the use of process-driven techniques to alter the way in which patients interact with private experiences, rather than attempting to change those experiences directly. These techniques appear to have specific benefits for the treatment of treatment resistant patients. Openness to collaboration between researchers with different theoretical orientations could prove pivotal to the progression of clinical research.

APPENDIX A

Unpublished Materials of Study I and II

Demographics Questionnaire

Please indicate answers in the spaces provided

1. Are you male or female? _____
2. How old are you? _____
3. What country were you born in? _____
4. What country do you live in? _____
5. What is your current occupation? _____
6. If you are a student, what subject have you been studying? _____
7. If you are a student, how many years have you been studying? _____
8. Have you ever sought treatment for a psychological problem (e.g., depression, anxiety, bereavement)? _____
9. If yes to qu. 8, did you take any medication for this problem?* _____
10. If yes to qu. 8, did you receive any psychological treatment for this problem (e.g., 'counselling', 'psychotherapy', 'cognitive behaviour therapy')? _____
11. If you have received psychological treatment, how many types of therapy have you tried? _____
12. Approximately how many sessions did you attend for the first type of therapy? _____
13. Approximately how many sessions did you attend for the second type of therapy? _____
14. Approximately how many sessions did you attend for the third type of therapy? _____

* Item included in study 2 only.

Maladaptive behaviours Questionnaire

This questionnaire is designed to ask you about a range of behaviours that you may, or may not, engage in. It includes 49 statements and you are required to rate the extent to which each statement characterises you, using the scale below

1 - - - - - - - - -	2 - - - - - - - - -	3 - - - - - - - - -	4 - - - - - - - - -	5 - - - - - - - - -	6
Very unlike me	Quite unlike me	A little unlike me	A little like me	Quite like me	Very Like me

For example, if you read a statement and think "it's very unlike me to do X" you would write a "1" next to the statement. If you think "that's only very slightly like me" write '4', or if you think "it's very like me to do that", write '6'.

Before completing the questionnaire, please take note of the following points:

Where questions refer to internet use, this means non-work related use such as chat rooms, surfing the net etc. Where questions refer to sexual behaviours, this includes both foreplay and all forms of sexual intercourse. Where questions refer to drugs, this means the use of illegal drugs. This would include, for example, Cannabis, Cocaine, Ecstasy etc. Where questions refer to smoking, this means tobacco.

Please read each statement carefully and answer as honestly as possible. **All answers are anonymous. Please do not leave any answers blank**.

It's like me ....

1	to eat anything I want anytime I want to	1 2 3 4 5 6
2	to avoid cigarette smoke if I am unwell	1 2 3 4 5 6
3*	to say no to drugs, including cannabis	1 2 3 4 5 6
4*	to be pre-occupied by thoughts about smoking when smoking is prohibited	1 2 3 4 5 6
5*	to sometimes consume more than 6 alcoholic drinks in one evening	1 2 3 4 5 6
6	to eat large amounts of food in secret	1 2 3 4 5 6
7	to feel contented if I am prevented from surfing the net/playing video games for a prolonged amount of time	
8*	to ignore dietary details (e.g., calorie content) when choosing something to eat	1 2 3 4 5 6
9*	to exercise even when I am feeling tired and/or unwell	1 2 3 4 5 6
10	to physically threaten or hurt someone	1 2 3 4 5 6
11*	to sometimes intentionally prevent scars or wounds from healing	1 2 3 4 5 6
12	to plan significant purchases (e.g., clothes, electronic goods) in advance	1 2 3 4 5 6
13*	to smoke tobacco	1 2 3 4 5 6

14*	to surf the net/play computer games before doing something else that needs doing	1 2 3 4 5 6
15*	to generally have no interest in taking drugs, including cannabis	1 2 3 4 5 6
16*	to sometimes engage in sexual activities with someone I have only just met.	1 2 3 4 5 6
17*	to find that my work performance or productivity suffers because of my internet/video game use.	1 2 3 4 5 6
18*	to never resort to violence.	1 2 3 4 5 6
19*	to sometimes actively seek out drugs for personal use, including cannabis.	1 2 3 4 5 6
20*	to feel irritation/frustration if I am in a non-smoking environment.	1 2 3 4 5 6
21*	to sometimes scratch or bite myself to the point of scarring or bleeding.	1 2 3 4 5 6
22*	to sometimes feel pre-occupied with the internet/computer games.	1 2 3 4 5 6
23*	to skip doing exercise for no good reason.	1 2 3 4 5 6
24*	to drink a lot more alcohol than I initially intended.	1 2 3 4 5 6
25*	to have a long list of things that I dare not eat.	1 2 3 4 5 6
26*	to feel excitement and/or tension in anticipation of getting drunk.	1 2 3 4 5 6
27*	to be content if I am prevented from exercising for a week.	1 2 3 4 5 6
28*	to always stop eating when I feel full.	1 2 3 4 5 6
29*	to prefer being in places where smoking is prohibited.	1 2 3 4 5 6
30	to use a condom throughout sexual intercourse with a new partner	1 2 3 4 5 6
31*	to control my temper.	1 2 3 4 5 6
32*	to deliberately take small helpings as a means of controlling my weight.	1 2 3 4 5 6
33*	to exercise more than three times a week.	1 2 3 4 5 6
34	to sometimes conceal the full extent of my purchases to friends and family	1 2 3 4 5 6
35*	to sometimes eat to the point of physical discomfort.	1 2 3 4 5 6
36*	to sometimes feel tension and/or excitement in anticipation of doing exercise.	1 2 3 4 5 6
37*	to sometimes cause myself direct bodily harm by, for example, cutting or burning myself.	1 2 3 4 5 6
38*	to only eat when I am hungry.	1 2 3 4 5 6
39*	to unsuccessfully try to cut back my use of the internet/computer games	1 2 3 4 5 6

40	to always take steps to protect myself against pregnancy and sexually transmitted diseases	1 2 3 4 5 6
41*	to be excited by the opportunity of taking drugs, including cannabis	1 2 3 4 5 6
42	to prefer evenings that do not involve drinking alcohol	1 2 3 4 5 6
43*	to sometimes get so angry that I beak something	1 2 3 4 5 6
44	to sometimes use laxatives, diuretics or abuse diet pills to control my weight	1 2 3 4 5 6
45	to avoid objects or activities that could cause me physical pain	1 2 3 4 5 6
46	to always pass up opportunities for causal or illicit sex	1 2 3 4 5 6
47*	to sometimes have more than one sexual partner.	1 2 3 4 5 6
48*	to sometimes buy things for the sake of it, rather than because I actually need them	1 2 3 4 5 6
49*	to sometimes engage in sexual actives with someone when really I shouldn't	1 2 3 4 5 6
50*	to sometimes feel a strong impulse to buy things that I don't really need	1 2 3 4 5 6
51*	to easily limit my use of the internet or video games	1 2 3 4 5 6
52*	to feel the urge to have a cigarette.	1 2 3 4 5 6
53*	to sometimes feel that I need to take drugs (this includes cannabis)	1 2 3 4 5 6
54*	to go out with friends who are drinking, but opt to stay sober	1 2 3 4 5 6
55	to easily get into arguments when someone disagrees with me	1 2 3 4 5 6
56	to think carefully before I buy something	1 2 3 4 5 6
57	to avoid objects or activities that could harm my body	1 2 3 4 5 6
58*	to sometimes think that I might have a drugs problem (this includes cannabis).	1 2 3 4 5 6
59*	to avoid eating when I am hungry	1 2 3 4 5 6
60*	to find it difficult to stop eating after certain foods	1 2 3 4 5 6
61*	to be aggressive when sufficiently provoked	1 2 3 4 5 6
62*	to feel the urge to intentionally harm myself	1 2 3 4 5 6
63	to experience guilt if I do not exercise	1 2 3 4 5 6
64*	to sometimes feel that I need an alcoholic drink	1 2 3 4 5 6
65*	to sometimes claim I have already eaten when this is not true	1 2 3 4 5 6
66*	to sometimes experience a powerful urge to spend money	1 2 3 4 5 6

*Items that *were* in the final version (see Chapter 4 for exclusion process).

APPENDIX B

Description of Study III Participants

Information about patients participating in Study 3 is described below. Please note that some aspects of this information have been altered (e.g., age, gender) so as to ensure patient confidentiality.

Participant 1 was a 24 year old male presenting with sever and recurrent depression and clinical levels of anxiety. He also reported clinical levels of passive-aggressive personality traits. At intake, he was signed off from work due to his psychological difficulties. He had received the following psychological treatment before attending the ACT groups: 3 months of individual psychotherapy, and a brief period (4 sessions) of individual CBT-based counselling.

Participant 2 was a 37 year old single female presenting with sever and recurrent depression, panic attacks and clinical levels of anxiety. At intake, she described experiencing several panic attacks a day and described these as preventing her from most 'normal' daily activities such as taking the children out. Her presenting problem at intake was incapacitating recurrent anxiety and panic attacks. Previous therapeutic care included: 2 months of individual counselling, 3 months individual psychologist, 4 months cognitive therapy and 2 anxiety groups.

Participant 3 was a 46 year old divorced woman with two children. At intake she presented with significant distress related to the break-down of an intimate relationship. She had persistent intrusive and obsessive thoughts and engaged in repetitive reassurance behaviours such as phoning the GP, family and friends up to 30 times a day. She also voiced persistent suicidal thoughts, had experienced recurrent episodes of depression and had clinical levels of anxiety. Although she had no history of BPD, she was exhibiting several BPD symptoms in her most current relationship. Previous therapeutic care included: two episodes, equating to 8 months of individual psychotherapy (CBT in orientation), and 3 ½ months of CBT.

Participant 4 was a 41 year old female. She was also currently signed off work due to her mental health concerns. At intake she presented with Avoidant Personality Disorder, compulsive personality traits, moderate to severe depression and clinical levels of anxiety. Previous therapeutic care: two episodes of individual counselling.

Participant 5 was a 43 year old female experiencing recurrent depression and significant problems with perfectionism. She also met lifetime diagnostic criteria for Obsessive Compulsive Personality Disorder, Depressive Personality Disorder and Borderline Personality Disorder (symptomatic for the first two at intake). In addition to her psychological difficulties, she was also presented with sever migraines, IBS and ME. She further explained that her father had committed suicide when she was 20 years old, which contributed to several severe episodes of Anorexia Nervosa, also presented by her siblings. This individual first presented to the services in 1991, and had tried four different types of individual psychology (including CBT) prior to attending the ACT group (40 sessions in total).

Participant 6 was a 58 year old female presenting with an intense fear of death. Psychometric measures indicated that she met criteria for Somatoform disorder, sever and recurrent depression and clinical levels of anxiety and phobic anxiety. Previous therapeutic care included; individual psychiatrist, individual counsellor and psychotherapy (19 individual sessions).

Participant 7 was a 34 year old woman presenting to the psychological services with recurrent pain attacks and agoraphobia that began to occur after a road traffic accident. Additionally, during her teenage years, this patient was diagnosed with anorexia nervosa; however, she was not symptomatic at intake. Prior to attending the group, she had tried several interventions including; group therapy, individual psychotherapy, hypnosis and meditation. At the time of assessment, she was experiencing several panic attacks a day, which precipitated symptoms of agoraphobia.

