

**Factors Affecting Treatment  
Completion and Treatment Outcome in  
a Naturalistic Study of Psychological  
Therapy for Personality Disorder**

**Susie Rudge**

**D.Clin.Psy Thesis (Volume 1)**

**2014**

**University College London**

**UCL Doctorate in Clinical Psychology**

## **Thesis Declaration Form**

I confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

**Signature:**

**Name:**

**Date:**

## Overview

This three-part thesis focuses on psychological therapy for personality disorders (PDs) and factors that influence both treatment completion and outcome.

Part one is a literature review investigating documented mechanisms of change in the cognitive behavioural therapy (CBT) and dialectical behavioural therapy (DBT) treatment for borderline personality disorder (BPD) in current research. Although much research has focussed on improving treatment outcomes for BPD, there is very little research investigating the proposed changes by which these outcomes might occur. Three distinct categories of mechanism of change were found to be consistent across the literature examined. These categories are discussed in detail along with implications for future research and clinical practice.

Part two presents a longitudinal empirical study of factors which affect treatment completion and treatment outcome in the CBT or DBT treatment of PD. Data spanning a six year period was collected and analysed for 231 patients. Results showed that therapist expertise was the only variable examined associated with treatment completion: more experienced therapists retained their patients in treatment for longer than less experienced therapists. Therapeutic dose (number of sessions attended), therapist expertise and substance misuse all predicted changes in risk outcome (deliberate self-harm, suicide attempts) and in number of PD diagnoses following treatment. Only therapeutic dose predicted change in other clinical diagnoses following treatment. Implications and strength of these findings are discussed in relation to problems with incomplete data, statistical analyses and non-representative sampling issues.

Part three is a critical appraisal of the entire research process reflecting upon its challenges and successes. This section also includes a commentary on the field of PD research in general, and considers issues pertinent to future research.

## Table of Contents

<b>Declaration</b> .....	2
<b>Overview</b> .....	3
<b>Table of Contents</b> .....	4
<b>List of Tables</b> .....	5
<b>List of Figures</b> .....	6
<b>Acknowledgements</b> .....	7
<b>Part One: Literature Review</b> .....	8
Abstract.....	9
Introduction.....	10
Method.....	14
Results.....	18
Discussion.....	35
References.....	43
<b>Part Two: Empirical Paper</b> .....	52
Abstract.....	53
Introduction.....	54
Method.....	66
Results.....	74
Discussion.....	89
Conclusion.....	99
References.....	100
<b>Part Three: Critical Appraisal</b> .....	115
References.....	126
<b>Appendices</b> .....	132
Appendix A: Downs & Black's Criteria for Critical Appraisal of Studies.....	133
Appendix B: Ethical Approval Letters.....	134
Appendix C: Christo Inventory for Substance Misuse Services (CISS).....	135

## List of Tables

### Part One: Literature Review

Table 1	Papers included in the review.....	20
Table 2	Checklist appraisal of DBT studies.....	22
Table 3	Checklist appraisal of CBT studies.....	22

### Part Two: Empirical Paper

Table 1	Demographic characteristics of treatment completers and non-completers at baseline.....	69
Table 2	Treatment type of treatment completers and non-completers at baseline.....	70
Table 3	Personality disorder profiles of treatment completers and non-completers at baseline.....	70
Table 4	Mean number of sessions attended, clinical comorbidity and substance misuse for treatment completers and non-completers.....	77
Table 5	Predictors of treatment completion correlation matrix.....	78
Table 6	Logistical regression exploring the role of therapist expertise in treatment completion.....	79
Table 7	Treatment completion category and therapist expertise.....	81
Table 8	Predictors of treatment outcome correlation matrix.....	82
Table 9	Change in risk, change in PD diagnoses and change in clinical syndrome diagnoses.....	83
Table 10	Multiple hierarchical linear regression: prediction of risk change outcome.....	85
Table 11	Multiple hierarchical linear regression: prediction of PD diagnoses outcome.....	86
Table 12	Change in clinical comorbidity between baseline and treatment completion/dropout.....	87
Table 13	Multiple linear regression: prediction of change in risk, change in PD diagnoses and change in clinical syndrome diagnoses - treatment completers only.....	89

## List of Figures

### Part One: Literature Review

- Figure 1 Flow chart of review database.....19
- Figure 2 Relationship between emotion dysregulation and borderline  
behaviour patterns (Linehan's (1993) biosocial theory).....37

## **Acknowledgements**

I would like to thank my supervisor, Dr. Janet Feigenbaum for her extremely valuable contribution to this project, as well as the clinical staff of the service in which the data was collected. I would like to extend a special thank you to Oliver English and Leng Song, Assistant Psychologists, for their hard work assisting with data collection and scoring. I would also like to thank Ravi Das, UCL Stats Demonstrator for his input during the data analysis part of this project.

Personally, I would like to thank my parents, Gayle and Rod for encouraging and supporting me in my pursuit of the Doctorate in Clinical Psychology, as well as always believing in me in everything I strive for. Special thanks also for providing invaluable childcare when I most needed it!

Finally, the biggest thanks go to my wonderful husband, Kev for his endless love, patience and support and to my beautiful baby daughter, Aimee. Thanks for getting me through this and for making my dream of finishing my Doctorate as a wife and mother a reality.

## **Part 1: Literature Review**

# **Mechanisms of Change in Cognitive Behavioural Therapy and Dialectical Behaviour Therapy for Borderline Personality Disorder**



## Abstract

**Aims:** Little is known about the 'active ingredients' of psychological therapy for Borderline Personality Disorder (BPD) despite a growing evidence base documenting its clinical effectiveness. This review analyses studies investigating potential mechanisms underlying therapeutic change in Cognitive Behavioural Therapy (CBT) and Dialectical Behaviour Therapy (DBT) for BPD.

**Method:** A thorough search of the PsychInfo, CINAHL Plus, PubMed, MEDLINE and EMBASE databases revealed empirical research on the potential mechanisms of change.

**Results:** One hundred and four references were identified and 34 abstracts reviewed. After a full text screen of the most relevant studies, nine met inclusion criteria. Seven examined DBT and two CBT. Mechanisms of change identified broadly fell into three categories: *emotion regulation/self-control*, *skills use* and *therapeutic alliance/investment in treatment*. Outcomes measured included general clinical syndromes (anxiety/depression) and BPD-specific symptoms (self-harm/suicidality, impulsivity, substance misuse, anger).

**Conclusion:** Further empirically-robust research is required to test hypotheses about the influence of the proposed mechanisms on therapeutic change in treatments for BPD.

## Introduction

Personality Disorder (PD) is a condition previously defined on Axis II of the Diagnostic and Statistical Manual of Mental Disorders-IV-TR (DSM-IV-TR; American Psychiatric Association, 2000) as, “an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual’s culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment” (p. 685). However, although the newly published fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) uses the same definition of PD, it no longer makes a distinction between Axis I (clinical syndromes such as anxiety and depression) and Axis II (entrenched, pervasive patterns of behaviour reflecting an individual’s inherent personality characteristics), instead combining the first three Axes outlined in previous editions into one Axis incorporating all mental and other medical diagnoses. It is thought that this change will benefit both clinical practice and scientific research (American Psychiatric Association, 2013).

The ten distinct types of PD remain in DSM-5 as they were in DSM-IV-TR, dividing into three clusters based on their descriptive similarities. The current review, however, will focus solely on Borderline Personality Disorder (BPD), described by Bateman and Fonagy (2004) as, “a complex and serious mental disorder that is characterised by a pervasive pattern of difficulties with emotion regulation and impulse control and instability both in relationships and self-image” (p.1). BPD is arguably one of the more common personality disorders seen in PD services (Coid, Yang, Tyrer, Roberts & Ullrich, 2006; de Ruiter & Greeven, 2000) and is highly studied as it is associated with high rates of suicide, self-harm, violence, and drug/alcohol addiction (American Psychiatric Association, 2013), therefore resulting in a high level of service usage (Bender et al., 2001; Comtois et al., 2003) and high

mortality rates (American Psychiatric Association, 2001). Many research efforts have therefore attempted to identify effective treatment for the condition.

One of the obstacles influencing effective treatment for BPD is the problem of treatment adherence. Several characteristics of the disorder (e.g. impulsivity, recurrent suicidal behaviour) unfortunately lend themselves to early disengagement from treatment and difficulty committing to and engaging with the therapeutic process.

BPD is characterised by difficulties in establishing trusting and collaborative interpersonal relationships and, “frantic efforts to avoid real or imagined abandonment” (American Psychiatric Association, 2013) which naturally extend to difficulties in the therapeutic relationship, thus presenting further challenges for treatment.

However, contrary to previous opinion which held that due to its entrenched roots in childhood personality development, PD was largely untreatable, there is now evidence to suggest that BPD and other PDs are not immutable and are likely to change over time with successful psychological treatment (e.g. Bateman & Fonagy, 2000; Bateman & Fonagy, 2004; Bateman & Fonagy, 2008; Bloom, Woodward, Susmaras & Pantalone, 2012; Kliem, Kröger & Kosfelder, 2010; Panos, Jackson, Hasan & Panos, 2013).

Many research efforts have attempted to ascertain the efficacy of different psychotherapies used for the treatment of BPD including Dialectical Behaviour Therapy (DBT) for women which is currently the only treatment recommended for BPD by the National Institute for Clinical Excellence (NICE, 2009; Stoffers et al., 2012). NICE does, however, suggest that should the evidence base be produced, future revisions may advocate the treatment of BPD using Cognitive Behaviour Therapy (CBT; Beck, Freeman & Davis, 2004), Mentalization Based Therapy (MBT; Bateman & Fonagy, 2008; Bateman & Fonagy, 2004) and Schema Therapy (Kellogg & Young, 2006). DBT could be described as a third-wave CBT therapy and has a

growing large and robust evidence base (e.g. Bloom et al., 2012; Feigenbaum et al., 2011; Feigenbaum, 2007; Kliem et al., 2010; Linehan et al., 2006; Panos et al., 2013). Likewise, strong bodies of empirical evidence reliably document the effectiveness of both generic CBT (Butler, Chapman, Forman and Beck, 2006) and CBT specifically for BPD (Davidson et al., 2006). The current review will therefore focus solely on the CBT and DBT treatment of BPD.

CBT uses traditional cognitive and behavioural techniques to teach patients to identify dysfunctional thoughts and core beliefs and to learn to challenge and modify them. CBT for BPD focuses particularly on developing functional new core beliefs. The therapeutic relationship is seen as a vital means for exploring the patient's style of relating to others and for fostering more adaptive interactions in the future. More specifically, Arntz (1994) describes CBT for BPD as consisting of five stages: i) construction of a working relationship, ii) symptom-management, iii) correction of thinking errors, iv) emotional processing and cognitive re-evaluation of childhood trauma and schema changes, and v) termination. Davidson et al. (2006) conducted a large randomised controlled trial (RCT) of individual CBT for BPD verses treatment as usual (TAU) which found that CBT was roughly equivalent to TAU on outcomes of suicidal behaviour, presentation to A&E services and number of inpatient psychiatric days over the two year study period. However, CBT was found to be superior to TAU in reducing the number of suicidal acts and decreasing dysfunctional beliefs, state anxiety, and psychiatric symptom distress. Leichsenring and Leibing (2003) reported significant effects for more specific measures of PD pathology for CBT over that of psychodynamic therapy for PD.