Participant 8 was a 38 year old female who had been sexually abused by her grandfather and brother in childhood and raped in late adolescents. Six years prior to the current group she was diagnosed with PTSD, recurrent depression, and met the SCID-II diagnostic criteria for Borderline Personality Disorder, Obsessive Compulsive Personality disorder and Avoidant Personality Disorder. Psychiatric records indicated a history of suicidal behaviour dating back to a first suicide attempt at 12 years old. She also has a history of DSH, which she had been abstinent from for 1 year 9 months prior to attending the group. She also had a history of substance abuse. Previous therapeutic care included; individual consultant psychotherapist for psychodynamic therapy (2 years); DBT (2 years); relaxation group (6 months); and a CBT-based Childhood Sexual Abuse survivors group (3 months). These interventions spanned back-to-back for 5 years (2001-2006). Assessment using the SCID-II indicated that at intake, this patient was presenting with Avoidant PD, Obsessive Compulsive PD, sub-threshold BPD (4/5 criteria), sub-threshold depressive PD (4/5 criteria), and clinical levels of anxiety and severe depression.

Participant 9 was a 46 year old female suffering from recurrent depression, high levels of anxiety and avoidant personality disorder. She described having formed very few friendships during her life because of intense feelings of inadequacy. During her childhood, she described being physically and verbally abused by her stepfather. For the 10 years prior to attending the ACT group, she had tried four different therapies including individual and group therapy (CBT in orientation). This treatment totalled over 140 treatment sessions.

Participant 10 (Elaine) was a 36 year old female with a long history of psychiatric care for BPD, which dated back over 10 years. During this time, this participant had received many structured intervention, including; Cognitive Analytic Therapy, CBT, the full DBT treatment program, Gestalt therapy and Psychodynamic therapy. This individual had a history of chronic self-harm (abstinent for 3 years prior to attending the group), alcohol and drug abuse (abstinent for at least 6 months prior to the group), impulsive spending that resulted in high levels of debt. She also had several inpatient admissions for suicide attempts. She also experienced high levels of depression, met diagnostic criteria for co-morbid personality disorder, and experienced recurrent episodes of dissociation when distressed.

Appendix C
Valued Directions Questionnaire

Below are areas of life that some people value. We are concerned with your quality of life in each of these areas. One aspect of quality of life involves the importance that you put on different areas of living. First rate the importance of each area by circling a number on the scale of 0-2. Not everyone will value all of these areas, or valued all of these areas the same. Rate each according *to your own sense of importance.* If you rate an area as unimportant (0), move right on to rate the next area. If you rated an area as moderately or very important (1, 2) make a rating of how satisfied you are with the quality and depth of your experience in this area of life. Then rate how often you have done something to move you forward in this area in the last week.

Questions and rating scale for each domain

How important is this area to you?		
0 = not at all	1 = moderately	2 = very important
Overall, how satisfied are you with the quality and depth of your experience in this area of life?		
0 = not at all	1 = moderately	2 = very satisfied
How frequently have you done something to move you forward in this area during the last week?		
0 = no action	1 = once or twice	2 = three or four times
3 = more than four times.		

1) **Family (other than marriage of parenting):** *how do you want to interact with your family members? What type of sister or brother do you want to be? What type of son or daughter do you want to be?*

2) **Intimate Relationship (e.g., marriage, couples):** *What is your ideal relationship like? What type of relationship would you like to have? What kind of partner would you want to be in an intimate relationship with? How would you treat your partner*

3) **Parenting:** *What type of parent do you want to be? How do you want to interact with your children?*

4) **Friends/social Life:** *What type of friend do you want to be? What does it mean to be a good friend? How would you behave towards your best friend? Why is your friendship important to you?*

5) **Work/career:** *What do you value about your work? Financial security? Intellectual challenge? Independence? Prestige? Getting to interact with others? Helping people?*

6) **Education/training:** *Why is learning important to you? Are there any skills you'd like to learn?*

7) **Recreation/fun:** *What type of activities do you enjoy? What type of activities would you really like to engage in? Why do your enjoy them?*

8) **Spirituality:** *This domain is about faith and spirituality rather than a specific religion. Why is faith important to you? If this is important to your life, what is it that makes it so important?*

9) **Citizenship/community life:** *What can you do to make the world a brighter place? Are community activities (e.g., volunteering, voting, recycling) important to you? Why?*

10) **Health/ physical self-care:** *What issues related to health and physical well-being do you care about (e.g., sleep, diet, exercise)? Why and how do you take care of yourself?*

Cognitive Fusion

Please answer the following questions, basing your answers on the last two weeks.

1) On average, how often have you experienced unwanted or instructive thoughts/feelings?

1	2	3	4	5	6	7
never	less than once a week	about once a week	several times a week	daily	more than once a week	almost constant

2) On a scale of 1-10, how much do you believe these unwanted/intrusive thoughts and feelings are real and meaningful?

1	2	3	4	5	6	7	8	9	10
Not at all real and/or meaningful									very real and/or meaningful

Appendix D

Study III Patient Information Sheet and Information for General Practitioners

Patient information sheet

You are being invited to take part in a research study. Before you decide on whether you would like to participate, it is important for you to understand why the research is being done and what it will involve. Please take time to read the following information carefully, and to talk to others about the study if you so wish.

- Part 1 tells you the purpose of this study and what will happen to you if you take part.
- Part 2 gives you more detailed information about the conduct of the study.

If there is anything that is not clear, or if you would like more information, please do not hesitate to contact Sue Clarke on (contact details) or Jess Kingston (a member of the research team) on (contact details).

We ask that you inform Sue Clarke or Jess Kingston of your decision within one week.

Part 1

What is the Purpose of this Study?
The purpose of this study is to provide patients with a 16 week, group-based delivery of Acceptance and Commitment Therapy (ACT). The aim of the research component is to evaluate how effective this therapy is. We will do this by comparing pre-intervention data to post-intervention data. Below you will find some information about ACT.

ACT is a therapy currently being used in America for patients with a range of mental health problems. ACT proposes that many mental health problems arise from, and can be made worse by, the avoidance of thoughts, feelings, and bodily sensations. The main focus of ACT is to increase one's willingness and acceptance for distressing thoughts and feelings, and to help patients live in a way that is consistent with their life values. Previous research has found ACT to be an effective intervention for a range of psychological disorders (e.g., depression, anxiety, post traumatic stress disorder, anorexia).

Why Have I Been Chosen?
We are contacting patients who are currently on the Dorset HealthCare NHS Trust waiting list for general adult mental health, and who have had at least one form of therapy in the past.

Do I Have to Take Part?
No. It is completely up to you whether you decide to take part or not. If you do decide to take part, you will be asked to sign a consent form. ***You are still free to withdraw at any time and without giving a reason. A decision to withdraw, or a decision not to take part, will not affect the standard of care you receive.***

What will happen to me if I take part; what will I have to do?

Assessment Phase
We are initially inviting you to an assessment at the Intensive Psychological Therapies Service (IPTS) in Poole. At this assessment session, the therapist will describe Acceptance and Commitment Therapy to you, and assess whether you meet our inclusion criteria for this trial. **If you do not meet the inclusion criteria, you will remain on the general adult mental health waiting list.** If you do meet the inclusion criteria, you will be given a week to decide whether you would like to participate. If you would like to participate, you will be asked to sign a consent form.

Research Phase 1:
Having provided consent, you will be sent a questionnaire pack to fill out in your own time and return to the clinic before starting therapy. This pack should take you about 90 minutes to complete. A member of the research team will be available if you have any difficulties completing these forms (either over the phone or in person if necessary). You will also be booked in for a 90 minute session at the Intensive Psychological Therapies Service (IPTS) in Poole. This will be arranged for a time that is convenient for you. During this session you will be interviewed by a member of the research team about your current psychological difficulties and about the therapy that you have had in the past.

Intervention:
You will then be invited to attend 16 weeks of group therapy. Sessions will be weekly, and will last for approximately 2 ½ hours (with a 20 minute break) per week. There will be a total of 10 patients attending each group session and two clinicians. These sessions will be held at (time) at the IPTS. As with many forms of therapy, the clinicians will often set tasks for you to complete between therapy sessions.

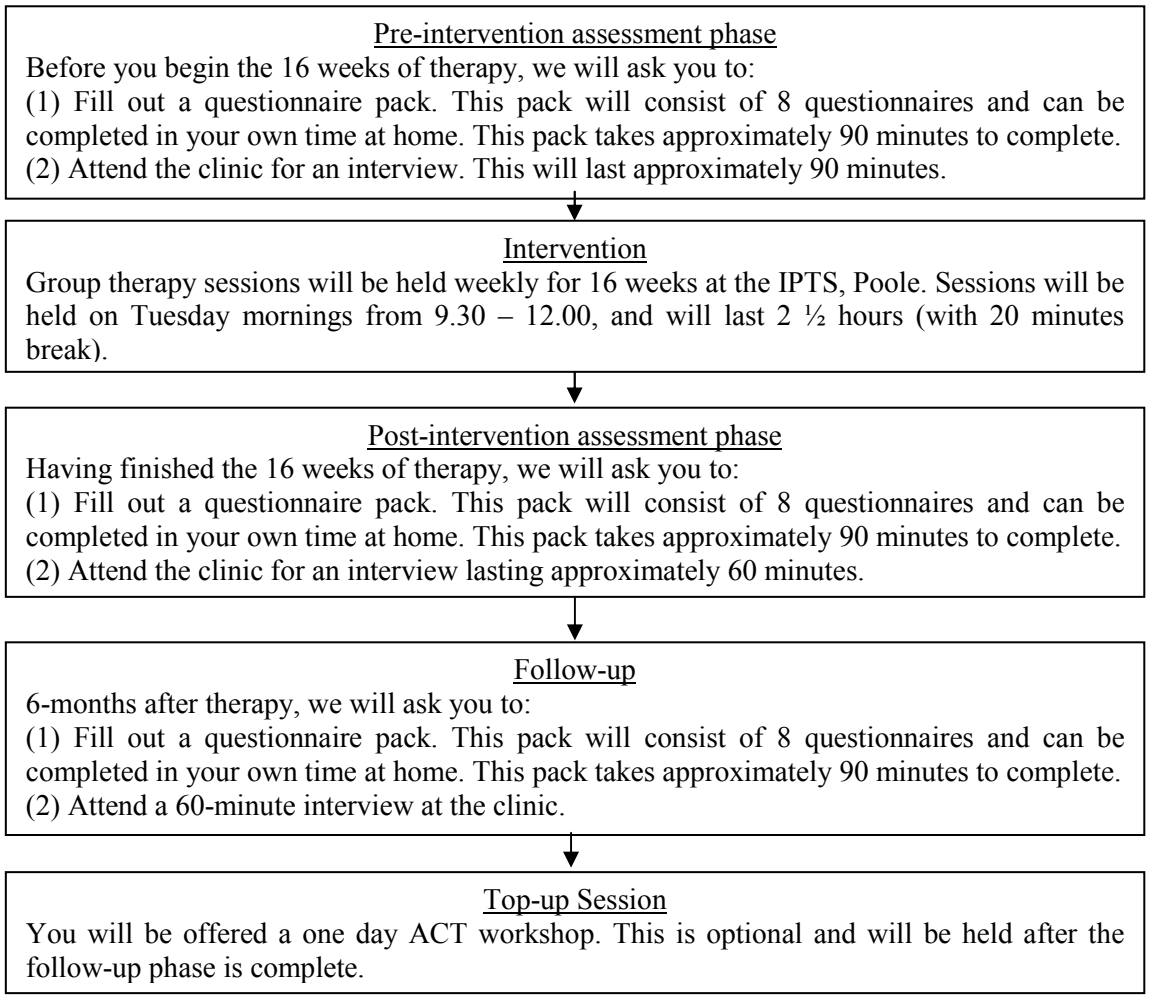
We hope to audio tape all therapy sessions and interviews. Therapy sessions will be taped to ensure that the therapy is delivered as anticipated, and to help the therapists develop their skills. Only members of the research team will have access to these tapes. Tapes will be securely stored in a locked cabinet.

Research Phase 2:
After these 16 weeks of therapy, you will be asked to complete a second set of questionnaires (in your own time) and attend another interview at the IPTS. This interview will ask about your experiences of the therapy. Six months after therapy we will ask you to attend a final interview and complete a final set of questionnaires.

All interviews and questionnaire components are part of the research, allowing us to evaluate how effective the group was.

Top Up Session
You will be invited to a 1 day “top-up” therapy session after the 6 month assessment.

All components of the trial are displayed in the flowchart overleaf.



During this trial, you will be able to continue taking any medication that you are currently taking. We will ask that you refrain from attending any other form of psychotherapy for 6 months after therapy. This is routine practice and is referred to as a consolidation phase. This allows us to assess how effective the therapy is 6 months after the intervention. Once we have collected your follow up data you will be able to opt in for standard care if you so choose.

We ask that you attend all scheduled visits and therapy sessions, and that you complete all the questionnaires. If you fail to attend four therapy sessions in a row, you will no longer be able to participate in the trial. The reason for this is that it helps you from unintentionally drifting out of therapy, and it facilitates group moral. If you stop coming to sessions without informing the clinic, the clinician will contact you. If you have changed your mind and decided that you would no longer like to attend the sessions, the clinician can help you arrange alternative care.

Unfortunately we will be unable to provide you with any compensation for your travel expenses to the clinic.

What Stage of Development is ACT at?
The first trial delivering ACT was in 1986 to a group of patients with depression. Both this trial and many subsequent trials have indicated that ACT is a successful therapy for a range of psychological difficulties, such as Post Traumatic Stress Disorder, Generalized Anxiety Disorder and addictions. This is the first trial to bring the intervention over to England, and to deliver the therapy to a group of individuals who have a range of different (as opposed to the same) psychological difficulties. The therapy sessions will be based on the ACT self-help workbook; *Get Out of Your Mind and into your Life* (Hayes & Smith, 2005).

What are the Alternatives for Treatment?
The alternative treatment on offer is standard care. This is *usually* individual psychological therapy or counselling. The benefits of receiving standard care as opposed to participating in this trial are that standard care will (1) have more flexibility in when the therapy ends and (2) will be more likely to offer you individual therapy (although this is not always the case). The benefits of participating in this therapy as opposed to standard care are that (1) you will receive therapy sooner, (2) you will be receiving an intervention that has a history of good outcomes across many types of mental health problems, and (3) you are likely to acquire positive outcomes from participating in a group-based intervention. If you decide to participate in this trial, you will be able to opt in for standard care after the 6 month follow-up assessment if you so choose.

What are the Possible Risks or Disadvantages of Taking Part?
Like any psychotherapeutic intervention, you may feel emotional distress during the course of therapy. Your well-being will be monitored by clinicians during every session, and they will ensure that no patient leaves the session significantly distressed. Patients who struggle with problems that cannot be addressed adequately in the group setting will be provided with an individual therapy session with one of the clinicians. Both clinicians are ACT trained, and have extensive experience in delivering therapy. One of the founders of this therapy will also be providing the clinicians with consultation throughout the trial.

A possible disadvantage of participating in this trial is the inconvenience of the research components. These have been kept to a minimum and will be conducted in a way that is as convenient to you as possible. These aspects are necessary for us to see how effective the therapy has been and we will provide you with a letter detailing all research findings.

What are the Potential Benefits of Taking Part?
Based on the evaluation of previous clinical trials, ACT has potential benefits for a wide range of psychological disorders. For example, ACT reduced rehospitalisation of patients suffering from psychotic symptoms by up to 50%; reduced rates of drug use in opiate addicts significantly more than methadone treatment; and decreased feelings of anxiety in socially anxious individuals. There is also evidence to suggest that ACT is effective for treating depression, post-traumatic stress disorder and panic disorder. Although we cannot promise health improvements, we anticipate that this treatment will decrease psychological distress, increase quality of life and teach you skills to help during everyday living. The group delivery is also aimed at helping to develop social skills and provide validation (e.g., “I am not the only one who feels like this”).

The trial will also help us to learn more about the effective delivery of this therapy. The interviews after the intervention are designed so that you can tell us exactly what was and was not good about the therapy. In this way, you will be helping us learn more about the intervention and better ways to deliver it in the future.

What Happens when the Research Study Stops?
You will be provided with a mindfulness meditation CD to help you practice skills learnt in the group. You will also be offered a one day ACT "top-up" session approximately 6-7 months after therapy. After the 6 month follow-up assessment phase, you are free to opt in for standard care if you so choose.

What if there is a Problem?
Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. The detailed information on this is given in Part 2.

Confidentiality –Who will know that I am taking part in this study?
All the information about your participation in this study will be kept confidential. The details are included in Part 2.

For Further Information
If you would like any further information about ACT or the trial, please do not hesitate to contact Professor Sue Clarke (contact details), or Miss Jess Kingston (contact details).

This completes Part 1 of the Information Sheet.

If the information in Part 1 has interested you and you are considering participation, please continue to read the additional information in Part 2 before making any decision

Part 2

What if relevant new information becomes available?
Members of Sue Clarke's research team are currently monitoring and will continue to monitor the ACT internet server and discussion forum. If any evidence comes to light that there are any adverse effects to ACT, your clinician will inform you of these details and ask you whether you would like to continue with the trial. If you decide to continue in the study you will be asked to sign an updated consent form. If you decide not to continue with the trial your continuing care will be arranged. It is also possible that, on receiving new information, the clinician feels that it is in your best interests to withdraw from the study. In the unlikely event that this happens, she will explain the reasons and facilitate the continuation of your care. If the study is stopped for any other reason, you will be told why and your continuing care will be arranged.

What will happen if I don't want to carry on with the study?
You are free to withdraw from treatment at any stage. If you withdraw, we will need to use the data collected up to your point of withdrawal, but this will only be available to

members of the research team and will not be stored with information that can identify you. If you withdraw, we will try and contact you. This is only for us to check whether you have experienced any adverse effects of the therapy. If this is the case, we will arrange individual care for you.

What if something goes wrong?

It is unlikely that this therapy will cause you any harm. Trained clinicians will be available at every stage of the intervention and assessment. If any research comes to light which suggests ACT may have negative consequences, patients will be informed immediately. We would like to reassure you that to date, patients have not suffered any adverse effects from this intervention that we are aware of.

If you have a concern about any aspect of this study, you should phone Sue Clarke (contact details). If you remain unhappy, you have the right to complain to the NHS about any aspects of the way you have been approached or treated during the course of this clinical trial. In the event that something does go wrong and you are harmed during the research study there are no special compensation arrangements. If you are harmed and this is due to someone's negligence then you may have grounds for a legal action for compensation against Dorset NHS, but you may have to pay your legal costs.

Will my taking part in this study be kept confidential?

All information which is collected about you during the course of the research will be kept strictly confidential and will only be looked at by members of the research team. This includes both clinicians, members of the Dorset NHS HealthCare team, and two co-investigators at the University of Southampton. Data will be assessed at both of these sites. To ensure that confidentiality is maintained, your data will not be stored with any personally identifying information, and will be stored in securely locked cabinets and password protected computers. You will be asked at the beginning of the trial to choose a participant number. This will be stored with the data. Your personal details (e.g., name and address) will not be made available to anyone other than members of the research team and will be communicated between members of the research team in person.

Please be aware that if a member of the team is given reason to believe that you may harm yourself or others, confidentiality may be breached.

Healthcare professional involvement

If you decide to participate in this trial, we will inform any healthcare professionals (e.g., GP's) currently involved in you care, and provide them with some brief information on the therapy. If the clinician feels that it is necessary to share any information acquired during the trial with members of your healthcare team, you will be asked first. If this is of concern to you please contact Professor Sue Clarke.

What will happen to the results of this study?

The results of this study will form a report that will be available to Dorset HealthCare Trust staff. The results will also be published and made available to other patients on the service user's forum. The year of publishing will be around 2008. A copy of the report will be made available to you on your request. We would like to assure you that **results made available to people outside the research team will not include any information that makes you identifiable.**

Who is organising the study?

The study has been organised by Professor Sue Clarke and her research team. Sue is a Consultant Clinical Psychologist and is the Head of the Intensive Psychological Therapies Service in Poole. The intervention will be run by two experienced clinicians and a research team will be evaluating how effective this therapy is for the individuals involved.

LREC Approval
This study has been approved by the Dorset Research Ethics Committee and by the University of Southampton Ethics Committee.

Finally, we would like to reassure again of the following main points:

- **Participation is voluntary. You have the right to choose not to participate, or to stop participating in the trial at any point and without consequence.**
- **All the information you provide throughout the trial will be completely confidential. If a member of the team is given reason to believe that you may harm yourself or others, confidentiality may be breached.**
- **This information sheet is for you to keep. If you decide to participate, you will also be provided with a copy of the signed consent form.**
- **For any further information, please contact Jess Kingston (**contact details**).**

Thank you for taking time to read this information pack.

Information sheet for GPs, Consultants and other HealthCare Professionals

Research Background. ACT is a psychotherapy currently being used in the USA for patients with a range of mental health problems. ACT proposes that many mental health problems arise from, and can be made worse by, the avoidance of thoughts, feelings and bodily sensations. ACT aims to increase acceptance and willingness for distressing thoughts and feelings, and to motivate change through valued living. Research suggests that ACT is an effective therapy for a wide range of psychological difficulties, including; Opiate addiction (Hayes, Stroshal, et al., 2004); alcohol dependence (Heffner et al., 2004); psychosis (Bach & Hayes, 2002); anorexia (Heffner, 2002); depression (Zettle & Hayes, 1986); and PTSD (Orsillo & Batten, 2005)

Research Aims. To evaluate the effectiveness of ACT for treatment resistant patients; patients currently on the Dorset NHS, general adult mental health waiting list, who have received at least one form of psychotherapy in the past. The secondary aim is to investigate whether predicted mechanisms of change significantly increase from pre to post testing (e.g., acceptance)

Design. Therapy will run for 16 weeks and will be held by two ACT trained clinicians. Ten patients will be recruited for each group (two groups will be run sequentially). Groups will last approximately 2 ½ hours (with a 20 minutes break). Clinicians will monitor patients' progress throughout therapy and will be available after each session in case any patient is distressed. If any patient becomes stuck by barriers that cannot be adequately addressed in the group session, the patient will be offered an individual ACT session with one of the clinicians. For pre, post and 6 month follow-up assessment, patients will be asked to attend an interview at the IPTS (lasting between 60 and 90 minutes) and to complete a questionnaire pack in their own time (lasting approximately 90 minutes). A one day top-up session will be offered after this final assessment phase

Ethical considerations

1) All psychotherapeutic interventions can be experienced as distressing at times. For this reason, two clinicians will run each session, allowing for one to closely monitor the progress of each patient. Clinicians will be available during the break and after each session, and will ensure that no patient leaves the group significantly distressed.
2) Any patient who struggles with barriers that cannot be adequately addressed within the group setting will be offered an individual ACT session with one of the two clinicians running the group.
3) A clinician will be available during the collection of all pre and post data.
4) Participants will be asked to give full written informed consent before participating. Patients will also be provided with an information sheet explaining the aims of the study, detailing issues of confidentiality, and explicitly stating the right to withdraw at any time without effecting current or future rights to treatment.
5) All data collected will be treated in strict confidence and clients will be given anonymity.