Developed by Linehan (1993), DBT uses strategies from CBT to aid the regulation of emotions as well as teaching distress tolerance and using third wave approaches to promote awareness and acceptance. Linehan et al. (2006) concluded that DBT was superior in reducing suicide attempts in the treatment of BPD when compared to a community treatment that was specifically developed for the study

and delivered by experts. Brazier et al. (2006) conducted a systematic review summarising the available evidence on the clinical effectiveness of psychological therapies for BPD, concluding that DBT was equal to or superior to other treatments with regards to clinical effectiveness. A recent meta-analysis and systematic review by Panos et al. (2013) investigating the efficacy of DBT for BPD revealed a net benefit in favour of DBT when combining effect measures for suicide and parasuicidal (self-harm) behaviour. Regarding DBT in inpatients with BPD, Bloom et al. (2012) systematically reviewed 11 studies reporting pre- and post-treatment symptoms in the DBT treatment of BPD finding reductions in suicidal ideation, self-injurious behaviours, and symptoms of depression and anxiety in most studies. Importantly, follow-up data mostly revealed maintenance of symptom reduction between one and 21 months post-treatment.

Despite the plethora of empirical data documenting the efficacy of both CBT and DBT for BPD, most research to date has focused solely on outcome data with relatively few studies identifying the reasons why treatments are successful, and what might be the specific active processes or 'mechanisms of change' through which improvements occur. Clarkin and Levy (2006) highlight the difference between the vast number of outcome studies and the relatively few studies of mechanisms of change clarifying that, "the question of the mechanisms of change in psychotherapy seeks to learn how a particular therapy works, not what is the outcome of the treatment per se" (p. 405). Elliott (2010) refers to this research as 'change process research' describing it as, "a necessary complement to randomised clinical trials and other forms of efficacy research" (p. 123). Kazdin (2007) discusses the lack of evidenced explanation for why, in even the most rigorously researched psychotherapeutic interventions, researchers lack insight into the mechanisms through which these treatments result in successful outcomes. He advises that future investigations should strive towards this as the next step in psychotherapy research.

Identifying the specific mechanisms of change by which BPD patients improve through treatment has vast implications for the future of psychological therapy for BPD. Pre-assessment, this data could allow clinicians to predict which patients are more likely to do well from receiving CBT or DBT treatment and which patients may do better in receipt of alternative therapies.

This review therefore analyses the empirical literature to date, aiming to isolate and identify specific mechanisms of change in both the CBT and DBT treatment of BPD. In particular, the review aims to answer the question, *what are the specific mechanisms of change in the CBT and DBT treatment for BPD?*

## **Method**

Searches of paper titles, abstracts and full text content were performed in July and August 2012 and then repeated in February 2014 (see below), in the PsychInfo, CINAHL Plus, PubMed, MEDLINE and EMBASE databases. The search terms used were: a) “mechanism\* change borderline personality disorder”, b) “mechanism\* change” *and* “borderline personality disorder”, c) “mechanism\* change” *and* “BPD”, d) “mechanism\* change” *and* “borderline personality disorder” *and* “treatment” and e) “borderline personality disorder” *and* “therapeutic change.”

Studies included in the review involved i) participants who met diagnostic criteria for BPD, ii) who had received either CBT or DBT treatment for their BPD, iii) were either outpatients or partially hospitalised when they received their treatment (due to the limited number of manualised studies of inpatients with BPD), iv) were treated as part of full text peer-reviewed studies published in English since 1990 (as this was the earliest that the literature began to report CBT and DBT treatment of BPD), and v) were adults (aged over 18) at the time of their BPD treatment (as there

is a limited research presence investigating emerging BPD in adolescents). The review excluded: i) single case studies of BPD treatment.

An initial search of the CINAHL Plus and MEDLINE databases combined using search term a) with the limits 'January 1990 to present' yielded 8479 hits. The same search using search term b) yielded only six results. Re-running this search and additionally ticking the 'find all my search terms' box returned two additional results. All eight were added to a shortlist. Running the same search again with search term c) produced 13 hits, four of which were relevant but had already been returned in the previous two searches. It was clear from the titles of the remaining nine papers that they related to medical disorders not BPD. Search term d) also did not return any hits not previously revealed in earlier searches.

Searching the PubMed database with search term a) with no date limits revealed nine hits which were added to the shortlist. Search term b) revealed the same nine hits. Search term c) returned 14 hits; however, as previously, it was evident from their titles that ten of these related to specific medical conditions and not to BPD. The four relevant hits had already been revealed previously in searches using search terms a) and b). Search term d) did not return any hits not previously revealed in earlier searches.

Searching the PsychInfo database using search term a) with no limits returned 5684 hits. The search was therefore repeated with the addition of the following limits: 'full text only', 'peer-reviewed only', '1990-2012 only', 'English language only', 'adulthood only (aged 18 and up)'. This search returned only 19 hits. Three were added to the shortlist. The remaining 16 were not relevant as it was evident from their titles that they were purely pharmacological or medical studies. Search terms b) and c) did not reveal any additional results. Search term d) was conducted with the additional limits of 'human only' and 'outpatients only.' This search returned 30 hits, 11 of which were added to the shortlist. The 19 not added to the shortlist bore titles which related to disorders other than BPD or were clearly

medical, not psychological in nature. Running all four search terms in the EMBASE database yielded no results not already revealed in previous searches.

The fifth search term e) was added (“borderline personality disorder” *and* “therapeutic change”). Running this search term with no limits in CINHALL Plus, PubMed and PsychInfo did not reveal any results not previously achieved. Running this search term with no limits in the MEDLINE database revealed eight hits, five of which were relevant and added to the shortlist and three of which were excluded as irrelevant on the basis of their titles alone. The same search conducted in the EMBASE database with no limits revealed five hits, all of which were relevant but all of which had already been returned in the MEDLINE search and had therefore already been added to the shortlist.

Exclusion of non-relevant papers from a title screen only reduced 95 papers to 36 papers. Nine were removed as they were duplicates of papers already in the shortlist but not previously removed at earlier screening. Twenty seven papers remained and the full abstracts were reviewed for all 27. Following review of these abstracts, 14 papers were removed as they did not meet inclusion criteria. The most common two reasons for rejection at this stage were because papers either focused on treatments for BPD other than CBT or DBT or because they were literature reviews rather than empirical studies. This left 13 papers for which the full texts and reference lists were fully reviewed. No additional relevant studies not already included were found among reference lists. Of these 13 papers, three were excluded, one because it was comparing DBT across group and individual delivery and did not consider mechanisms of change in any detail, one because it became apparent that it was only concerned with MBT and one because it investigated only psychodynamic psychotherapy. A further two papers were initially thought to be appropriate for inclusion in the review but upon detailed review of the full texts were ultimately excluded as they could not provide any information on mechanisms of change in either CBT or DBT treatment of BPD. The first study (Yen, Johnson,



Costello & Simpson, 2009) set out to identify mechanisms of change in DBT for BPD among a sample of 50 patients but concluded that BPD is not homogenous and it is therefore impossible to identify any common mechanisms of change. A second study (Gratz, Lacroce & Gunderson, 2006) crucially failed to separate out the effects and potential benefits of several different treatment approaches, combining three different psychological therapies (DBT, CBT and psychodynamic psychotherapy) alongside both psychoeducation and psychiatric medication. This study additionally joined partial hospitalisation with intensive outpatient treatment, limiting the possibility for investigating mechanisms of change in outpatient CBT and DBT in isolation.

On review of one excluded paper (Barnicot et al., 2012; a systematic review of factors predicting outcome across all psychotherapeutic treatments for BPD), five further studies which empirically tested factors affecting treatment outcome were identified within the reference list. Of these five, four did not meet inclusion criteria for the current review, as factors considered were mainly pre-treatment static characteristics as opposed to dynamic mechanisms of change that could conceivably be altered during the course of therapeutic treatment. One further paper did meet inclusion criteria, however, taking the total number of papers to nine.

As a final check of the literature following a period of inactivity, in February 2014 identical searches were re-run in all five databases using search terms a)-e) and nine further references were revealed. With the exception of the PubMed database there were no changes to the initial search results. Searching term a) in the PubMed database revealed one additional paper which was then excluded following abstract screen as it reported results from an emotion regulation group, not CBT or DBT. Searching term e) in the PubMed database exposed a further relevant paper, ultimately excluded following a full text screen as it also did not investigate CBT or DBT. Seeking out the full text of this paper, however, revealed a special (2013) Personality Disorder edition of the journal *Psychotherapy Research*. After an

initial title screen of all seven published papers, five were found to be relevant. After screening full texts all but one were excluded for non-CBT or DBT treatment, or for not including patients with a diagnosis of BPD. On full review of the paper which initially appeared to meet inclusion criteria (McMain et al., 2013) it became clear that this study would be of limited use for the current review as although it focussed on changes in emotion processes in DBT for, and general psychiatric management of, BPD, a large proportion of data was missing limiting generalisability. Additionally, the number of participants in each treatment group was unbalanced which together with the relatively small sample size meant that power was not sufficient to test for differences between the two treatment types and therefore the mechanisms of change functioning in DBT could not be determined.

Following the 2014 search the total number of references screened rose to 104, with 34 abstracts and 19 full texts ultimately reviewed. The final total of papers included in the review remained at nine. See Figure 1 for flow chart illustrating the database search process.

## Results

Limiting the results as described yielded a total of 104 studies for review. After screening titles, abstracts and full texts, nine studies met criteria for inclusion in the final review. A list of these studies and their relevant features is provided in Table 1.

Each study is described in some detail under one of three broad categories of mechanism of change: *i) emotion regulation and self-control*, *ii) skills use* and *iii) therapeutic alliance and investment in treatment*. A critical evaluation of each study's methodology and findings are included, measured against a well-known critical appraisal checklist (Downs & Black, 1998, see Appendix A) which assesses the