Thank you for taking your time to read this information.

Appendix E

Protocol of ACT Treatment Sessions

Broad Structure:

- Mindfulness exercise (3-15 minutes)
- Mindfulness review
- Review Homework

BREAK

- Weekly Topic
- Final Summary
- Assign Homework
- Mindful Review.

Below is a review of each treatment session. The broad aims for each session are described, followed by possible exercises that could be useful to achieve those aims. In session examples are also provided to illustrate how clinicians addressed some of these aims in the treatment sessions of study 3. Techniques (e.g., metaphors, exercises) are referenced from Hayes et al. (1999) and Smith & Hayes (2005).

Creative Hopelessness

Session 1

Main Focus:

- Introduction, establish ground rules, commitment, and confidentiality.

BREAK

- To fully understand the nature of the difficulties the group present with and to take an inventory of their previous attempts to "control and eliminate this problem"
- To evoke a state of "creative hopelessness" by focusing on the relative failure of past attempts to control and eliminate their problems and the possibility that this is an "unworkable" system.
- To discuss human suffering as a ubiquitous and 'normal'; rather than 'abnormal'
- To expose participants to the possibility that there are other ways to relate to private events.

Possible exercises/metaphors:

- Suffering inventories
- Steering the car metaphor
- Digging out of the Hole metaphor

<u>Steering the car metaphor.</u> It's as if you got into your car and took off down the highway. Unfortunately, whoever taught you how to drive told you that the way to steer the car is by holding onto and turning the rear-view mirror. Now, you might be able to go a long way once you start driving, depending upon whether the road you're on is straight, or whether there is much oncoming traffic, etc. Eventually, however, the car is going to crash. The problem isn't with the car, or with the driver; the problem is that you can't steer a car with the rear-view mirror. This kind of therapy is not about how to turn the rear-view mirror, even though you may be convinced that that is what you need to learn. It's about how to put your hands on the steering wheel.

Homework: to fill out the suffering inventory during the following week.

In session example of creative hopelessness:

> Patient: I find my main problem is that I am too aware of what is going on in my body ... to try and make me more aware (of it), I think is absurd at the moment. Because I find I can't even lie down at night because of what's going on, and I don't want to keep on hearing all that... is that right or wrong?
>
> Therapist: if I'm hearing you right – if it's absurd to make you more aware, then actually your goal is to make yourself less aware … well how's that going for you?
>
> Patient: Umm, umm, yeah, I know what you are saying (PAUSE) it's not working very well really.

Session 2

Main Focus:

- Mindful awareness of breathing (5 minutes)
- Review of mindfulness exercise – what did participants notice? What did their minds have to say?
- Review thoughts and feelings about last week.

BREAK

- To socialise participants to a simplified version of the RFT model, aiming to show that minds have been trained to relate, evaluate, compare, judge, avoid (etc).
- Aim to undermine faith in the "control and eliminate agenda" by exploring further the possibility that avoidance, although natural and logical, may not be effective, because the struggle itself activates further similar processing.
- Consult the participants experience
- Mindful Review
- Homework setting

Possible exercises/metaphors:

- Gub-gub woo-woo
- See what our minds can relate
- Don't think about .... (yellow jeeps, thought X)
- Tug of war metaphor
- Chinese handcuffs

Homework: Coping strategies inventory

Chinese handcuffs: The situation here is something like those "Chinese handcuffs". Have you ever seen one before? It is a tube of woven straw about as big as your index finger. You push both index fingers in and as you pull them out the straw tightens and the harder you pull the tighter it gets and it traps your fingers. Once they're caught, you'd pull your fingers out their sockets trying to tug your way out. Maybe your situation is a little bit like that. Maybe the tubes are like life itself. There is no healthy way to get out of your life and the more you try the narrower your life becomes, the room gets restricted and you can't move. With this tube, the only way to free your fingers is to push them in, which makes the tube bigger. That's hard to do at first, because your mind tells you to look at

this problem in terms of "in and out" not "tighter and looser". Maybe you need to come at this situation from a different perspective from your logical mind perspective.

Tug of War: The situating you are in is like a tug of war with a monster. It is big and ugly and very strong. In between you and this monster is a pit, and so far as you can tell it is bottomless. If you lose this tug of war, you will fall into this pit and you will be destroyed. So you pull and pull and keep on pulling and the harder you pull, the harder the monster pulls, and you get closer and closer to the pit. Whilst you are struggling in this tug-of-war, the hardest thing for you to see is that your job isn't to win the tug-of-war..... it is to put down the rope.

Coping Strategies Diary					
Difficult Private Experience (thought, feeling memory)					
Distress/Disturbance Level	**1** Not distressing	**2**	**3**	**4**	**5** very distressing
Coping Strategy (my response)					
Short term Effectiveness	**1** Not effective	**2**	**3**	**4**	**5** very effective
Long Term Effectiveness	**1** Not effective	**2**	**3**	**4**	**5** very effective

In session example of avoidance:

Patient: I am going to go home and ring 'Mandy' because I can't cope with these thoughts going on in my head.

Therapist: and what's going to happen? What does your experience tell you is going to happen?

Patient: she will speak to me and for about 5 minutes and I'll feel calmer

Therapist: Great! Problem solved?

Patient: problem solved until 5 or 10 minutes later

Therapist: Yeah?

Patient: And I still feel bad and she hasn't made it go away.

Therapist: And then ..?

Patient: it's going to come back again

Therapist: see … it's a bit like going for a walk and if you always tread the same path, what happens? If you just keep walking in the same direction? Two things happen I think. One is that as the path gets deeper and deeper it just gets more and more engraved... If you always do what you have always done, then you'll always get what you've always got. Maybe there is another path ...? (*left as an open ended question*)

Control is the problem

Session 3

Main Focus:

- Mindful awareness of sight (5-7 minutes)
- Review mindfulness exercise
- Review thoughts and feelings about last week.
- Review Coping Inventories

BREAK

- Explicitly name *experiential avoidance* as the problem
- Discuss the pain of presence (the pain of the private events that the participant has but does not want) and the pain of absence (the pain of what their struggling prevents them from doing in life).
- Why do we do what doesn't work? The sheer logic of control and eliminate agenda.
- Rules for the 'outside of your skin' ('if you don't like it, work out how to get rid of it, and then get rid of it') rules for the 'inside of your skin' ('if you aren't willing to have it, you've got it')
- Use experiential exercises, suffering inventories and group discussion to explore EA
- Mindful review
- Homework setting

Possible exercises/metaphors:

- Polygraph machine metaphor
- Riding the mind train
- Thought controlling exercises

Homework: What have I given up for X?

Polygraph machine. Suppose I had you hooked up to the best polygraph machine that's ever been built, and I tell you, all you have to do here is stay relaxed. This is a perfect machine, the most sensitive ever made, so there's no way that you can be anxious and I won't know it. But I want to give you a little motivation. I happen to have a hand gun which I'll hold to your head. So I tell you, if you just stay relaxed I won't shoot you, but if you get nervous (and I'll know it because you're wired up to this perfect machine), I'm going to have to kill you. So, just relax! What do you think would happen: It's pretty clear, but notice this: If I told you, vacuum up the floor or I'll shoot you, you'd vacuum the floor. If I said paint the house or else, you'd be painting. But if I simply say, Relax, not only will it not work, but it's the other way around. The very fact that I ask you to do this under such circumstances would produce anxiety. But this isn't just a funny story. You have the perfect polygraph machine already hooked up to you: it's you own nervous system. And you've got something pointed at you that is more powerful and more threatening than any gun - your own self-esteem and success in life. It's like the gun, saying, Relax! Don't be anxious! And it's not working. If it's really, really important for you not to have a panic attack, guess what you get?

Riding The Mind Train: Living in your mind is like riding a mind train. A train has its own tracks and it goes where they lead. That's fine when the tracks lead where you want to go, but if you were going in the direction that you wanted to be going then you probably wouldn't be coming to this group. If the life that you want to live is off these tracks then you only have one option, you need to get off the train.... at least sometimes. Riding the mind train has become an automatic process. You believe the thoughts that you mind presents to you. Getting the train going in the first place happened innocently enough: you learned language, you learned how to speak, reason and problem solve. Once you did that, the mind train set up permanent residence in your life. There is no way that you can stop thinking and generating thoughts, you're mind will keep on running and language is very useful. But just because the mind keeps on running, doesn't mean that you always have to ride the train. On a real train, you can choose to ride when you want. When you take your thoughts literally, rather than merely a process of relating, you are riding the mind train. Would you like to have the choice of when to ride and when to get off?

In session example of control as the problem:

Patient: I'll say to my friend "Do you think I'm bored with my life?" and she'll say "No, of course not". (*starting to cry*)

Therapist: OK and that welling up happens?

Patient: she'll say "of course you're not, it's just one of those silly thoughts that you get". And the anxiety starts to come down. And I can walk away and then 2 seconds later it comes back and I feel the need to have to ask again and what if I am, and what if that thought is true?

Therapist**:** OK, and what happens to that panic?

Patient: it will go one notch higher. Higher than it was the first time

Therapist: So it's as if there's a tiger demanding to be fed. And when you're asking for reassurance, you're feeding the tiger. But he's so hungry that rather than creeping off he comes back and asks for more and more food. Is that right?

Patient: Yeah, that's exactly right.

Therapist: So the tiger gets fatter and bigger?

Patient: Exactly ... I go to try and get on with something, and then I think well what if they were wrong, and what if ...

Therapist: and then you have to ask someone else ... who is more credible?

Patient: and all that's happening is my anxiety level is just rising and rising and rising

Therapist: OK. So in the long term this tiger's getting bigger and bigger until your whole life is about feeding him to keep him at bay.

Patient: yes, that's right.

Therapist: So your experience tells you that this tiger is insatiable. What does your mind say?

Patient: I might just find the answer if I can find the right person

Therapist: Right. And I bet you thought that (therapist 2) and I might have the right answer?

Patient: yes

Therapist: (PAUSE). So the hungry tiger is always close demanding to be fed. (PAUSE). So what is, if anything, is it that he's keeping you from?

Patient: My life

Session 4

Main Focus:

- Mindful awareness of sound (5-7 minutes)
- Review of mindfulness exercise – what did participants minds do? What did they notice? What judgements, preconceptions, thoughts etc came up?
- Homework review

BREAK

- Acceptance as the alternative: what acceptance is and is not.
- Testing "If you're not willing to have it then you have got it" - Willingness to be out of breath.
- To be gentle, loving and caring towards yourself and your history
- Prompt participants awareness to direct experience
- What does your mind say about acceptance?
- Mindful review
- Homework setting

Possible exercises/metaphors:

- Willingness to be out of breath
- Thought controlling exercises
- Joe the Bum metaphor

Homework: Suppose it was the case that in order to feel a live a healthy, vital, meaningful and satisfying life you needed to give up trying to control your internal thoughts and feelings, before you could move in the direction you want to go. Would you be willing to do that?