Figure 1: *Flow chart of review database*

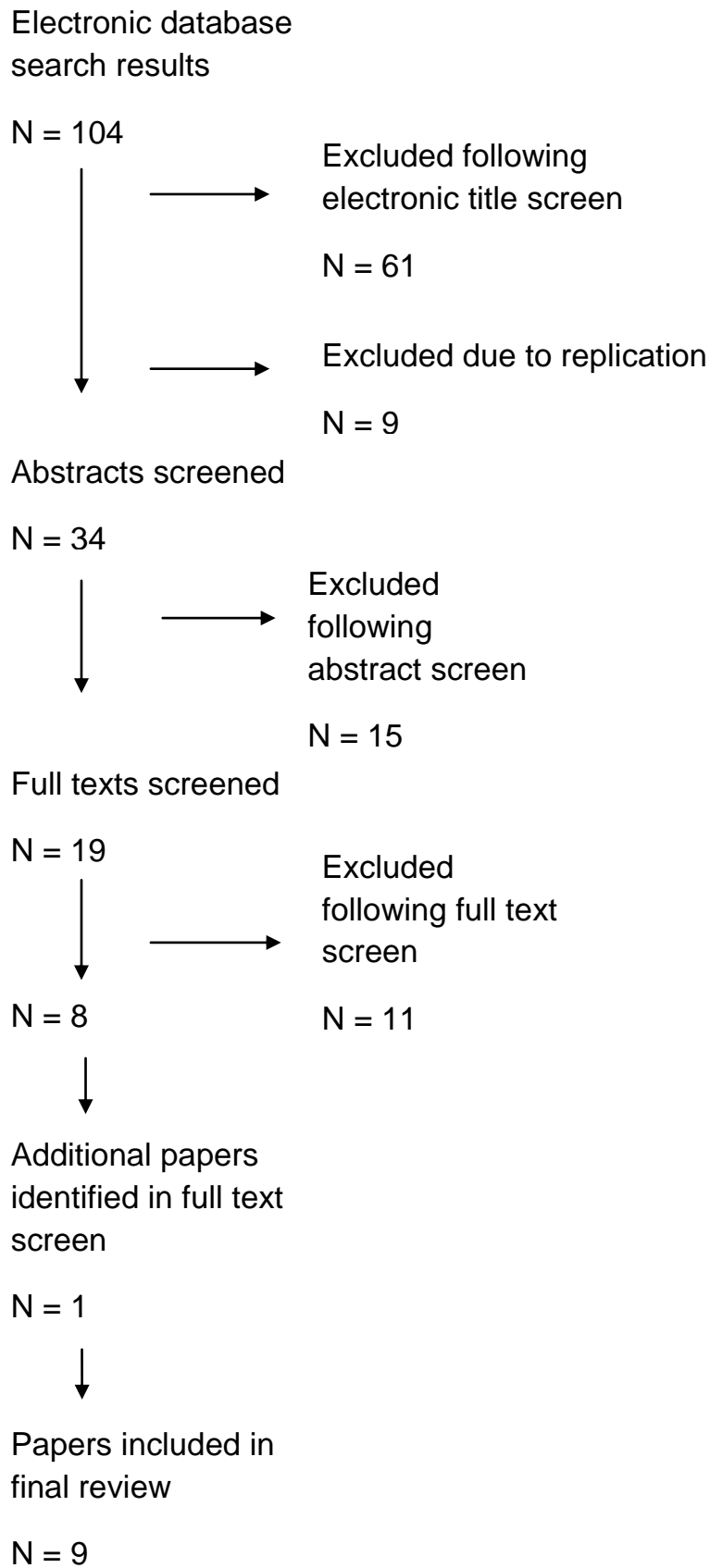


Table 1  
*Papers included in the review*

Paper	Primary therapeutic orientation	Control group	Sample size	Sample demographics	Mechanism(s) of change	Main findings
Axelrod et al. (2011)	DBT	No	27	Women with BPD and substance dependence	Improvements in emotion regulation	Improved emotion regulation can account for increased behavioural control
Bedics et al. (2012)	DBT	Yes	101	Women with BPD	Personality factors and intrapsychic change	DBT patients reported greater self-affirmation, protection, love and less self-attack
Davenport et al. (2010)	DBT	Yes	17	Men and women with primary diagnosis of BPD	Personality and self-control	Pre-treatment participants lower on self-control, conscientiousness and agreeableness than post-treatment participants
Gibbons et al. (2009)	CBT	Yes	34	Men and women with primary diagnosis of BPD	Self-understanding and compensatory skills	Change in compensatory skills observed in BPD group
Lenzenwenger et al. (2012)	DBT	Yes	58	Men and women meeting criteria for BPD, aged 18-50	Anger and aggression, global functioning and social adjustment, affective dyscontrol	No significant difference in changes on proposed mechanisms of change between treatment types.
Neacsiu et al. (2010)	DBT	Yes	108	63 recurrently suicidal women with BPD/45 women with BPD	Increasing use of DBT skills	DBT skills use mediated decrease in suicide attempts and depression
Perroud et al. (2012)	DBT	No	52	Men and women with suicidal /para-suicidal behaviour and BPD	Mindfulness and acceptance	Increases in skill of accepting without judgement correlated with improvement in BPD symptoms

Turner (2000)	DBT	Yes	24	Men and women with BPD	Quality of therapeutic alliance	DBT group improved more than controls on most outcomes
Wenzel et al. (2006)	CBT	No	27	Men and women with BPD	Belief change, reduction in hopelessness, improvement in attitude towards treatment	Positivity to treatment correlated with improvement in BPD diagnostic criteria

---

methodological quality of both randomised and non-randomised studies of healthcare interventions. This 27 item checklist assigns a numerical score out of a maximum of 32. Due to difficulty in ascertaining reliable scores for the final item concerning power analyses (which awards up to five points), this item has instead been scored either 'zero' (no power calculation completed or power not met) or 'one' (power calculation completed, and met). Therefore, a maximum score of 28 is possible (item five only is worth up to two points). A summary of each study's performance against the checklist can be found in Table 2 (DBT) or Table 3 (CBT).

Table 2:  
*Checklist appraisal of DBT studies according to Downs & Black (1998)*

Paper	Strong points according to checklist	Weak points according to checklist	Total score (/28)
Axelrod et al. (2011)	Outcome measures, reporting, sampling	Attrition (44.4% did not complete treatment), non-randomisation, lacked control	16
Bedics et al. (2012)	Randomisation	Unable to determine treatment compliance	21
Davenport et al. (2010)	Control comparison group	Non-randomisation of participants to control/treatment condition	16
Lenzenwenger et al. (2012)	Randomisation	Non-blinding of participants	20
Neacsiu et al. (2010)	Randomisation, control group, blind assessors	Non-blinding of participants	26
Perroud et al. (2012)	Outcome measures, sampling	Lack of control group	19
Turner (2000)	Randomisation, blind independent assessors	Lack of information about non-completers	21

Table 3  
*Checklist appraisal of CBT studies according to Downs & Black (1998)*

Paper	Strong points according to checklist	Weak points according to checklist	Total score (/28)
Gibbons et al. (2009)	Randomisation, large sample, several comparison groups	Non-blinding of participants	20
Wenzel et al. (2006)	Management of data of participants lost to follow up	Lack of control group	19

## **Emotion regulation and self-control**

Axelrod, Perepletchikova, Holtzman and Sinha (2011) concentrated their study on the DBT principle that dysregulation of emotions is central to the dangerous impulsivity associated with BPD and is hence targeted as a primary mechanism of change in DBT. The researchers posited that an improvement in the ability to regulate emotions would lead to a decline in impulsivity, and this would be evidenced by a decrease in substance misuse, as having greater control of emotions thus being less impulsive should lead to a reduced need to regulate mood/self-medicate by using substances. This study investigated the above in a sample of 27 females with substance dependence and BPD who were receiving a 20 week course of DBT in an outpatient service. The researchers assessed emotion regulation using the Difficulties in Emotion Regulation Scale (Gratz & Roemer, 2004) and recorded substance use for 30 days preceding treatment and for the last 30 days of treatment, corroborated by weekly patient self-report, clinician assessment, urine toxicology and alcohol breathalyser tests. The study concluded that improvements in emotion regulation explained the variance in decreased substance use frequency. One way repeated measures ANOVAs indicated a significant interaction between frequency of substance use and emotion regulation ( $F(1, 21) = 8.202, p = 0.009$ ) and changes in substance use lost their significance when improvement in emotion regulation was controlled for ( $F(1, 21) = -0.112, p = ns$ ). This demonstrates that improved emotion regulation in BPD patients treated with DBT can account for increased behavioural control using substance use as the outcome measure of behavioural control.

While this is an interesting DBT study which has collected useful data about the relationship between increased emotion regulation skills and substance use, it has several limitations. Firstly, the authors use the term 'behavioural control' as their primary outcome measure but this is only measured by substance use. There are other aspects of behavioural control (impulsivity) relevant to the diagnosis of BPD

that could also have been measured to add more weight to the argument that increased emotion regulation skills gained via receipt of DBT act as a mechanism of change for improved behavioural control. Some obvious examples are evident in the DSM-5 criteria for BPD (American Psychiatric Association, 2013) under the section considering impulsivity which asks about impulsive sexual behaviour, reckless driving, uncontrollable eating and unnecessary overspending. Additionally, deliberate self-harm or suicidal ideation could have been measured to assess dangerous impulsive behaviour. The authors concede that the study is limited by its lack of a controlled treatment condition which of course impedes the possibility of attributing improvements observed in emotion regulation purely to DBT treatment. Further, most prior studies of DBT have provided a longer treatment period than that delivered by Axelrod et al. (2011). It is therefore unclear as to whether their study represented a full and comparable treatment 'dose' of DBT. A sample of just 27 participants would be regarded as relatively small in the experimental literature and to ensure findings were more generalisable and statistically robust, a larger sample would be preferable.

Similarly, the study's all-female sample does not facilitate conclusions about emotion regulation in males receiving DBT treatment for BPD, although it is perhaps justified (and samples in other studies are also likely to be female-heavy) due to the larger number of treatment-seeking females diagnosed with BPD than males and the fact that current NICE (2009) guidance for BPD recommends the use of DBT treatment for females only. The female to male ratio for BPD diagnosis has most recently been estimated at 3:1 (American Psychiatric Association, 2000), however, this disparity has recently been called into question with suggestions that the gender balance may actually be more equally distributed (see, for example, Sansone & Sansone, 2011). The weaknesses in Axelrod et al.'s (2011) study design are reflected by their score of only 16/28 on Downs and Black's (1998) checklist, the joint lowest score and below the average of 18.5.



Using data from the Cornell Personality Disorders Institute RCT (Clarkin, Levy, Lenzenwenger & Kernberg, 2004), Lenzenwenger, Clarkin, Levy, Yeomans and Kernberg (2012) set out to investigate the changes which occur in therapeutic treatment of BPD. A sample of 58 predominantly female participants with BPD were randomly allocated to receive either DBT, Transference-Focused Psychotherapy (TFP) or a third condition, Supportive Treatment (SPT) which was intended to control for attention and support received. As well as regularly completing self-report measures, each participant was evaluated by an experienced clinician at baseline and subsequent three month intervals to assess change. The researchers discovered that participants showed change over time in several dimensions simultaneously and noted that these dimensions could be clustered into similar domains representing broader categories of change in functional/psychological features mostly relating to emotional and behavioural control. The three domains of change identified were aggressive dyscontrol, social adjustment/self-acceptance and conflict tolerance/behavioural control. Positive improvements were seen in all three areas following treatment with DBT, TFP or SPT suggesting that different areas of impaired functioning will change at different rates and that variables can be clustered into sets which will largely change simultaneously with other similar variables. In order to test whether treatment type was related to the three factors, the researchers conducted one-way ANOVAs on the three factor scores. These tests did not reveal any significant differences between treatment type (DBT, TFP and SPT: multivariate one-way ANOVA:  $p > 0.60$ ; all univariate one-way ANOVAs:  $p \geq 0.40$ ), suggesting that the same change domains were produced regardless of treatment condition.

Lenzenwenger et al. (2012) do however concede that their sample size of 58 is relatively small for their factor analytic methodology and that had they followed patients up over a longer time period than one year, different predictors of change may have been revealed. Nevertheless, reflected in its score of 20/28 on Down's

and Black's (1998) checklist, this is a solid study based on robust RCT data which corroborates previous studies' evidence for emotion regulation and behavioural self-control as central mechanisms of change in the treatment of BPD. It is important to emphasise, however, that these mechanisms of change were not found to be exclusive to the DBT treatment of BPD but occurred simultaneously in TFP and SPT also.

Davenport, Bore and Campbell (2010) set out to investigate changes in self-control in 17 (again, predominantly female) BPD patients. Participants were divided into two groups: a control group of individuals who were either on a waiting list for therapy, or who had started, but not completed, their first eight week skill-building module and a second group who had successfully graduated from a DBT program in the past three years. In this between-subjects design, two self-report measures of self-control were mailed to participants and status in treatment was used to allocate participants to either the control (pre-treatment) or treatment (post-DBT treatment) group. Data supported the researchers' hypothesis that pre-treatment participants would be significantly under-controlled in measures of self-control when compared to post-treatment participants and that post-treatment participants would be more conscientious and agreeable, when compared to pre-treatment participants. Overall, the research revealed significant personality differences between pre- and post-treatment groups. Participants who had not yet received DBT had lower self-control, were less agreeable and less conscientious compared to the post-treatment group. Participants who had received DBT were just as self-controlled, agreeable and conscientious as the data used for normative comparisons. The researchers concluded that DBT appeared superior in aiding participants' development of self-control, suggestive of a more ordered personality.

As with all between-subject designs, however, Davenport et al.'s (2010) study does not prevent the possibility of differences between the two groups accounting for the change, rather than the pre- and post-treatment variables.

Randomisation was not used, and as with some of the previous studies, the sample size was small, limiting the generalisability and reliability of the data. Additionally, reliance on self-report methods to gain a measure of self-control opens up the possibility of response bias. This could have perhaps been corroborated using a behavioural measure and/or clinician report. These methodological oversights mean that Davenport et al.'s (2010) study achieved the lowest score of 16/28 on Down's and Black's (1998) checklist.

Despite their limitations, the three studies summarised all provide empirical evidence for improvements in emotion regulation and behavioural self-control with DBT treatment. Changes within these domains are likely to produce more positive results in several outcomes of BPD symptomology.

### **Skills use**

A key aspect of DBT is the teaching of specific behavioural skills with the aim of helping individuals to replace maladaptive behaviours with more adaptive responses (Linehan, 1993). However, Neacsiu, Rizvi and Linehan (2010) noted that no study to date had directly tested this mechanism of change and they therefore set out to investigate the improvement in skills on outcomes of BPD treatment. The study consisted of a female-only sample of 108 patients with BPD participating in a one year RCT with a four month follow-up period. Participants included 63 recurrently suicidal women and 45 women with drug dependence but the researchers note that there were no significant differences in demographic characteristics between the suicidal group and the drug dependent group. Participants either received DBT or one of three control conditions: Community Treatment by Experts (CTBE), Treatment As Usual (TAU) or Comprehensive Validation Therapy (CVT), in conjunction with a 12-step program. Measures of DBT skills use, anger, suicidal/self-injurious behaviour and depression were gathered using a combination of self-report and semi-structured interviews. The researchers

used a hierarchical linear modelling approach to analyse their data. Although anger suppression and expression was not found to mediate outcome, significant mediation effects did indicate that the use of DBT skills fully mediated decreases in suicide attempts and depression symptom severity and an increase in the control of anger over time. The use of DBT skills also partially mediated the decrease of non-suicidal self-injury over time. Participants who received DBT reported using three times more skills by the end of their treatment (mean DBT skills use increased by 15.3%), as compared to participants in receipt in one of the control conditions (mean skills use increased by only 4.6%). At follow up, DBT participants maintained their increased skill use but control participants had decreased their skill use by 5%.

Although this data supports a DBT skills deficit model of BPD by demonstrating via robust methodology that improved skills use is a mechanism of change for suicidal behaviour, depression, and anger control, Neasciu et al.'s (2010) study is limited by its primary reliance on self-report as a measure of skills use. Some individuals may have over or underestimated their proficiency in using DBT skills. Assessing skills use on a daily basis using a more objective measure would increase the reliability of the findings. When using a standard mediation analysis, an assumption is made that there are no confounds manipulating the mediator and outcome (Robins & Rotnitzky, 2005) and it is possible that uncontrolled extraneous variables influenced the mediational analysis in this study such that an increase in DBT skills use was not the only variable influencing positive outcomes in suicidal behaviour, depression and anger control. Nevertheless, methodologically, this remains a robust study, reflected by the highest score awarded by Downs and Black's (1998) checklist of 26/28.

Gibbons et al. (2009) set out to examine what they considered to be theoretically important mechanisms of change in outcomes between psychodynamic and cognitive-based therapy. They were specifically interested in the acquisition of compensatory skills and self-understanding and perception. Thirty four patients with

a primary diagnosis of BPD received one year of a version of cognitive therapy tailored to BPD, Schema-Focused Cognitive Therapy. Although this was not described by the researchers as CBT per se, it is a form of cognitive therapy for BPD which is very similar to CBT, and quite different to DBT. No BPD patients received any type of dynamic therapy; this was reserved for patients with depression or anxiety in another arm of the research trial. Outcomes were measured using well-known, validated self-report measures of depression, anxiety and quality of life. Self-understanding as a mechanism of change was measured using two self-report questionnaires and acquisition of compensatory skills was measured using the Ways of Responding Questionnaire (WOR; Barber & DeRubeis, 1992) which presents eight different stressful scenarios to participants along with an initial negative automatic thought. Participants are required to state their feelings, thoughts and possible reactions to each scenario which is then rated by independent judges for the presence of a list of possible positive and negative compensatory skills. High inter-rater reliability and internal consistency of items was recorded by the researchers. The data for the effects of cognitive therapy for BPD showed no overall change in self-understanding across treatment (all  $p$  values  $< 0.1$ ). However, the researchers did conclude that change in compensatory skills was apparent in the BPD group and that in particular, a decrease in negative compensatory responses/negative thinking co-occurred with symptom improvement. This large scale, robust clinical trial therefore demonstrated that the attainment of new skills acquired through cognitive therapy acts as a mechanism of change in BPD, improving the symptoms of depression and anxiety.

As Gibbons et al.'s (2009) study was a generic trial which also included participants with a primary diagnosis of depression or anxiety, the outcome measures were perhaps too broad to capture some of the additional symptoms experienced by people with BPD. This could be improved by the inclusion of additional outcome measures testing concepts key to the diagnosis of BPD such as

impulsivity, anger, self-harm and interpersonal function. Like other studies included in this review, the present study relied heavily on the use of self-report measures which are open to biased responding, however the WOR (Barber & DeRubeis, 1992) was rated by clinicians and found to have high inter-rater reliability, adding to the reliability of the measure of skills acquisition. The researchers concede that the relatively small within-study sample sizes and their associated limitations on statistical power for testing interactions meant that the use of a pooled database was not the best way to investigate mechanisms of change in specific treatments for specific diagnostic categories (in this case, cognitive therapy for BPD). This study nevertheless achieved a fairly high score of 20/28 on Downs and Black's (1998) checklist.

Perroud, Nicastrò, Jermann & Huguelet (2012) investigated improved skills in mindfulness, a key component of DBT treatment for BPD. They examined changes in and correlates of mindfulness skills over a one year follow-up period including a four week dose of intensive DBT followed by ten months of standard DBT. The researchers studied 52 participants (90% females) with a BPD diagnosis and administered the Kentucky Inventory of Mindfulness Skills (KIMS; Baer, Smith & Allen, 2004) which describes mindfulness in four discrete dimensions: observing, describing, acting with awareness and accepting without judgment. Standard self-report measures of depression and hopelessness were also administered at regular time intervals, as were standardised diagnostic clinician-administered assessments of BPD psychopathology. Results showed that DBT was associated with an increase in mindfulness skills over time and that of the four dimensions of mindfulness, accepting without judgement was the only dimension found to significantly increase over time following statistical adjustment for potential confounds. Increases in accepting without judgement additionally correlated with improvement in BPD symptoms, suggesting that it is this specific mindfulness skill that acts as a mechanism of change reducing BPD symptomatology.

As before, Perroud et al.'s (2012) reliance on self-report as a measure of mindfulness opens the data up to the possibility of response bias and does not provide an accurate, objective measure of this skill, although this is perhaps something inherently difficult to measure objectively. Crucially, this study lacked a control group, limiting the possibility of drawing conclusions about whether observed improvements are exclusive to the acquisition of the accepting without judgement skill or whether they are partially or otherwise explained by a natural change in mindfulness skills and/or correlate with an uncontrolled confound. As only one of the mindfulness dimensions was found to increase significantly, the researchers could not conclude that mindfulness skills per se function as a mechanism of change in DBT for BPD symptomatology, rather that the specific skill of accepting without judgement becomes enhanced via DBT and may therefore function alone as a mechanism of change. These limitations therefore meant that this study achieved only a slightly above average score of 19/28 on Downs and Black's (1998) checklist.

The studies summarised above do in the main demonstrate that increased use of skills gained through either DBT (Neasciu et al., 2010; Perroud et al., 2012) or cognitive therapy (Gibbons et al., 2009) lead to favourable outcomes on self-harm/suicidality, anger, depression, anxiety and standardised BPD symptomatology, providing compelling evidence for the deployment of skills acquired through therapeutic techniques as a mechanism of change in the treatment of BPD.

### **Therapeutic alliance and investment in treatment**

Across a range of psychotherapies the therapeutic alliance is considered a helpful factor in retaining patients in therapy as well as contributing to positive outcomes (Horvarth & Luborsky, 1993). However, it remains a difficult concept to quantify and could easily be conflated with other mechanisms of change such that its role as an independent factor in its own right becomes less clear. The most sensible definition for considering therapeutic alliance as a mechanism of change

might be to measure a change in the alliance over time to show that as it develops (and hopefully improves) so BPD symptoms reduce - a positive, measureable outcome.

Bedics, Atkins, Comtois and Linehan (2012) aimed to explore the therapeutic alliance as a mechanism of change in DBT for BPD. One hundred and one females were randomised to receive either DBT or a control condition, Community Treatment Delivered by Experts (CTBE). CTBE treatment was uncontrolled by the researchers, however, they note that, "selected therapists described their theoretical orientation as "eclectic" or "mostly psychodynamic" (i.e., there were no cognitive behavioural therapists in the CTBE condition)" (p.68). As well as meeting criteria for BPD, all participants included in the study had had a history of self-harm defined by at least two suicide attempts or non-suicidal self-injury in the past five years and at least one incident in the past eight weeks prior to commencement of the study. The quality of the therapeutic alliance was rated by patients using the Structural Analysis of Social Behaviour (SASB; Benjamin, 1974). Results showed that in comparison to CTBE participants, DBT participants reported their therapists as increasingly more affirming, protecting, and controlling during treatment, supporting the researchers' hypothesis. Additionally, DBT participants reported a stronger association between increased therapist affirmation and protection with decreased non-suicidal self-harm, showing that positive developments in the therapeutic alliance correlate with the positive and desired outcome of BPD symptom reduction (self-harm in this case).

Despite the strength of the RCT data, the reasonable sample size and the use of multiple time points for the assessment of symptomatic change and the therapeutic relationship, Bedics et al.'s (2012) study is not without limitations. Assessment of BPD symptoms was limited to self-harm and the researchers note the value that further research could add in extending these results to other domains relevant to BPD such as interpersonal functionality and emotion regulation. Additionally, the reliability of the data is limited, being taken only from participants'



perspective. Tighter control could be achieved by utilising therapists' assessment of the therapeutic relationship, as well as that of an impartial observer, blind to the treatment condition to which each participant was randomised. Nevertheless, this study provides valuable, current data regarding the effect of the therapeutic relationship as a mechanism of change in DBT for BPD and methodologically scored above average on Downs and Black's (1998) checklist (21/28).

Turner (2000) tested the effects of DBT versus a Client-Centred Treatment control condition (CCT) in a naturalistic evaluation of 24 primarily female patients with a diagnosis of BPD. In order to understand its role in the differences in outcomes (depression, anxiety, anger, self-harm/suicidality, required hospitalisation days) between the two therapies, the quality of the therapeutic alliance was measured using the Helping Relationship Questionnaire (HRQ; Luborsky, 1984). Participants were randomly assigned to receive either DBT or CCT and outcomes were evaluated using a combination of self-report and a rating assessor who was blind to each participant's treatment condition. Patients receiving DBT therapy showed greater improvement than patients receiving CCT regarding suicide and self-harm, depression, anger, and a decrease in the number of admissions to psychiatric hospitals. Importantly, the quality of the therapeutic alliance was found to account for significant variance in patients' outcomes across both DBT and CCT but no significant difference in therapeutic alliance was observed between the two treatments. This suggests that the alliance accounted for as much variance in symptom improvement as did the differences in the treatment conditions themselves.

Turner's (2000) study however, rated the quality of the alliance at one single time point rather than measuring a change (improvement) in alliance over time, making it harder to infer its role as a mechanism of change linked explicitly to the outcome of improved symptoms. Like other studies reviewed, this study relied heavily on self-report measures and used a relatively small sample size. However,

randomisation and use of a control group add to its reliability and validity, meaning that it scored fairly highly on Downs and Black's (1998) checklist (21/28).