Mindfulness exercise: Use the back of the chair for back support and notice the contact between the chair and your body, and rest your hands… Take your attention to the sounds that you can hear and notice the noises inside and outside the room… maybe the noises inside your own body…… every time your mind has something to say about those noises, judging – "this is good", "this is bad", just notice that that is what your mind is up to. Just come back to noticing the tiny details of the noises. And every time your attention wonders off… feelings thoughts and emotions… just notice and come back to the sounds that you can hear…. Again, just check where the attention is and if it

has wondered, bring the attention back to sounds….just see if you can notice this process of wondering off in your mind and then just coming back to the sounds

Willingness to be out of Breath Take a deep breath and hold it as long as you can. When you are finished, write down how long you held your breath for. I held my breath for. *Later in session:* We are going to hold our breath again. This time, I want you to: (1) Notice ***where*** the urge to breath begins and ends. (2) Just feel the feeling and see it as an opportunity to practise letting go. (3) Notice thoughts and thank your mind for that thought, without being controlled by it. (4) Notice and make room for your emotions. (5) In addition to your urge to breath, notice your bodily sensations and that your body continues to function. (6) Imagine that ***you*** are creating your urge to breathe. (7) Try to shift from seeing your urge as something unwelcome to something you have created deliberately, for the sake of the experience.

In session example of discussing acceptance:

Patient 1: I don't want to accept what I have got

Patient 2: That's what I was thinking

Therapist: Ok, "I don't want to do it".

Patient 1: If I umm, give in and accept what I have got I'll get worse, I really will.

Patient 2: But I have tried fighting and it didn't work.

Therapist: So suppose it were true 'patient 1', just for a moment, that the more you don't want something the more you have got it... the more unwilling you are to have it, the more you have got it. I'm not asking you to believe it, but just for a moment, suppose it was true. What would the implications be?

Patient 1: I feel if I try and accept it I will get worse and I will end up staying in hospital more … and when I to do things to avoid getting that way I feel that I am controlling it ...

S: ok, so you keep fighting

Patient 1: Doing the things I do throughout the day, I think I would just get worse.

Therapist: Ok, so that's what your mind tells you that you - it will end up getting worse

Patient 1: Yeah, that I will end up with people caring for me again. I really do.

Therapist: OK. And so all your investment goes into fighting it, fighting it…

Patient 1: Yep

Therapist: Not accepting it

Patient 1: Nope

Therapist: Fighting it. I've had some experience of doing that too, so I know what you are talking about. I don't think there s a person in this room that doesn't know what you are talking about. And does fighting it bring you closer to or further from the life you want?

Acceptance and Defusion

Session 5

Main Focus:

- Mindful awareness of touch (7-10 minutes)
- Review of mindfulness exercise
- Homework review

BREAK

- Noticing the process of thinking
- What are you thinking right now?
- Guide participants in feeling memories as memories, thoughts as thoughts etc
- Create exercises for the participant to experience fusion and defusion
- Watching the mind train: watching where you mind goes rather than riding in mindlessly
- Mindful review
- Homework setting

Possible exercises/metaphors:

- Watching the mind train
- What am I thinking right now?
- The virtues of saliva
- Milk, Milk, Milk

Homework: Bring a painful event to mind, keep a week's account of what you notice in your body and your mind when this shows up for you.

The Virtues of saliva: Saliva has many virtues - helps us swallow, digest food, protects gums etc. Imagine a spotless, beautiful crystal glass. Each time you have little extra saliva, release it into the glass, until the glass is full. Now really imagine drinking from this glass of saliva until it's empty. For most of us the idea of doing this is disgusting. A wonderful substance becomes a disgusting substance, just through thought

What are you thinking? Sit quietly for a few minutes and try writing down your thoughts as they run through your mind right now? What did you find? How many thoughts could you describe? Did thoughts about thoughts pop up?

Watching the Mind Train: Imagine you are standing on a railway bridge gazing down on three train tracks, with a slow, seemingly endless coal train on each track. On the left are things you notice in the present moment - bodily sensations, emotions, perceptions. On the right are urges to act - your pull to avoid, look away. In the middle are your thoughts, evaluations, predictions, self-conceptualizations. Looking down on these three tracks can be seen as a metaphor for looking at your mind. Start by thinking of something that's been troubling you lately, then close your eyes and picture the 3 tracks. Your job is to stay on the bridge, looking. If you find your mind has gone elsewhere, or you're in one of the cars, moving down the track, struggling with the content, notice what just hooked you and then mentally return to the bridge and look down again.

Session 6 and 7

Main Focus:

- Mindful awareness of taste (7-10 minutes)
- Review of mindfulness exercise
- Homework review

BREAK

- Looking *at* thoughts rather than *through* thoughts
- Explore the aims and goals of defusion; continue to create novel exercises allowing the participant multiple examples of experiencing both and their differences.
- For example: Labelling thoughts "I am having the thought that..." Leaves on a stream exercise. The pain creature: Describing thoughts and feelings in physical terms – what is its colour, size and character? Is there anything about the pain creature that the participant cannot bear to be present with?
- Mindful review
- Homework setting

Possible exercises/metaphors:

- Passengers on the bus
- Noticing thoughts in flight
- My mobile phone from hell
- "I am having the thought that..."
- What are my most favourite judgements about myself?
- Leaves on a stream exercise

Homework: Devise your own defusion exercises

Leaves on a stream: Imagine a beautiful and slow moving river. The water flows over rocks and trees and descends down hills and through valleys. Once in a while a big leaf drops into the stream and floats away down the river. Imagine you are sitting beside the stream on a warm, sunny day, watching the leaves float by. Now bring some awareness to your thoughts. Each time that a thought pops into your head, imagine that it is written on a leaf. If you think in words, then put the words onto the leaf. If you think in pictures, put pictures on the leaf. The goal is to stay beside the stream and to allow the leaves to keep flowing by. Don't try to make the stream go faster or slower and don't try to change what shows up on the leaves. If the leaves disappear, or if you mentally go elsewhere, or if you find that you are in the stream or on a leaf, just stop and notice that what has happened. File that knowledge away and then once again return to the stream, watch a thought come into your mind, write it onto the leaf and then let it float away downstream. You can think of the moments when the stream would not flow as moments of fusion, and those when the river did flow as moments as defusion.

In session mindfulness example:

> Therapist: Mindfulness is a practise of noticing where your attention is and noticing where your mind wonders to and then bringing it back to the here and now. It just so happens that in the 'here and now' toady is a raspberry or a raisin. In the same way as looking at pictures in detail, at the textures and shape, what we will do today is eating in great detail, eating very slowly so that you have the chance to notice the detail of how things taste.

Because naturally we eat quickly, or we're also nattering or watching the telly, we don't actually notice a great deal about what we are eating. This is a fun way of doing mindfulness in a different kind of way. I am going to talk us thought the experiencing the first raspberry or raison, and then you can then do it by yourselves for the second one.

Make a decision which you are going to eat first. I want you to really focus your attention on it, the weight on your hand. I am shaking a little so I notice the raspberry is shaking slightly. Notice colour, the way the light shines on it if that is happening. And then if you pick up, and very slowly start lifting it towards your mouth and just notice what happens, if your eyes have to refocus, what happens in your mouth and your hand. Bring it right up to your face and then breathe and see if you can notice any smell.

And at this point you might want to close your eyes – sometimes we can tune into smell if we don't have the visual data coming in. And if your mind wanders then that is fine because that's what they do. Just notice where it has wondered to and then come back to the real physical sensations – the smell and the touch.

And in your own time put it in your mouth but don't bite it straight away, have the first sensation of having this raspberry or raisin in your mouth and notice what happens, sharpness or sweetness, and then slowly chew and in your own time chew and swallow. And you are trying to notice every detail.

<u>In session defusion exercise:</u>

Therapist: in the presence of this thought "maybe it's a heart attack"... it's like narrowing down, you get ridged and stuck in the face of this thought. So what I am wondering is whether it is possible for us to hang out with this thought and have some different experiences with it. Because you are only having the stuck one. And I am making this up as I go, so if you think I'm off course by all means intervene! So this thought; if it had a shape what would that be?

Patient: crumbs! A circle.

Therapist: Shown me with your hands –what kind of size? And if it had a colour?

Patient: Red

Therapist: no hesitation. If it spoke with a foreign accent what would it be?

Patient: German

Therapist: So this is red circle with a German accent. So could you just say the thought for me- it doesn't have to be in a German accent.

Patient: The thought?

Therapist: Can you say the words "Maybe this time it's a heart attack"

Patient: Maybe this time it's a heart attack.

Therapist: OK this will get weirder and you can say no. Are you willing to get up and walk round with me with that thought? ...

Patient: Yes

Therapist: OK so I'm willing to do daft things with you. We are going to walk round and say "Maybe this time it's a heart attack!"

(*Both walk around saying it*)

Therapist: You can change the emphasis, play with it…… Would you be willing to do it in a German accent! (*they do it in a German accent*)

Therapist: So this is about playing with a set of words, a set of words that has the power to narrow down your willingness and flexibility to do what you want to be doing in life...

Defining the Self

Session 8

Main Focus:

- Mindful awareness of thoughts (10 minutes)
- Review of mindfulness exercise
- Homework review

BREAK

- Elicit key self-conceptualisations
- Practise defusion techniques with those self- conceptualisations
- Introduce the idea that this is just one type of 'self'
- Describe and provide participants with exercises to experience: Self-as-context, Self-as-process
- Mindful review
- Homework setting

Possible exercises/metaphors:

- The observing self
- The Chessboard
- The Rock Metaphor

Homework: Write down the key fact about yourself, for example, I have two parents, I am shy, I get depressed... etc. Then write a story about these facts in a way that is different from your life story. When this has been completed, write another possible story about the historic facts of your life.

Chessboard. Imagine a chess board that goes out infinitely in all directions. It's covered with different coloured pieces, black pieces and white pieces. They work together in teams, like in chess-the white pieces fight against the black pieces. You can think of your thoughts and feelings and beliefs as these pieces; they sort of hang out together in teams too. For example, "bad" feelings (like anxiety, depression, resentment) hang out with "bad" thoughts and "bad" memories. Same thing with the "good" ones. So it seems that the way the game is played is that we select which side we want to win. We put the "good" pieces (like thoughts that are self-confident, feelings of being in control, etc.) on one side, and the "bad" pieces on the other. Then we get up on the back of the white queen ad ride to battle, fighting to win the war against anxiety, depression, fear, sadness, whatever. It's a war game. The idea is that you knock enough of them off the board that you eventually dominate them. Except in this metaphor, it seems as though the battle

can't ever be won, because the black pieces can't ever be knocked off the board. So the battle goes on, every day, for years. You feel hopeless, you have a sense that you can't win, and yet you can't stop fighting. If you're on the back of that white horse, fighting is the only choice you have. But there's a logical problem here, and that is that from this posture, huge portions of yourself are your own enemy. And it appears that you're on the same level as them, and sometimes, that they are even bigger than you, that they're winning the war. But now suppose I were to say that, within the metaphor, those pieces aren't you anyway? Can you see, in that metaphor, who you would be? (Respond to all client's answers; ultimate answer is, "You are the board") Within this metaphor, if there were no board, what would happen to all the pieces? They'd just go away. Notice that if you're the pieces, the game is very important; you've got to win, your life depends on it. But if you're the board, it doesn't matter if the war stops or not. The game may go on, but it doesn't make any difference to the board. As the board, you can see all the pieces, you can hold them, have them played out on you, but it doesn't matter. It takes no effort.