Wenzel, Chapman, Newman, Beck & Brown (2006) proposed that change in dysfunctional beliefs, reduction in hopelessness, and improvement in attitude toward treatment all function as mechanisms of change associated with CBT. The researchers used data from their open cognitive therapy trial (Brown, Newman, Charlesworth, Crits-Christoph & Beck, 2004) which consisted of a primarily female sample of 32 patients diagnosed with BPD. Clinical evaluations were conducted at baseline, six months and 12 months then again six months after treatment was terminated at 18 months. Baseline assessments involved clinician-administered interviews, self-report questionnaires and review of previous treatment records in order to ascertain participants' diagnoses, suicide risk, psychiatric history and current physical, psychological, and social adaptation. Attitude towards treatment was measured using the Attitudes and Expectations Questionnaire (ERQ) which was adapted from Elkin et al. (1989). Results showed that 66.7% of the patients who had positive attitudes toward treatment no longer met criteria for BPD after 12 months of treatment, as compared to only 14.3% of the patients who had a negative attitude toward treatment, suggesting that a positive view of therapy may be one of the factors influencing a reduction in BPD symptomology. However, this may be a spurious link; it is not clarified how changes in attitude towards treatment specifically influenced outcome and without the benefit of data obtained at more than one time point in order to measure how a change in attitude associates with a reduction in symptomology, it is perhaps not reliably classed as a mechanism of change in the CBT treatment of BPD. The researchers additionally investigated other factors which they hypothesised to be functioning as mechanisms of change in the CBT treatment of BPD concluding, in support of their hypotheses, that a reduction in hopelessness was associated with significant reductions in borderline beliefs between baseline and termination. However, this conclusion may not be shedding much light on the

specific processes by which change occurs, as both belief change and reduction in hopelessness might be more reliably conceived as outcomes rather than mechanisms of change.

Wenzel et al.'s (2006) relatively small sample size precludes the possibility of making generalisable inferences about the mechanisms of change in CBT for BPD to wider samples and the standard critique of self-report measures also applies, although this study sought to use clinician-administered assessments and reviews to counteract self-report. According to Downs and Black's (1998) checklist, this study obtained an average score of 19/28.

Together, these three studies provide some evidence that a strong therapeutic alliance and a positive attitude towards and investment in treatment are in some way associated with change in BPD symptomatology in both DBT (Bedics et al., 2012; Turner, 2000) and CBT (Wenzel et al., 2006) although the exact mechanisms of change at work are somewhat unclear.

## **Discussion**

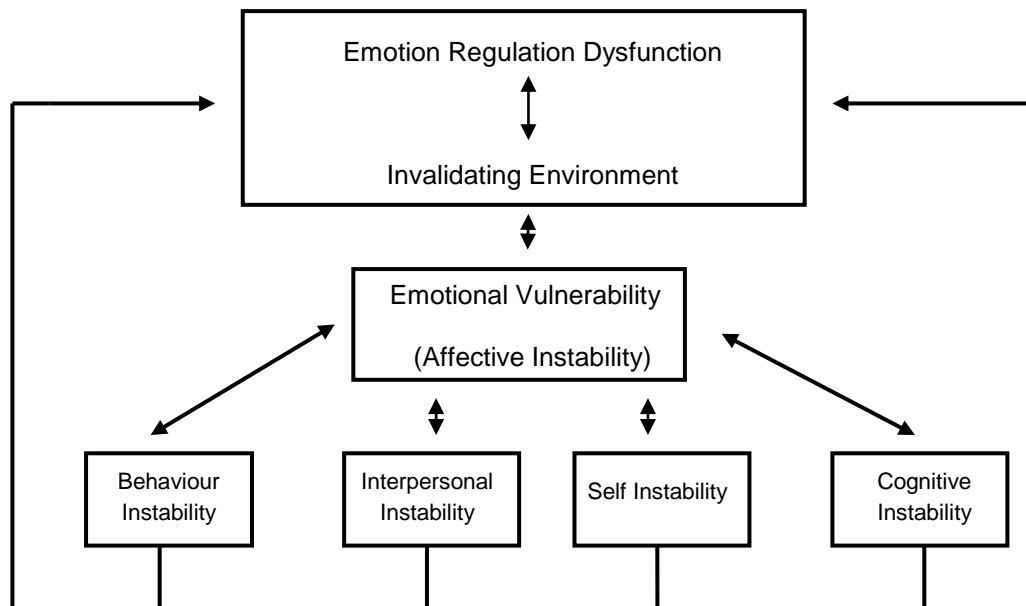
Despite a continuously expanding evidence base demonstrating the clinical effectiveness of both DBT and CBT in treating BPD pathology over the past twenty years, very little is known about the mechanisms of action by which these documented positive changes take place. This review sought to evaluate the relatively few empirical studies available that have investigated the processes by which therapeutic change occur in these therapeutic treatments for BPD.

The nine studies reviewed could be broadly classified into three categories of change: i) *emotion regulation and self-control* (Axelrod et al., 2011; Davenport et al., 2010; Lenzenwenger et al., 2012), ii) *skills use* (Gibbons et al., 2009; Neasciu et al., 2010; Perroud et al., 2012) and iii) *therapeutic alliance and investment in treatment* (Bedics et al., 2010; Turner, 2000; Wenzel et al., 2006).

The first category was primarily concerned with how individuals benefit from a therapeutic approach geared towards helping them to recognise, understand and better regulate their emotions and moods, and to exercise self-control with regard to impulsive behaviours. The three studies that primarily investigated mechanisms of change relevant to these domains were solely concerned with DBT; there was no CBT study investigating emotion regulation improvements in BPD. Although all three studies produced solid empirical evidence to support increased control and regulation of emotion and/or behaviour leading to favourable outcomes, Lenzenwenger et al. (2012) concluded that this mechanism of change was not unique to DBT – it was the same mechanism also identified in both SPT and TFP treatments for BPD. However, this category’s overriding finding of improved emotion regulation following DBT treatment is perhaps unsurprising given that Linehan’s DBT biosocial theory of emotion dysregulation views BPD as a disorder of persistent emotional dysfunction occurring largely due to deficits in the ability to regulate difficult emotions and because of emotional instability and vulnerability (Linehan, 1993, see Figure 2, below).

In their paper discussing the DBT treatment of emotion dysregulation in BPD, McMain, Korman and Dimeff (2001) agree that, “the primary goal in the first stage of DBT is to treat out-of-control behaviours that threaten the individual’s life, treatment, and quality of life” (p. 195). They go on to discuss the techniques that DBT therapists employ in order to help their patients achieve better regulated emotions including exposure-based procedures, validation, and the enhancement of capacities such as diverting attention away from cues associated with negative emotions and beginning to observe, describe, and understand the function of their emotions. Accordingly, it is promising that all studies reviewed found evidence of increased emotional and/or behavioural self-control as primary mechanisms of therapeutic change, suggesting that this is a vital and necessary process in the successful DBT treatment of BPD.

Figure 2: *The relationship between emotion dysregulation and borderline behaviour patterns according to Linehan's (1993) biosocial theory*



In reviewing the evidence for skills use as a mechanism of change in the therapeutic treatment of BPD, results were also encouraging. Both Neacsiu et al. (2010) and Perroud et al. (2012) studied the acquisition of skills in DBT treatment for BPD and found that increasing DBT skills use partially mediated the decrease of non-suicidal self-injury over time (Neacsiu et al., 2010) and that an increase in use of the mindfulness skill of accepting without judgement correlated with improvement in BPD symptoms (Perroud et al., 2012). Neacsiu et al. (2010) were able to make stronger inferences about the use of DBT skills in their BPD sample because of the use of a control group who did not report the same high levels of skills use as the DBT group. Both studies support the skills deficit theory of BPD that underlies DBT (Linehan, 1993). Accordingly, in testing out the skills deficit model, Lindenboim, Comtois & Linehan (2007) studied 49 women to ascertain whether practice of behavioural skills taught in the group skills training component of their DBT program was partly responsible for positive treatment outcomes. In accordance with Perroud et al. (2012) they found that mindfulness skills and additionally, crisis survival skills,

were practiced most frequently by participants and that the majority of participants practiced their DBT-learned skills on most of their treatment days.

Regarding the other treatment of interest, Gibbons et al. (2009) studied skill acquisition in cognitive therapy for BPD concluding specifically that compensatory skills used to achieve a reduction in negative cognitions correlated with BPD symptom improvement, which is perhaps not surprising as a reduction in negative thinking is a primary goal of CBT treatment. Moreover, Arntz (1994) lists 'correction of thinking errors' as one of five main components to be addressed in CBT for BPD.

The final category concerned with therapeutic alliance and patients' investment in their treatment proved problematic in terms of identifying specific processes which could reliably be classed as mechanisms of change. Wenzel et al. (2006) found that a positive attitude towards treatment was associated with a reduction in BPD symptomatology although it was unclear how much this factor alone was responsible for patients no longer meeting criteria for a BPD diagnosis following treatment. A more reliable finding might have been possible if attitude towards treatment had been measured at more than one time point, allowing for the effects of time on change to be incorporated into analyses. Bedics et al. (2010) and Turner (2000) both studied the effects of therapeutic alliance in DBT treatment for BPD, producing contradicting evidence on the importance of the alliance as a positive change process. Although both studies concluded that a more positively-perceived therapeutic alliance led to improved outcomes for patients, when comparing the alliance in DBT to their control condition (CTBE), Bedics et al. (2012) found that the alliance was reported more favourably in DBT than CTBE whereas Turner (2000) found no significant difference between patient-reported therapeutic alliance between DBT and his control condition (CCT). Both studies reported a correlation between the therapeutic alliance with improved BPD symptoms and importantly Bedics et al. (2012) found that this was true for the most concerning of BPD symptoms, self-injurious behaviour. This fits with Linehan's (1993) model as

instead of the invalidating environment that BPD patients are accustomed to, therapists provide warm and emotionally-validating environments which foster increased emotion regulation skills (as well as other increased skills use, described above) and decreased instability and impulsivity (manifesting in behaviours such as deliberate self-harm). Importantly, this conclusion regarding the necessity of the therapeutic alliance is in agreement with a recent review of factors predicting outcome in BPD treatment which listed a stronger therapeutic alliance as the main factor predicting therapy outcome for patients with BPD (Barnicot et al., 2012) and is particularly promising given the difficulty BPD patients have with interpersonal relationships which one would naturally assume would extend to the therapeutic relationship. However, as discussed above, a more convincing measure of therapeutic alliance as a mechanism of change would be to measure change in therapeutic alliance and attitude towards treatment longitudinally rather than cross-sectionally to attempt to link that change with reduction in BPD symptomology.

Martin, Garske and Davis (2000) conducted a large meta-analysis of studies measuring alliance and concluded that the overall relationship between the alliance and outcome is moderate but consistent regardless of any hypothesised confounds. They noted that the large and varied range of measures available all had adequate reliability regardless of the method used (independent rater, self-report or therapist report). Given that they found a large diversity in alliance measures available, research on the alliance may not always be reliable and easily replicable, perhaps partially accounting for why the papers reviewed here struggled to isolate and quantify the alliance as a reliable mechanism of change, and why there was no agreement regarding the superiority of the alliance in the two studies primarily investigating DBT.

To conclude, this review of the mechanisms of change in both DBT and CBT treatment for BPD is a start in a long journey towards being able to confirm the mechanisms which effect change in these two treatments for this complex and

challenging disorder. Three broad categories of mechanism of change were identified which are well-explained by Linehan's (1993) DBT biosocial model of BPD: initial deficits in *emotion regulation and self-control* are improved via the *therapeutic alliance and investment in treatment* which result in increased *skills use* leading to favourable outcomes on general clinical syndromes (depression, anxiety) and on measures of BPD symptomology such as self-harm, impulsivity, substance misuse and borderline beliefs. In this review the DBT model more aptly explains the mechanisms of change identified herewith than a CBT model, due to the aforementioned weighting in favour of DBT in the studies analysed, although with regards to the emotion regulation and therapeutic alliance categories, two studies found that these mechanisms identified were not unique to DBT, and held for control therapies too – SPT and TFP (Lenzenwenger et al., 2012) and CCT (Turner, 2000).

In terms of limitations, this review was slightly diverted from its initial equal interest in both the CBT and DBT treatment of BPD because the studies included were heavily balanced in favour of DBT, likely due to its prominence in the most current clinical guideline for BPD (NICE, 2009). This does, however, suggest that further research into the mechanisms effecting change in the cognitive behavioural treatment of BPD is warranted, especially as NICE (2009) advises that should this data be produced, future revisions may recommend CBT for BPD. The empirical data reviewed has highlighted three categories of therapeutic change (emotional and behavioural regulation and control, increasing skills use and therapeutic alliance/investment in treatment) which were observed by more than one set of researchers. It has, however, also highlighted the difficulty in demonstrating causality, much of the evidence relying on associative relationships.

Additionally, the majority of studies reviewed have revealed difficulties in obtaining large enough sample sizes and in establishing satisfactory scientific rigour from which to base their conclusions, evidenced by some relatively low scores on Downs and Black's (1998) checklist. Further robust research and hypothesis testing



will help to corroborate the mechanisms identified in this review, concentrating future efforts on the most important and research-worthy processes of therapeutic change in DBT and CBT identified in this review as improved emotion regulation and increased skills use. This review has identified some agreement in the mechanisms of change in the DBT treatment of BPD but more studies investigating mechanisms of change in CBT for BPD would be useful, as would information on both SFT and MBT, given their likely inclusion in future NICE guidance for BPD treatment. Efforts to reliably prove the therapeutic alliance as a mechanism of change in its own right would additionally be beneficial, given that we know the therapeutic relationship is a factor influencing treatment outcome regardless of other variables which may influence this relationship (Martin et al., 2000). More consistent data documenting the role of therapeutic alliance in CBT for BPD in particular would be welcomed as Arntz (1994) posits that a primary tenet of CBT is to use the therapeutic relationship as an important path towards fostering more adaptive future interpersonal interactions, suggesting it should act as a useful change process in the CBT treatment of BPD.

Clinically, further mechanism of change data will assist practitioners in focusing on those aspects of treatment that are most likely to lead to positive outcomes, particularly benefitting those for whom only brief treatments are available. Therapists would be able to focus their efforts on the techniques and aspects of DBT and CBT which have been reliably proven to function as therapeutic change mechanisms such as validating and naming difficult emotions in an effort to enhance emotional regulation and self-control, and teaching and promoting new skills through which to achieve this. Should the data on therapeutic alliance as a reliable mechanism of change be produced, further research could build upon this to ascertain the specific clinical techniques therapists could use to foster positive developments and changes in the alliance (and in attitude towards treatment) which,

together with the emphasis on the aforementioned mechanisms of change, would lead to positive therapeutic outcomes for those with BPD.

## References

- American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5<sup>th</sup> Edition). Arlington, VA: American Psychiatric Publishing.
- American Psychiatric Association (2001). Practice guideline for the treatment of patients with borderline personality disorder—introduction. *American Journal of Psychiatry*, 158, 1-52.
- American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders* (Revised 4<sup>th</sup> Edition – Text Revision). Washington DC: American Psychiatric Association.
- Axelrod, S. R., Perepletchikova, F., Holtzman, K. & Sinha, R. (2011). Emotion regulation and substance use frequency in women with substance dependence and borderline personality disorder receiving dialectical behaviour therapy. *The American Journal of Drug and Alcohol Abuse*, 37, 37–42.
- Arntz, A. (1994). Treatment of borderline personality disorder: A challenge for cognitive behavioural therapy. *Behaviour, Research and Therapy*, 32, 419-430.
- Baer, R. A., Smith, G. T. & Allen, K. B. (2004). Assessment of mindfulness by self-report: The Kentucky Inventory of Mindfulness Skills. *Assessment*, 11, 191-206.

- Barber, J. P. & DeRubeis, R. J. (1992). The ways of responding: A scale to assess compensatory skills taught in cognitive therapy. *Behavioural Assessment*, 14, 93–115.
- Barnicot, K., Katsakou, C., Bhatti, N., Savill, M., Fierns, N. & Priebe, S. (2012). Factors predicting the outcome of psychotherapy for borderline personality disorder: a systematic review. *Clinical Psychology Review*, 32, 400-412.
- Bateman, A.W. & Fonagy, P. (2008). 8-year follow-up of patients treated for borderline personality disorder: Mentalization-based treatment versus treatment as usual. *American Journal of Psychiatry* 165, 631-638.
- Bateman, A. W. & Fonagy, P. (2004). Psychotherapy for borderline personality disorder: Mentalization-based treatment. Oxford: Oxford University Press.
- Bateman, A. W. & Fonagy, F. (2000). Effectiveness of psychotherapeutic treatment for personality disorders. *British Journal of Psychiatry*, 177, 138-143.
- Beck, A. T., Freeman, A., & Davis, D. D. (2004). *Cognitive therapy of personality disorders*. (2<sup>nd</sup> Edition). New York: Guilford.
- Bedics, J. D., Atkins, D. C., Comtois, K. A. & Linehan, M. M. (2012). Treatment differences in the therapeutic relationship and introject during a 2-year randomised controlled trial of dialectical behaviour therapy versus non-behavioural psychotherapy by experts for borderline personality disorder. *Journal of Consulting and Clinical Psychology*, 80, 66-77.

- Bender, D. S., Dolan, R. T., Skodol, A. E., Sanislow, C. A., Dyck, I. R., McGlashan, T. H., Shea, M. T., Zanarini, M. C., Oldham, J. M. & Gunderson, J. G. (2001). *American Journal of Psychiatry*, 158, 295–302.
- Benjamin, L. S. (1974). Structural analysis of social behaviour. *Psychological Review*, 81, 392–425.
- Bloom, J. M., Woodward, E. N., Sasmaras, T., Pantalone, D. W. (2012). Use of dialectical behaviour therapy in inpatient treatment of borderline personality disorder: a systematic review. *Psychiatric Services*, 63, 881–888.
- Brazier, J. E., Tumor, I., Holmes, M., Ferriter, M. Parry, G., Dent-Brown, K. & Paisley, S. (2006). Psychological therapies including dialectical behaviour therapy for borderline personality disorder: a systematic review and preliminary economic evaluation. *Health Technology Assessment*, 10, 1-138.
- Brown, G. K., Newman, C. F., Charlesworth, S. E., Crits-Christoph, P., & Beck, A. T. (2004). An open clinical trial of cognitive therapy for borderline personality disorder. *Journal of Personality Disorders*, 18, 257–271.
- Butler, A., Chapman, J. E., Forman, E. M. & Beck, A. T. (2006). The empirical status of cognitive-behavioural therapy: A review of meta-analyses. *Clinical Psychology Review*, 26, 17-31.
- Clarkin, J. F. & Levy, K. N. (2006). Psychotherapy for patients with borderline personality disorder: Focusing on the mechanisms of change. *Journal of Clinical Psychology*, 62, 405-410.

- Clarkin, J. F., Levy, K. N., Lenzenwenger, M. F. & Kernberg, O. F. (2004). The Personality Disorders Institute/Borderline Personality Disorders Research Foundation randomised control trial for borderline personality disorder: Rationale, methods and patient characteristics. *Journal of Personality Disorders*, 18, 52-72.
- Coid J., Yang M, Tyrer, P., Roberts, A., & Ullrich, S. (2006). Prevalence and correlates of personality disorder in Great Britain. *British Journal of Psychiatry*, 188, 423-431.
- Comtois, K. A., Russo, J., Snowden, M., Srebnik, D., Ries, R. & Roy-Byrne, P. (2003). Factors associated with high use of public mental health services by persons with borderline personality disorder. *Psychiatric Services*, 54, 1149–1154.
- Davenport, J., Bore, M. & Campbell, J. (2010). Changes in personality in pre- and post-dialectical behaviour therapy borderline personality disorder groups: A question of self-control. *American Psychologist*, 45, 59-66.
- Davidson, K., Norrie, J., Tyrer, P., Gumley, A. I., Tata, P., Murray, H., & Palmer, S. (2006). The effectiveness of cognitive behaviour therapy for borderline personality disorder: Results from the BOScot Trial. *Journal of Personality Disorders*, 20, 450–465.
- de Ruiter, C. & Greeven, P. J. G. (2000). Personality disorders in a Dutch forensic psychiatric sample: convergence of interview and self-report measures. *Journal of Personality Disorders*, 14, 162-170.

- Downs, S. H. & Black, N. (1998). The feasibility of creating a checklist for the assessment of the methodological quality both of randomised and non-randomised studies of health care interventions. *Journal of Epidemiological Community Health, 52*, 377-384.
- Elkin, I., Shea, M.T., Watkins, J.T., Imber, S.D., Sotsky, S.M., Collins, J.F., Glass, D. R., Pilkonis, P. A., Leber, W. R., Docherty, J. P., Fiester, S. J. & Parloff, M. B. (1989). National Institute of Mental Health Treatment of Depression Collaborative Research Program: General effectiveness of treatments. *Archives of General Psychiatry, 46*, 971–982.
- Elliott, R. (2010). Psychotherapy change process research: realizing the promise. *Psychotherapy Research, 20*, 123-235.
- Feigenbaum, J. D., Fonagy, P., Pilling, S. Jones, A., Wildgoose, A. & Bebbington, P. E. (2011). A real-world study of the effectiveness of DBT in the UK National Health Service. *British Journal of Clinical Psychology, 51*, 121-141.
- Feigenbaum, J. (2007). Dialectical behaviour therapy: An increasing evidence base. *Journal of Mental Health, 16*, 51-68.
- Gibbons, M. B. C., Crits-Christoph, P., Barber, J. P., Wiltsey Stirman, S., Gallop, R. Goldstein, L. A., Temes, C. M. & Ring-Kurtz, S. (2009). Unique and common mechanisms of change across cognitive and dynamic psychotherapies. *Journal of Consulting and Clinical Psychology, 77*, 801–813.

Gratz, K. L., Lacroce, D. M. & Gunderson, J. G. (2006). Measuring changes in symptoms relevant to borderline personality disorder following short-term treatment across partial hospital and intensive outpatient levels of care. *Journal of Psychiatric Practice*, 12, 153-159.

Gratz, K. L. & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathological Behaviour Assessment*, 26, 41–54.

Horvath, A. O. & Luborsky, L. (1993). The role of the therapeutic alliance in psychotherapy. *Journal of Consulting and Clinical Psychology*. 61, 561-573.

Kazdin, A. E. (2007). Mediators and mechanisms of change in psychotherapy research. *Annual Review of Clinical Psychology*, 3, 1-27.

Kellogg, S. H. and Young, J. E. (2006). Schema therapy for borderline personality disorder. *Journal of Clinical Psychology*, 62, 445-458.

Kliem, S. Kröger, C. & Kosfelder, J. (2010). Dialectical behaviour therapy for borderline personality disorder: a meta-analysis using mixed-effects modelling. *Journal of Consulting and Clinical Psychology*, 78, 936–951.

Leichsenring, F. & Leibing, E. (2003). The effectiveness of psychodynamic therapy and cognitive behaviour therapy in the treatment of personality disorders: a meta-analysis, *American Journal of Psychiatry*, 160, 1223–1232.