In session example of distinguishing the different selves:

> Therapist: I'm just thinking that our minds hate this stuff. We are the dominant species and our mind hates the idea that this (*avoidance*) doesn't work. Luckily, there is more to us than our minds. It doesn't always feel this way, but… when you were having the thoughts like "I'll have to ask my friend for more reassurance" … who was it that noticed that thought? Pause…
>
> Patient silent but engaged and reflective
>
> Therapist:... Could it be possible that there is a "you" that notices where your mind is going ...? (PAUSE). Might it just be the case that even if your mind can't accept this possibility (*of willingness and valued living*), that you can? (PAUSE)
>
> Even with that chatter "…if I stop fighting I'll get worse" could it be that the person that notices this could be willing; even if your mind is not?

Session 9

Main Focus:

- Breathing Space and unguided mindfulness (10 minutes)
- Review of mindfulness exercise
- Homework review

BREAK

- Mindfulness: What and Why? The point isn't to relax, but to be aware of what is going on for you, without avoidance or fusion; to be able to flexibly respond and behave when your thoughts are dominating your experience
- The aim of mindfulness: To develop and deepen experience by paying attention to different aspects of experience.
- Mindfulness as a challenge: How to integrate mindfulness into your life
- Prompt participants to be aware of aware of direct experience rather than codified experience
- Mindful review
- Homework setting

Possible exercises/metaphors:

- Leaves on the stream

- Sitting meditation
- Body Scan

Homework: practise mindfulness for 15 – 20 minutes every day and jot down your experiences.

In session mindfulness exercise:

> Therapist: If you put your feet on the ground, sit back in your chair, trying to find a fairly upright position and rest your hands where ever is most comfortable. Try to settle yourself in a fairy upright and dignified way which shows your intent to be mindful and aware.
>
> So if you just be aware of the points of contact in the chair and just notice your feelings of your feet against the floor and of your hands. This week we are going to focus on breathing. So if you take your attention inwardly and just notice the fact that you are breathing and try to notice any physical sensation of air going in and out. Not trying to breath in any particular way, bringing a kind curiosity to your breathing.
>
> And if you notice that you are having thoughts and feelings about the breathing then that's fine, that's just what our minds do, so just notice where your attention has gone, just gently but firmly bring yourself back to the physical sensations of breathing in and out again.
>
> Sometimes when we take our attention to breathing we can unintentionally change the way that we are breathing by speeding it up … and this can feel uncomfortable. If you notice that, that's fine, that's what we do, and you can choose to keep your awareness with your breathing in a curious and non-judgemental way or you could choose to change your breathing slightly, but do that in a mindful way ...
>
> And now if you take either your left or right hand, up to you, and place it down on your abdomen and just lightly place it so it is comfortable. Notice the physical feelings of any movement of breathing against where your hand is resting on your abdomen.... And if your mind tries to contribute and say things, that's fine, just thank your mind and bring your attention back down to the feelings of movement against your hand as you breath in and out.
>
> And if you can't feel much movement then that's also fine, just notice what that's like, whatever is there for you is absolutely fine....
>
> Now broaden out your awareness to include the whole of your body in this chair so again just notice the feet on the ground, fitness of the ground, notice where your body rest, head down to toes. And in your own time open your eyes.

Session 10

Main Focus:

- Breathing Space and unguided mindfulness (20 minutes)
- Review of mindfulness exercise
- Homework review

BREAK

- Willingness: Saying 'yes' to the universe of private experience in the moment. The flexibility and freedom to choose action.
- Asking the participant to consider whether this is the right time for them to say yes
- The willingness scale exercise

- Mindful review
- Homework setting

Possible exercises/metaphors:
- The willingness scales
- What needs to be accepted?

Homework: What needs to be accepted? What would you be willing to have in the service of a richer life?

Willingness Scale. Imagine there are two scales, like the volume and balance knobs on a stereo, One is called "Anxiety" (or depression, or unpleasantness, etc). It can go from 0 to 10. The other is called "Willingness," and it can also go from 0 to 10. See what you think, but it is my hunch that what brought you in here is this: "My anxiety is too high. It's way up here and I want it down here and I want you [the therapist] to help me do that". But now there's also this other scale; it's been hidden but the past couple weeks we've been bringing it around to look at. This other scale, the Willingness scale, is really the more important of the two, because this is the one that makes the difference. When anxiety is up here at 10, and the willingness scale is down at 0, when you're trying hard to control this anxiety, make it go down, and you're unwilling to feel this anxiety, then by definition this means that anxiety is something to be anxious about. It's as if when anxiety is high, the willingness dial goes right down and this locks the anxiety into place. It's like trying to use a wrench when the ratchet is turned the wrong way. You turn the ratchet the wrong way and no matter what you do with that tool, it drives it in tighter. So, what we need to do in this therapy is shift our focus from the anxiety to the willingness scale. You've been trying to control anxiety for a long time, and it just doesn't work. It's not that you weren't clever enough; it simply doesn't work. Instead of doing that, if we turn our focus to the willingness scale, and let it go up, stop trying to control the anxiety, I guarantee you that your anxiety will be low... or it will be high! I promise you! It will be either low or it will be high. When it's low, it will be low... until it's high again! And it will be high, until it's not high, and then it will be low. We're not talking about going from `control' to `no-control', because that's really just doing the same thing, but at opposite ends of the continuum. The problem is that you're on this continuum at all. What is needed is a totally new context from which to operate.

Values

Session 11
Main Focus:
- Unguided mindfulness (15 minutes)
- Review of mindfulness exercise
- Homework review

BREAK
- The Life Question: Are you willing to feel, think, sense, and remember all your private experiences, fully and without defence, as you directly experience them to be, not as what your mind says they are *and* do whatever it takes to move you in the direction that you truly value? Yes or No – it is a real question.
- Learning to jump and the willingness scale. What psychological barriers stand between the participant and what is important to them? Am I willing to feel X?

- What does the participants mind say about willingness? Defusing from (e.g., physicalising) the difficulties their mind throws up.
- Mindful review
- Homework Setting

Possible exercises/metaphors:

- The willingness scales
- Physicalising
- Taking the problem apart; breaking the problem down.
- Creating a situation to test your willingness dial

Homework: Acceptance in real time. Write down ten scenarios that would bring up the negative content that you have been struggling with. Rate them from 1 – 10 and start with the first one this following week. The key here is to set your willingness high and your avoidance at 0. Decide when, where and how long you will do the action for and make a commitment to yourself to do it.

<u>In session example of values and willingness:</u>

Therapist: I'll start with a quote:

"*Security is mostly superstitious. It doesn't exist in nature, nor do the children of human kind as a whole experience it. Avoiding danger is no safer in the long run than out right exposure. Like is either a daring adventure or it is nothing at all*". (Helen Keller)

And this is very pertinent for what we are talking about today. So we are going to be working on this theme of values and learning how to jump. Having said yes to your internal experiences, how then do you being to make that move into a valued direction? ... (PAUSE)

I want you to settle into your chair, adopt a position of mindfulness and get present in the moment. So starting from the place in which there's a distinction between you as a conscious human being on the one hand, and all the private experiences that you are conscious of - and that sometimes struggle with - on the other hand, I'm going to ask you a question for you to sit with rather than answer here today. Pause Are you willing to feel, think, sense and remember all those private experience fully and without defence, as you directly experience them, as they are and not what you mind says they are? <u>And</u> do whatever it takes you to move in the direction of that which you truly value at this particular moment in this particular situation. And I want you just to sit within that question. Yes or no?

Now answering "yes" to that question is an example of jumping. It's not about getting rid of or managing your private experiences and history. It's about embracing them, picking them up and carrying them with you in a direction that you truly value. It's a jump in which you let go of the struggle with your history and become more concerned with being alive than with being right.

Importantly, *you don't have to say* yes, it's a real question. The other therapist, I, this group, life, will accept a yes or a no. Probably you know that your life has already accepted a no, but knowing that there are costs to silence and saying no, knowing that there is serious costs:… Pause and another quote: "*Most men lead lives of quiet desperation and go to the grave with the song still in them".* (Henry Thorea).

Session 12

Main Focus:

- Unguided mindfulness (15-20 minutes)
- Review of mindfulness exercise
- Homework review

BREAK

- Discuss what values are and help the participant to consider their key values
- Return to "passengers on the bus metaphor". The sign on the front of your life bus says "Values": these are your chosen life direction. Values are vitalising and empowering: not another mental club to beat yourself with
- Describe values as a compass set to go 'east', an intangible destination that you cannot arrive at but that makes following a particular path meaningful;
- What values are not (reasoned judgements, outcomes, feelings...)
- Values and pain, values and failing
- Choosing values
- Mindful review
- Homework setting

Possible exercises/metaphors:

- Skiing metaphor
- Making valued choices versus reasoned judgements
- Making a choice based on something other than logic: choosing between A and Z

Homework: what do you really want your life to be about? What matters to you? Work your way up your top painful scenarios (from session 11 homework task).

<u>The Skiing Metaphor:</u> Suppose you are skiing. You take a lift to the top of the mountain and you are just about to head down the slopes when a man comes along and he asks you where you are going? "I'm going to the lodge at the bottom" you reply. So he says: "I can help you with that" and he grabs your arm and he flings you into a helicopter and takes you to the bottom of the mountain. You are dazed, so you head back up, but just as you are about to head down, he comes back and does it again. You'd be upset – no? Skiing isn't just about getting to the lodge, it's about the journey. The lodge is your goal, but the skiing is your value. Of course, we need goals, but we must hold them lightly, so that the real point of living can emerge.

<u>In-session example (Passengers on the Bus):</u>

> Therapist: Values underpin the direction that you choose for your life; like to be a loving parent or to be present for your children. So if you think about life being like a bus journey. Imagine yourself as the bus driver and as you go thought life, various passengers get on your bus …. So you pick up some experiences, some memories and some rules that you have acquired along the way…. Some of these passengers are welcome, you are glad to have them as passengers on your bus. Others, however, are unwelcome. These ones you'd prefer not have on your bus. But the thing about this bus is that passengers can only get on, they can't get off. Once you have picked up these passengers, they will be with you until the end of the journey... (PAUSE)
>
> So you might try to strike deals with them: "I don't want you on my bus, but if you could just slouch back I might not notice you so much and I'll be more comfortable in my bus" or "If I don't go down this road, or go to this party, or let myself fall in love, will you keep quiet?" The trouble is that the more deals you strike, the less freedom you have to

choose the direction that your bus can take. Even though you are in the driving seat, the deals that you strike with the passengers ultimately determine the direction your bus is heading in... (PAUSE)

The alternative is acceptance – accepting the passengers are on your bus for the entire trip, and choosing a direction you want your life to head; taking them with you. So at the front of the bus is a sign that says where you are going, and we are encouraging you, as a first step, to think about that direction; a chosen direction that comes from your values. (PAUSE)

In order to do this, we need to work out what values are. Well, one way to describe them is like a compass that guides the direction of your life but that you can never obtain in the literal sense. So, one way to understand values and goals is that values are like going east whereas goals are like going to London. If you head for London you'll get there but if you are travelling east you will always be on a journey. It's like being a loving parent – you never get there, but you can always act in ways that serve that value ...

Session 13

Main Focus:

- Unguided mindfulness (15-20 minutes)
- Review of mindfulness exercise
- Homework review

BREAK

- What do you want your life to serve?
- Attending your own funeral
- Exploring the ten top valued domains
- Mindful review
- Homework setting

Possible exercises/metaphors:

- Attending your own funeral

Homework: Ranking your values. Working your way up your top painful scenarios (from session 11).