Lenzenweger, M. F., Clarkin, J. F., Levy, K. N., Yeomans, F. E. & Kernberg, O. F.

(2012). Predicting domains and rates of change in borderline personality disorder. *Personality Disorders: Theory, Research and Treatment*, 3, 185-195.

Lindenboim, N., Comtois, K. A. & Linehan, M. M. (2007). Skills practice in dialectical behaviour therapy for suicidal women meeting criteria for borderline personality disorder. *Cognitive and Behavioural Practice*, 14, 147—156.

Linehan, M., Comtois, K. A., Murray, A. M., Brown, M. Z., Gallop, R. J., Heard, H. L., Korslund, K. E., Tutek, D. A., Reynolds, S. K., Lindenboim, N. (2006). Two-year randomized controlled trial and follow-up of dialectical behaviour therapy verses therapy by experts for suicidal behaviours and borderline personality disorder. *Archives of General Psychiatry*, 63, 757-766.

Linehan, M. (1993). Cognitive-behavioural treatment of borderline personality disorder. New York: The Guilford Press.

Luborsky, L. (1984). Principles of psychoanalytic psychotherapy. New York: Basic Books.

Martin, D. J., Garske, J. P. & Davis, M. K. (2000). Relation of the therapeutic alliance with outcome and other variables: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, 68, 438-450.

- McMain, S., Links, P., Guimond, T., Wnuk, S., Eynan, R., Bergmans, Y., Warwar, S. (2013). An exploratory study of the relationship between changes in emotion and cognitive processes and treatment outcome in borderline personality disorder. *Psychotherapy Research, 23*, 658-673.
- McMain, S. Korman, L. M. & Dimeff, L. (2001). Dialectical behaviour therapy and the treatment of emotion dysregulation. *Journal of Clinical Psychology, 57*, 183-196.
- National Institute for Health and Clinical Excellence (2009). Borderline Personality Disorder: Treatment and management. CG78. London: National Institute for Health and Clinical Excellence.
- Neacsiu, A. D., Rizvi, S. L. & Linehan, M. M. (2010). Dialectical behaviour therapy skills use as a mediator and outcome of treatment for borderline personality disorder. *Behaviour, Research and Therapy, 48*, 832-839.
- Panos, P. T., Jackson, J. W., Hasan, O. & Panos, A. (2014). Meta-analysis and systematic review assessing the efficacy of dialectical behaviour therapy (DBT). *Research on Social Work Practice, 24*, 213-223.
- Perroud, N. Nicastrò, R., Jermann, F. & Huguelet, P. (2012). Mindfulness skills in borderline personality disorder patients during dialectical behaviour therapy: Preliminary results. *International Journal of Psychiatry in Clinical Practice, 16*, 189–196.

- Robins, J., & Rotnitzky, A. (2005). Estimation of treatment effects in randomised trials with non-compliance and dichotomous outcome using structural mean models. *Biometrika*, 91, 763-783.
- Sansone, R. A. & Sansone, L. A. (2011). Gender patterns in borderline personality disorder. *Innovations in Clinical Neuroscience*, 8, 16-20.
- Stoffers, J.M., Völlm, B.A., Rucker, G., Timmer, A., Huband, N. & Lieb, K. (2012). Psychological therapies for people with borderline personality disorder. *Cochrane Database of Systematic Reviews 2012*, Issue 8. Art. No.: CD005652
- Turner, R. M. (2000). Naturalistic evaluation of dialectical behaviour therapy-oriented treatment for borderline personality disorder. *Cognitive and Behavioural Practice*, 7, 413-419.
- Wenzel, A., Chapman, J. E., Newman, C. F., Beck, A. T. & Brown, G. K. (2006). Hypothesized mechanisms of change in cognitive therapy for borderline personality disorder. *Journal of Clinical Psychology*, 62, 503-516.
- Yen, S., Johnson, J., Costello, E. & Simpson, E. B. (2009). A 5-day dialectical behaviour therapy partial hospital program for women with borderline personality disorder: Predictors of outcome from a 3-month follow-up study. *Journal of Psychiatric Practice*, 15, 173–182.

## **Part 2: Empirical Paper**

### **Factors Affecting Treatment Completion and Treatment Outcome in a Naturalistic Study of Psychological Therapy for Personality Disorder**

## Abstract

**Aims:** Patients receiving therapy for personality disorder (PD) are likely to disengage prematurely and little is known about factors predicting treatment completion. For those that do complete treatment, there is a lack of research regarding factors that predict outcome. Predicting both completion and outcome of treatment is important for service planning. This study therefore aimed to identify predictive factors in individuals receiving either Cognitive Behavioural Therapy (CBT) or Dialectical Behaviour Therapy (DBT) for their PD.

**Method:** This is a correlational study of variables predicting dropout and clinical outcome from a naturalistic sample of 231 male and female patients receiving outpatient CBT or DBT in a specialist PD service. Clinical measures were collected at initial assessment and post-treatment. Information regarding demographics and attendance were gathered from NHS electronic notes systems.

**Results:** Therapist expertise was revealed as the only significant predictor of treatment completion; therapists defined as more expert retained patients in treatment longer. Multiple hierarchical regressions revealed that better therapy attendance and having a more expert therapist predicted decreases in risk (suicide attempts/self-harm) and in number of PD diagnoses. Better attendance predicted a decrease in number of clinical syndrome diagnoses (e.g. anxiety/depression). The presence of comorbid substance misuse at initial assessment predicted increases in risk and number of PD diagnoses.

**Conclusion:** The importance of therapist expertise in treatment completion appears to be somewhat novel and warrants future replication. Findings are in agreement with previous literature documenting poorer outcomes for those with comorbid substance misuse. Encouraging patients to comply with therapy attendance to the point of completion is crucial in order to obtain the best possible reductions in risk behaviour and PD/clinical diagnoses. Other patient variables that are likely to predict treatment completion and outcome should be considered by future research.

## Introduction

Personality Disorder (PD) is defined by the newest edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013) as, “an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual’s culture, is pervasive and inflexible, has an onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment” (p. 645). The diagnosis of PD is rarely restricted to one PD (Tyrer & Ferguson, 2000) and currently, DSM-5 classifies PD into ten distinct diagnostic classifications. Contrary to previous versions of the DSM which categorised PD onto its own Axis (Axis II) and placed clinical syndromes such as anxiety and mood disorders onto Axis I, since July 2013, the new edition categorises both clinical syndromes and PDs onto Axis I, with PDs forming their own subset of this Axis. Throughout the rest of this paper the former Axis I/Axis II distinction will be made using the terms ‘clinical syndromes’ (referring to mood and anxiety disorders such as depression) and ‘PDs’ (referring to any diagnostic category of PD). Collectively, the presence of clinical syndromes and PDs will be referred to as ‘clinical comorbidity’ – any clinical and PD diagnoses the patient has in addition to their primary PD diagnosis.

Using the Structured Clinical Interview for DSM-IV-Axis I (SCID-I; First, Gibbon, Spitzer, Williams & Benjamin, 1997), Coid, Yang, Tyrer, Roberts and Ullrich (2006) estimated the prevalence of PD to affect approximately five per cent of the population. While the current study is interested in all PDs (with the exception of schizoid or antisocial PD (ASPD) which are excluded from the service providing data for this study), it will focus primarily on Borderline Personality Disorder (BPD), largely because of its higher prevalence among patients presenting to services (Coid et al., 2006; de Ruiter & Greeven, 2000) and because of its prominence in PD treatment research (Feigenbaum et al., 2011). BPD has been defined as, “a

complex and serious mental disorder that is characterised by a pervasive pattern of difficulties with emotion regulation and impulse control and instability both in relationships and self-image” (Bateman & Fonagy, 2004; p.1).

### **Current treatment of personality disorders**

Contrary to previous opinion which held that due to its entrenched nature and roots in childhood, PD was largely untreatable, there is now evidence to suggest that BPD and other PDs can be successfully treated using psychological therapy (e.g. Binks et al., 2006; Panos, Jackson, Hasan & Panos, 2013; Stoffers et al., 2012). Many research studies have attempted to ascertain the efficacy of different psychotherapies used for the treatment of BPD including Dialectical Behaviour Therapy (DBT; Linehan, 1993) for women which is currently the only treatment recommended for BPD by the National Institute for Clinical Excellence (NICE, 2009). NICE suggests that should the evidence base be produced, future revisions may advocate the treatment of BPD using Cognitive Behaviour Therapy (CBT; Beck, Freeman & Davis, 2004), Mentalization Based Therapy (MBT; Bateman & Fonagy, 2008; Bateman & Fonagy, 2004) and Schema Therapy (Kellogg & Young, 2006). DBT could be described as a third-wave form of CBT and has a growing large and robust evidence base (e.g. Bloom, Woodward, Sasmaras & Pantalone, 2012; Feigenbaum et al., 2011; Feigenbaum, 2007; Kliem, Kröger & Kosfelder, 2010; Linehan et al., 2006a; Panos et al., 2013). Likewise, strong bodies of empirical evidence reliably document the effectiveness of both generic CBT (Butler, Chapman, Forman and Beck, 2006) and CBT specifically for BPD (Davidson et al., 2006). The current study focuses solely on the CBT and DBT treatment of PD.

CBT is a structured, time limited, problem-focused treatment that uses traditional cognitive and behavioural techniques to teach patients to identify dysfunctional thoughts and core beliefs and to learn to challenge and modify them. CBT for BPD focuses particularly on altering core beliefs and is described by

Davidson et al. (2006) as less intensive with regards to therapist time than other forms of psychotherapy developed for BPD, such as DBT. A large randomised controlled trial (RCT) of individual CBT for BPD verses treatment as usual (TAU) found that CBT was roughly equivalent to TAU on most outcomes (including depression) and superior on outcomes of state anxiety, dysfunctional core beliefs and quantity of suicidal acts at two year follow-up (Davidson et al., 2006). Leichsenring and Leibing's (2003) meta-analysis reported significant effects for a reduction in depressive symptoms for CBT treatment for PD over that of psychodynamic therapy for PD. Weinberg, Gunderson, Hennen and Cutter (2006) developed and studied a form of cognitive therapy, Manual Assisted Cognitive Treatment (MACT) aimed specifically at treating self-harming or suicidal patients with BPD. Their findings demonstrated that MACT was associated with significantly less frequent deliberate self-harm at the end of treatment as well as less severity of self-harm at six month follow-up. Using the same treatment programme, Freije, Dietz and Appelo (2002) found improvements in borderline symptoms, global function, depression, anxiety and interpersonal sensitivity in 85 patients with BPD.

Developed by Linehan (1993), DBT uses strategies from CBT to develop and generalise emotion regulation skills as well as teaching distress tolerance and using third wave approaches such as Mindfulness (Kabat-Zinn, 1994) to promote awareness and acceptance. The DBT model posits that problems encountered by the individual are a result of emotion dysregulation which is maintained by certain personal and environmental reinforcers, integrating a biopsychosocial understanding of factors contributing to maladaptive behaviour. DBT aims to teach the necessary skills for effective interpersonal function and self-regulation (Winston, 2000). Sessions are delivered both in weekly individual format and through skills development groups to maximise patient benefit, and are therefore more time-intensive for both the patient and the therapist.



To date several RCTs and naturalistic studies have demonstrated the efficacy of DBT for BPD (e.g. Feigenbaum, 2007; Koons et al., 2001; Linehan et al., 2006a; Verheul et al., 2003) and these findings have been pooled and confirmed by recent meta-analyses (Kliem et al., 2010; Öst, 2008; Panos et al., 2013) and systematic reviews (Binks et al., 2006; Bloom et al., 2012; Stoffers et al., 2012), although a need for replicatory studies persists (Stoffers et al., 2012).