<u>In session exercise (Attending your own funeral):</u>

Therapist: If you could live your life so that it's actually about what you would choose it to be about, from here until it's over, what would be evident? That is, what would be clear about the sort of life you have led? This is not a prediction, guess or description. The question is not about what you have done or expect to do. It's not a question about social approval. The question is: "what would people be able to see if you could freely choose what your life stood for". I am asking you to open yourself up to your own yearning to be about something. If it were just between you and your heart, if no one would laugh and say it was impossible, if you were bold about your inner most aspiration, what would your life be about. And for it to be so powerful that it was evident to those people around you. (PAUSE)

So this is not about facing your death, it's about facing your life. But any value carries with it knowledge about how finite and limited our lives are. So this exercise is about imagining attending your own funeral, which obviously is an odd thing to do!

It may stir up strong feelings and thoughts, so just be kind and gentle with yourself. And if you find that you are getting very full of difficult thoughts and feelings then you know some of the steadying exercises we have done that you can bring to bear.

So what I am going to ask of you now is to close your eyes and take a few deep breaths. And then imagine that you have died, but that by some miracle you are able to witness your own funeral in a spirit form. So think about where it would be and what it would look like. Take a few moments visualising the picture of your future funeral service. And then imagine that a family member or friend, someone who knew you well, was there and that they had been asked to stand at your funeral to say a few words about what your life stood for. About what you cared about and the path you took. And you are going to write this eulogy in two ways.

First: I want you to write down what you are afraid might be said, if the struggle you are engaged in continued to dominate your life or even if it grew. So suppose you back off from what you really want to stand for, and you go for one of avoidance and mental entanglement and emotional control and back off from what you really stand for. I won't ask you to share this if you don't want to, so do not censoring what you say. And this, I imagine will be painful....

Secondly I want you to imagine from here forward you live your life to that which you most value. It doesn't mean that all your goals will be magically attained, it means the direction you are taking in life is evident and clear and manifest. Now imagine who is at your funeral, certainly loved ones, children, closest friends, people who care about you, people from work, people you studied with, people from other organisations, church … anyone you like can come to this funeral. Look at their faces; watch them watching your funeral. So now you choose one of them again to stand up and say a few words about you, if your life had been true to your inner most values, imagine what you would most want to have manifest in your life. You won't be judged on this and you may choose never to share this with anyone. And this isn't a prediction, and this isn't self praise, let these words reflect the meaning you would most like to create.

Committed Action

Session 14

Main Focus:

- Unguided mindfulness (15-20 minutes)
- Review of mindfulness
- Homework review

BREAK

- Is the participant willing to accept whatever discomfort their mind provides AND commit to the values they have explored AND to the behaviour changes they imply?
- Creating a road map by setting goals
- Short term goals and long term goals
- Making goals happen through commitment to action
- Psychological barriers
- Building patterns of effective action: old behaviour =____ new behaviour =___
- Breaking up old patterns by staying mindful of values.
- Mindful review

- Homework setting

Possible exercises/metaphors:
- Goal setting
- Climbing mountains metaphor
- Practising making commitment

Homework: Keep a weekly record of how important each of the ten valued domains is to you and consistent your behaviours were with each value. Note down barriers to change.

Session 15
Main Focus:
- Unguided mindfulness (20 minutes)
- Review of mindfulness
- Homework review

BREAK
- The crucial fork in the road. The participants' opportunity to choose a direction; the well trodden and familiar path of avoidance or following values?
- Discuss that following values can be vulnerable and 'risky'. It can and will be painful. The participant has the ability to choose for themselves.
- The avoidance cycle or the acceptance cycle: which will it be?
- The real choice is not whether or not to have pain, but whether or not to live a valued and meaningful life
- Mindful review
- Homework setting

Homework: Commit to a valued goal towards the top of your "top ten" scenarios from session 11.

Session 16
Main Focus: Whole session allocated to participants commitment to a valued action (however small). Chairs faced forward in the room and each participant stands in front of the group, one at a time, and makes a commitment to a valued change in their life.

<u>In session example of the commitment exercise:</u>

> We are going to ask you to come up in front of the group in turn, and get present to each other. Get present to the fact that there are eight human beings here, people who have been here with you for the past 16 weeks. So, if you can, we invite you to settle in to this moment and appreciate these other people here today. And in your own time, in your own way, we invite you to express to the other members of the group what's important to you in your life, what really matters to you. And then say something about what you've noticed you've been doing, for however long it has been, and what the costs have been for you. And if you can, and if you're willing, we invite you to say something about what you may commit to doing differently, in the service of what matters to you: your values. We know that it won't always be possible to tread this valued path, for example you may not *always* be available for your kids. So, your commitment may be that when you notice that you're not going in a valued direction, you turn back in the direction of your chosen path....

Appendix F

Study IV Patient Information Sheet and Information for General Practitioners

In the following information sheet, all references to ACT and CBT (i.e., in titles and text) were randomised so as to prevent implicit bias.

A Pilot Randomised Trial Investigating the Effectiveness of Cognitive Behavioural Therapy (CBT) and Acceptance and Commitment Therapy (ACT).

Patient information sheet

You are being invited to take part in a research study. Before you decide whether you would like to take part, you need to understand why the research is being done and what it would involve. Please take time to read the following information carefully. Talk to others about the study if you so wish.

- Part 1 tells you the purpose of this study and what will happen to you if you take part.
- Part 2 gives you more detailed information about the conduct of the study.

If there is anything that is not clear, or if you would like more information, please do not hesitate to contact Sue Clark on (contact details) or Debbie Lee (contact details). Please could we ask that you contact the Intensive Psychological Therapies Services (IPTS: contact details) within one week of receiving this information sheet to let us know whether you would like to participate.

Part 1

What is the purpose of this study?
This is a small scale pilot trial, which is designed to make some preliminary investigations about the effectiveness of Cognitive Behavioural Therapy (CBT) and Acceptance and commitment Therapy (ACT) for patients who have already received psychological care in the past. This study has been designed by a senior clinical (Prof. Susan Clarke) and a senior academic (Prof. Bob Remington) researcher; and some of the information collected from this trial will contribute to the completion of a PhD thesis.

Why have I been chosen?
We are recruiting patients who are currently on the Dorset HealthCare NHS Trust waiting-list for general adult mental health, and who have received at least one form of therapy in the past that lasted at least 8 sessions. You were identified as currently awaiting therapy at The Chines. Our records indicate that you meet these criteria and we would therefore like to offer you a place on the trial. We aim to recruit a maximum of 40 patients; 20 patients for therapy groups that are due to run from April to July, and 20 patients for groups due to run from September to December.

Do I have to take part?
It is up to you whether you take part. If you do decide to take part, you will be asked to sign a consent form.

Having signed the consent form, you are still free to withdraw at any time and without giving a reason. A decision to withdraw, or a decision not to take part, will not affect the standard of care you receive.

What will happen to me if I take part; what will I have to do?
Sometimes we don't know which way of treating patients is best. To find out, we need to compare different therapies. In order to compare different therapies, we collect information about patients both before therapy and after therapy. We then put patients into groups and give each group a different therapy. To make sure the groups are the same to start with, each patient is put into a group by chance (randomly). Patients then complete the same questionnaires and attend an interview a few weeks after therapy and 6 months after therapy. This allows us to see whether there have been any changes before and after therapy, and whether these changes last over time. Because this is a pilot trial we will not be able to test whether one is better than the other, but we will gather information about how helpful they both are.

Before therapy. If you decide to take part, you will be sent a questionnaire pack and invited to an interview. The questionnaires take about 60-90 minutes to complete. These are for you to complete in your own time, but assistance is available if you would like. The interview will be held at the IPTS and will last about 60-90 minutes. This interview will ask about the therapy you have had in the past and about your psychological difficulties.

Therapy. You will then be randomly assigned to either ACT or CBT. You have 50% chance of receiving 16-weeks of group ACT and 50% chance of receiving 16-weeks of group CBT. We will write to you within a week of the interview to let you know which group you have been allocated to.

After therapy. After the 16 weeks of therapy you will be asked to complete another set of questionnaires and come to second, shorter interview. In this interview we will ask you how you found the group. Six months after therapy you will be asked to complete a final set of questionnaires and attend a final interview. After therapy you will also be provided with a CD to help practise skills learnt in the group.

Data and audio taping. To make sure the interviews and group sessions are delivered correctly, we ask your permission to audiotape them. We will also ask your permission for members of the research team to have access to your questionnaire responses. All of your completed questionnaire responses will be stored anonymously in a secure cabinet either at the University of Southampton or at the IPTS.

Restrictions during and after therapy. If you take part, you can continue taking any medication. We ask that for 6 months after ACT/CBT you don't attend any other form of therapy. This is routine practice and is referred to as a consolidation phase. After we have collected the 6 month follow-up data you can opt in for standard care if you so choose.

Attendance. We ask you to come to all scheduled visits and to complete all the questionnaires. If you are going to miss a group, we ask you to let the clinic know beforehand. If you don't come to four therapy sessions in a row, you will not be able to

continue coming to the group. This is to help you from unintentionally drifting out of therapy. It also helps group morale.

This information has been put into a summary flow chart found on the back page.

What are the therapies that are being tested?

ACT is a therapy being used in America for patients with a range of psychological difficulties. ACT suggests that when people try to avoid distressing thoughts and feelings, they get entangled in a mental battle against themselves. ACT uses exercises such as mindfulness meditation to help people accept these thoughts and feelings as events of the mind that can be observed and then let go of. The effectiveness of ACT was first assessed in 1986 with a group of depressed patients. Both this trial and many subsequent trials have indicated that ACT can be successful for patients with various psychological difficulties. A group has recently been run at the IPTS in Poole, and this found that ACT helped to reduce patients' levels of depression and anxiety, and increase quality of life. Before therapy 100% of the group had clinical depression. After therapy, 5/6 patients had clinically reliable reductions in depression and 50% finished therapy with non-clinical levels of depression.

CBT takes a different perspective. CBT suggests that certain thoughts and feelings cause emotional distress. CBT identifies what these thoughts and feelings are and uses exercises to challenge how true they are. CBT helps patients by trying to change these thoughts and feelings. The first manual for CBT was published in 1979 for patients with depression. Since then, a lot of research has suggested that CBT is effective for patients with a range of psychological problems. CBT groups are currently being offered on the NHS as a 10 week program. This 16 week group has been designed to address the needs of a group who have already received some form of psychological care in the past. We are only offering these 16 week CBT groups to patients in this study.

What are the alternatives for treatment?

You have the choice not to participate in this research trial. If you don't want to take part in this trial, your name will stay on the waiting-list for therapy at the Chines.

If you don't want to participate in this study, please let us know by phoning the number provided.

What are the possible risks or disadvantages of taking part?

As with any therapy, you may sometimes feel emotionally distressed. Your well-being will be monitored by the clinicians during every session, and they will make sure that no one leaves the group significantly distressed. Patients who struggle with problems that cannot be addressed adequately in the group will be provided with an individual therapy session. The clinicians are all trained and have experience at delivering the relevant therapies. They will also receive supervision whilst the groups are running.

A possible disadvantage is the inconvenience of questionnaires and interviews. These have been kept to a minimum and will be done in a way that is as convenient as possible. We will offer you feedback on the questionnaires and interviews at the end of the 16 weeks. A second possible disadvantage is that you will be randomised to one of the two conditions (CBT or ACT) rather than choosing which therapy you would like.

What are the potential benefits of taking part?

If you take part you are guaranteed 16 weeks of therapy. If you are in the CBT group you will get 6 weeks more therapy than the CBT group currently offered by the NHS. In

addition to this, you will be attending a CBT group that has been designed to meet the needs of patients who have already had psychological care in the past. If you are in the ACT group, you will get a new and promising therapy that is not currently offered by the NHS.
Although group based therapy can seem daunting, it has many benefits. For example, you can develop both from active participation and from observation; you have the opportunity to give and receive immediate feedback; and you have the opportunity for support from people who are experiencing similar difficulties. Group based work can also help people to understand concepts discussed in therapy. For example, sometimes it is difficult to apply concepts or skills to one's own life, but seeing them worked out in another can help us come to grips with them. Many patients find that group-based delivery can actually enhance their experience during therapy. Participation in group therapy does not require you to share personal information.

Whilst we expect these groups to be of benefit to you, we cannot guarantee this.

What happens when the research study stops?
Immediately after the 16 weeks, and 6 months after the 16 weeks we will ask you to complete a questionnaire pack. Six months after therapy we will also ask you to attend an interview.

What if there is a problem?
Any complaint about the way you have been dealt with during the study or any possible harm you might suffer will be addressed. The detailed information on this is given in Part 2.

Confidentiality –who will know that I am taking part in this study?
All the information about your participation in this study will be kept confidential. The details are included in Part 2.

For further information
If you would like any further information about the trial, please do not hesitate to contact Professor Sue Clarke (contact details), or Miss Jess Kingston (contact details).

This completes Part 1 of the Information Sheet. If the information in Part 1 has interested you and you are considering participation, please continue to read the additional information in Part 2 before making any decision

Part 2

What if relevant new information becomes available?
Members of Sue Clarke's research team are currently monitoring and will continue to monitor the relevant internet servers and discussion forums. If any evidence comes to light that there are any adverse effects to either intervention, your clinician will inform you of these details and ask you whether you would like to continue with the trial. If you decide to continue in the study you will be asked to sign an updated consent form. If you decide not to continue with the trial your continuing care will be arranged. It is also possible that, on receiving new information, the clinician feels that it is in your best interests to withdraw from the study. In the unlikely event that this happens, she will

explain the reasons and facilitate the continuation of your care. If the study is stopped for any other reason, you will be told why and your continuing care will be arranged.

What will happen if I don't want to carry on with the study?
You are free to withdraw from treatment at any stage. If you withdraw, we will need to use the data collected up to your point of withdrawal, but this will only be available to members of the research team and will not be stored with information that can identify you. With your permission, we would also like you to complete post-intervention questionnaires and attend the interview despite you not completing the group. However, you will retain the right not to do this if you so choose.

What if something goes wrong?
It is unlikely that this therapy will cause you any harm. Trained clinicians will be available at every stage of your involvement.

If you have a concern about any aspect of this study, you should phone Sue Clarke (contact details). If you remain unhappy, you have the right to complain to the NHS about any aspects of the way you have been approached or treated during the course of this clinical trial. In the event that something does go wrong and you are harmed during the research study there are no special compensation arrangements. If you are harmed and this is due to someone's negligence then you may have grounds for a legal action for compensation against Dorset NHS, but you may have to pay your legal costs.

Will my taking part in this study be kept confidential?
All information collected about you during this study will be kept strictly confidential and any information about you that leaves the clinic's will have your name and addressed removed so that you cannot be recognized. Your data will not be stored with any personally identifying information, and will be stored in securely locked cabinets and password protected computers. You will be asked at the beginning of the trial to choose a participant number. This will be stored with the data. Your personal details (e.g., name and address) will not be made available to anyone other than members of the research team and they will be held in a secured office at either The Chines or The IPTS.

Please be aware that if a member of the team is given reason to believe that you may harm yourself or others, confidentiality may be breached.

Healthcare professional involvement.
If you decide to take part, we will inform any healthcare professionals (e.g., GP's) currently involved in you care, and provide them with some brief information about the therapy. If the clinician feels that it is necessary to share any information acquired during the trial with members of your healthcare team, you will be asked first. If this is of concern to you please contact Professor Sue Clarke.

What will happen to the results of this study?
The results of this study will form a report that will be available to Dorset HealthCare Trust staff. The results will also form part of a PhD thesis. We intend to publish our findings and to also make them available to other patients. The year of publishing will be around 2008. A copy of the report will be made available to you on your request. We

would like to assure you that **results made available to people outside the research team will not include any information that makes you identifiable.**

Who is organising the study?
The study has been organised by Professor Sue Clarke and her research team. Sue is a Consultant Clinical Psychologist and is the Head of the Intensive Psychological Therapies Service in Poole. The sponsors of this study will pay members of the research team for evaluating your participation in this study.

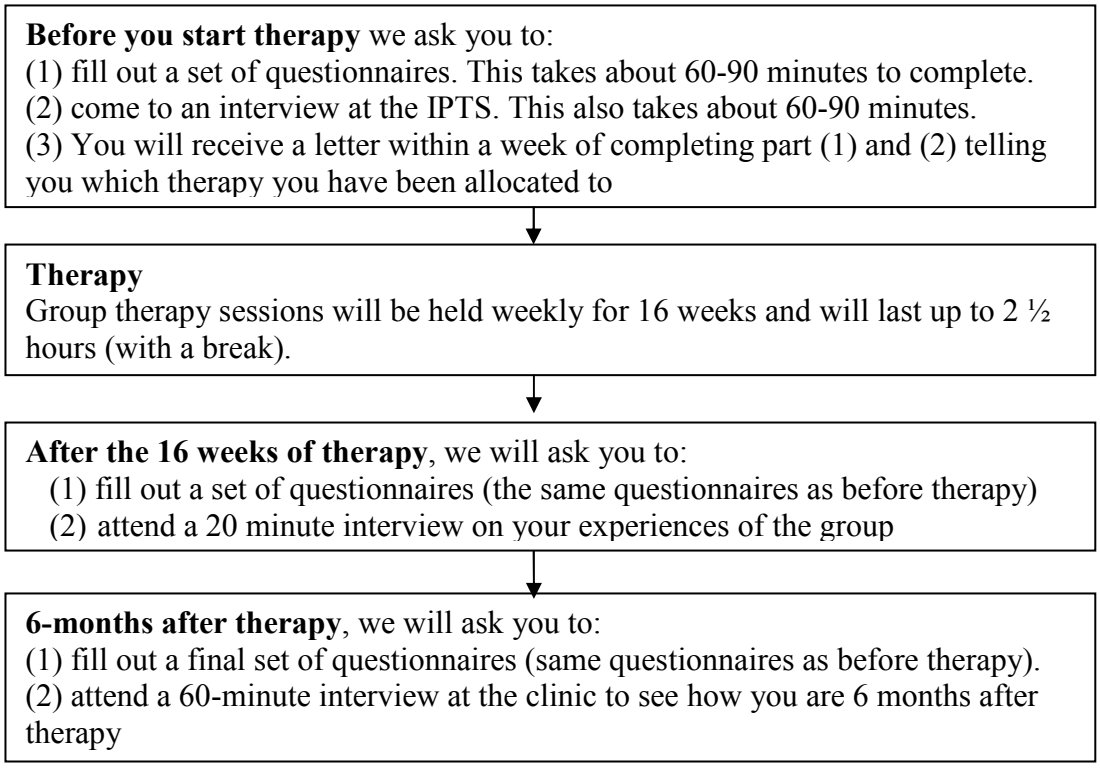
LREC Approval
This study has been approved by the Dorset Research Ethics Committee and by the University of Southampton Ethics Committee. If you have questions about your rights as a participant in this research, or if you feel that you have been placed at risk, you can contact the Chair of the Ethics Committee, Department of Psychology (contact details).

Thank you for taking time to read this information pack.

Summary

- **Participation is voluntary. You have the right to choose not to participate, or to stop participating in the trial at any point and without consequence.**
- **All the information you provide throughout the trial will be completely confidential. If a member of the team is given reason to believe that you may harm yourself or others, confidentiality may be breached.**
- **This information sheet is for you to keep. If you decide to participate, you will also be provided with a copy of the signed consent form.**
- **For any further information, please contact The IPTS (**contact details**).**

The following flowchart outlines what you will be asked of you if you decide to take part.



General Practitioner Information

A Pilot Randomised Trial Investigating the Effectiveness of Acceptance and Commitment Therapy (ACT) and Cognitive Behavioural Therapy (CBT): Information sheet for GPs, Consultants and other HealthCare Professionals

Research Background. ACT is a new psychotherapy currently being used in the USA for patients with a range of mental health problems. ACT proposes that many mental health problems arise from, and can be made worse by, the avoidance of thoughts, feelings and bodily sensations. ACT aims to increase acceptance and willingness for distressing thoughts and feelings, and to motivate change through valued living. Research suggests that ACT is an effective therapy for a wide range of psychological difficulties, including; Opiate addiction (Hayes, Stroshal, et al., 2004); alcohol dependence (Heffner et al., 2004); psychosis (Bach & Hayes, 2002) and depression (Zettle & Hayes, 1986). An ongoing, uncontrolled, pre-post trial at the Intensive Psychological Therapies Service (IPTS) has suggested ACT can decrease depression and anxiety in patients who have been resistant to other therapy in the past ("treatment resistant patients").

Research Aims. This trial aims to make preliminary investigation into how effective ACT is compared to Cognitive Behavioural Therapy (CBT). Both CBT and ACT will be offered as group therapy and will last 16-week. Treatment resistant patients have been defined as those patients currently on the Dorset NHS, general adult mental health waiting list, who have received a psychotherapeutic intervention at least once in the past, which lasted for at least 8 sessions.

Design
This is a randomised comparison trial with pre-post assessment. Patients will be randomly allocated to either ACT or CBT. The trial aims to recruit 8-10 patients per group. Both ACT and CBT will be run by two experienced and trained clinicians. Groups will run for 16 weeks. Clinicians will monitor patients' progress throughout therapy and will be available after each session in case any patient is distressed. If any patient becomes stuck by barriers that cannot be adequately addressed in the group session, the patient will be offered an individual session with one of the clinicians.

Methodology
Patients will attend one pre-intervention interview (the SCID-II and an assessment of previous therapy) and will be asked to complete a set of questionnaires in their own time. Interviews will be held at the Intensive Psychological Therapies Services (IPTS) and will last approximately 90 minutes. A similar assessment phase will also be held after the intervention. Six months after the intervention, patients will be asked to attend another interview and complete a final pack of questionnaires.

Ethical considerations.
1) All psychotherapeutic interventions can be experienced as distressing at times. For this reason, two clinicians will run each session. This allows for one clinician to closely monitor the progress of each patient. The clinicians will be available both

during the break and after each session, and will ensure that no patient leaves the group significantly distressed.

2) Any patient who struggles with barriers that cannot be adequately addressed within the group setting will be offered an individual session with one of the two clinicians running the group.
3) The researcher collecting pre and post assessment will be closely supervised by the Chief Investigator (Prof Susan Clarke) and a clinician will be on site during the collection of all pre-post assessment.
4) Participants will be asked to give full written informed consent before participating in the study. This consent will be accompanied by an information sheet explaining the aims of the study, detailing issues of confidentiality and randomisation, and explicitly stating the right to withdraw at any time without effecting current or future rights to treatment.
5) All data collected will be treated in strict confidence and clients will be given anonymity.

This study is funded by an Economic and Social Research Council (ESRC) CASE grant that has been awarded to Prof. Bob Remington and Prof. Susan Clarke. Miss J Kingston has been appointed as a PhD studentship to collect the pre and post assessments which, in addition to providing data for the proposed research, will contribute to her thesis.
This trial has been approved by the Dorset Ethics Committee and by the University of Southampton Ethics Committee.
Thank you for taking your time to read this information.

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