### **Randomised controlled trials verses naturalistic outcome studies**

It is important to note the crucial role of RCTs in establishing an evidence base for the effectiveness of psychotherapeutic treatment for PD (e.g. Bateman & Fonagy, 2008; Bateman & Fonagy, 1999; Davidson et al., 2006). Due to their tight control of extraneous variables, randomisation to comparison groups and the 'blinding' of participant, observer and sometimes researcher, RCTs have typically been considered the 'gold standard' of research studies and accordingly occupy the uppermost position in the hierarchy of evidence that organises healthcare research (described by Pistrang, Barker & Elliott, 2002). However there is a distinction between what can be concluded through a tightly controlled RCT with high internal validity (*research efficacy*) verses the conclusions which can be drawn from a less controlled but more generalisable and externally valid *clinical effectiveness* study (Pistrang et al., 2002; Roth & Fonagy, 2005; Seligman, 1995). There has been, and continues to be, a strong debate over which is more useful, both in general medicine (Grapow, von Wattenwyl, Guller, Beyersdorf & Zerkowski, 2006; Rothwell, 2005) and in psychotherapy (Chambless & Hollon, 1998; Fonagy, 1999; Leichsenring, 2004; Roth & Fonagy, 2005; Seligman, 1995), with researchers noting how vital it is for research studies to reflect 'real-world' healthcare provision, providing more generalisable and useful findings (Binks et al., 2006; Feigenbaum et al., 2011).

## **Predictors of treatment completion**

It is widely accepted that patients with PD are difficult to treat due to their tendency to engage in behaviours that interfere with the therapeutic process such as irregular attendance, disengagement and premature dropout from treatment (Fonagy & Bateman, 2006). Keeping patients attending therapy long enough to achieve beneficial effects is therefore a key treatment aim, particularly as non-completion hinders the patient's chance of symptom improvement (McMurran, Huband & Overton, 2010).

Linehan et al. (2006a) report that compared to a control treatment, DBT was twice as effective at retaining BPD patients in treatment (25% dropout rate in DBT verses 59% in control treatment) and similarly, Kliem et al.'s (2010) meta-analysis of 16 studies of BPD treatment reported an overall dropout rate of 27%. However, in Feigenbaum et al.'s (2011) study of 26 patients entering DBT treatment only eleven continued to complete one year of treatment, an attrition rate of 58%, similar to that reported by Clarkin, Levy, Lenzenweger and Kernberg (2007). There is a large discrepancy in dropout rates between studies, and Barnicot, Katsakou, Marougka and Priebe's (2011) meta-analysis of treatment completion in psychotherapy for BPD confirmed the same, concluding that this substantial variation remains unexplained and that further research into processes involved in treatment dropout or completion is warranted.

Defining early dropout as completing less than three months of therapy, De Panfilis et al. (2012) found that of the 162 patients with BPD studied, one third dropped out early. They investigated only patient factors involved in early dropout and found that a history of suicide attempts predicted early discontinuation while comorbid eating disorders were protective from early dropout. Importantly this study excluded those with comorbid substance misuse which seems a crucial factor to consider given its known association with PD (particularly BPD (American Psychiatric Association, 2013)), and in light of previous findings which concluded

that current or previous substance use was associated with non-completion of PD treatment (Karterud et al., 2003; Linehan et al., 2002; Linehan et al., 1999).

Barnicot et al.'s (2011) meta-analysis of psychotherapeutic treatments for BPD found that only eleven of the 41 studies they examined investigated predictors of treatment dropout and that there was thus far very little evidence on predictors. Nevertheless, they reported that no patient sociodemographic variables (age, gender, employment status) were associated with attrition from therapy for BPD. McMurrin and colleagues (whose work typically targets forensic and ASPD populations, in contrast to Barnicot who focuses more on BPD) conducted a systematic review of non-completion of PD treatments which concluded that non-completion was associated with younger age, lower education level and unemployment, as well as with having more PD diagnoses, meeting a higher number of PD criteria, and having a more severe level of depression (McMurrin et al., 2010). Feigenbaum et al. (2011) found that patients with comorbid PDs, (specifically paranoid PD and ASPD) comprised the majority of their high dropout rate.

This suggests that, for some PDs at least, as well as demographic variables, overall comorbidity (both clinical syndromes and PDs) is likely to reduce treatment adherence although this may depend on the type of comorbidity and the primary diagnosis of the sample population as opposing findings have been reported (De Panfilis et al., 2012). Research on predictors of treatment completion to date has varied substantially and clarification of these factors is a worthwhile endeavour.

### **Predictors of treatment outcome**

Despite a growing body of research into the treatment of PD, very little is known about predictors of treatment outcome (Robins & Chapman, 2004). Robins and Chapman (2004) summarise possible predictors in DBT treatment to involve characteristics of the patient, characteristics of the therapist and characteristics of

the combination of patient and therapist (including therapeutic relationship). Ryle and Golyukina (2000) investigated time-limited CAT for BPD concluding that worse outcomes were associated with greater severity of borderline features, a history of cutting, alcohol abuse and unemployment although more recent research using a novel treatment<sup>1</sup> has concluded that better outcomes for clinical symptoms were obtained for those with more severe BPD symptoms at baseline (Black et al., 2009). A recent systematic review by Barnicot et al. (2012) investigating factors which predict outcome of psychotherapy for BPD (psychotherapy consisted mainly of CBT, DBT, MBT and transference-focused psychotherapy) concluded that the therapeutic alliance was the most important common factor in predicting patients' therapeutic outcomes.

### **Outcome variables**

Following successful treatment of PD, previous studies have recorded improved outcomes in post-traumatic stress disorder (PTSD) symptom severity (Feigenbaum et al., 2011), self-harm and suicidality (Bohus et al., 1999; Feigenbaum et al., 2011; Linehan et al., 2006a; Linehan, Tutek, Heard & Armstrong, 1994; Low, Jones, Duggan, Power & MacLeod, 2001; Sanislow & McGlashan, 1998), interpersonal function (Linehan et al., 1994), anger reduction (Koons et al., 2001; Linehan et al., 1994), reduced psychiatric inpatient hospital admissions (Linehan et al., 2006a; Linehan et al., 1994) and a decrease in symptoms of depression and hopelessness (Koons et al., 2001).

---

<sup>1</sup> Systems Training for Emotional Predictability and Problem Solving (STEPPS; Blum et al., 2002). A group treatment combining CBT, skills training and a systems element which involves family, friends and other people who feature regularly in the patient's life.

## Current study

Given the importance of studying clinical effectiveness within the parameters of a 'real-world' National Health Service (NHS) PD service, the current study sought to investigate and identify the factors which influence treatment completion and treatment outcome in psychological therapy for BPD and other PDs. Data was generated by an established, dedicated NHS PD service over a period of eleven years and for the present analysis spanned a period of six years (2008-2014). It is of particular interest to identify factors which can predict treatment completion and treatment outcome to better understand for whom treatment is likely to work, and who would benefit from additional interventions/adaptations to facilitate their engagement and improvement.

Based on the existing literature, the following variables were considered as potential predictors of treatment completion and/or outcome:

*Therapeutic dose*<sup>2</sup>. Number of sessions attended can be thought of in terms of *therapeutic dose* which is likely to be a predictive factor as it has been demonstrated that different conditions begin responding at different doses of therapy. Howard, Kofta, Krause and Orlinsky (1986) found that a positive response in depressive patients began at the lowest dose of psychotherapy, anxious-neurotics at a somewhat higher dose and borderline-psychotics at the highest dosage of the three patient groups. Bowen, South, Fischer and Looman (1994) found that number of therapy sessions attended predicted a favourable outcome in patients with panic and agoraphobia in a behavioural/medication trial and in an RCT of CBT and imaginal exposure for patients with PTSD, Tarrier et al. (1999) concluded that patients whose symptoms worsened over time had a greater tendency to fail to attend sessions. With regards to PD treatment, McMurrin et al.'s (2010) systematic

---

<sup>2</sup> Therapeutic dose (number of sessions attended) was considered a predictor in analyses of treatment outcome only, given that it is inherently related to treatment completion and therefore would not be a useful predictor in treatment completion analyses (see *Hypotheses* section, below).

review concluded that not completing treatment meant that patients received a much less effective therapy than those who completed. Good therapy attendance is therefore a factor likely to have a significant effect on treatment outcome.

*Therapist expertise.* As well as patient variables, therapist factors should also be considered (Robins & Chapman, 2004) and could function as a predictor of treatment completion as well as outcome. Across 161 studies, Luborsky, Auerbach, Chauder, Cohen and Bachrach (1971) identified several therapist characteristics that influenced therapy outcome including experience, attitude/interest patterns and empathy. Collectively, these factors might be thought of as therapist expertise which is also likely to be influenced by training/professional development and receipt of supervision from a more experienced therapist. There is little evidence documenting the role of therapist expertise in treatment completion although Feigenbaum et al. (2011) note a higher dropout rate in PD patients receiving DBT from a particular therapist who it was agreed had been delivering poor-quality DBT. Most of the literature on this subject appears to concern therapists treating clinical syndromes such as anxiety and mood disorders. Franklin, Abramowitz, Furr, Kalsy and Riggs (2003) studied the relationship between therapist experience and outcome in a trial of exposure and response prevention for Obsessive Compulsive Disorder (OCD) and found that there was no difference in mean post-treatment OCD severity scores between the least experienced and most experienced groups (0-1 years or more than 9 years experience). However, they did note that case assignment in their naturalistic study meant that at pre-treatment those patients with more severe and difficult-to-treat OCD were assigned to more experienced therapists. Andrews (2001) notes that, "although the relationship between therapist experience and outcome is not linear, there are indications that more experienced therapists are likely to retain their clients longer and are more helpful to seriously impaired patients" (p.108). Despite the lack of existing research confirming the role of therapist expertise, the present study sought to examine the role of therapist

expertise in PD treatment completion and outcome based on the assumption that more experienced therapists are likely to retain their patients in therapy for longer, delivering a higher quality of treatment than less experienced therapists.

*Comorbid substance abuse.* Substance misuse is a common problem across most PDs, occurring in roughly two thirds of patients with BPD (Dulit, Feyer, Haas, Sullivan & Frances, 1990) and is in fact incorporated into one of the diagnostic features (impulsive and reckless behaviour) of BPD (DSM-5; American Psychiatric Association, 2013). Interestingly, Dulit et al. (1990) found that when substance misuse was not used as a diagnostic criterion for BPD, almost a quarter of patients no longer met DSM-IV (American Psychiatric Association, 2000) criteria for BPD. Westen, Novotny and Thompson-Brenner (2004) note the tendency for previous research to exclude difficult-to-treat BPD patients with comorbid substance misuse disorders regardless of it being a common feature of BPD. Substance misuse comorbidity has been reported to be associated with failure to achieve remission from BPD (Zanarini, Frankenburg, Hennen & Silk, 2003) and worse outcomes (Ryle and Golyukina, 2000) as well as with early and high levels of treatment dropout (Karterud et al., 2003; Linehan et al., 2002; Linehan et al., 1999). However, more promising results have been reported for recovery from substance abuse in people with BPD, suggesting that it may be possible to decrease some drug-related harmful behaviour that features in this group of patients, as well as to reduce overall psychopathology (Linehan, et al., 2002). An RCT of participants with BPD and drug-dependence who received either DBT or TAU found that after 12 months of treatment and a 16 month follow-up, patients receiving DBT were abusing drugs at a significantly lower level than the TAU group (Linehan et al., 1999). Nevertheless, substance abuse is likely to have an effect on completion and outcome.

Changes in the following treatment outcome variables were measured:

*High risk behaviour.* Several studies of PD treatment have reported a reduction in deliberate self-harm (DSH) and suicide attempts following treatment (Bohus et al., 1999; Bloom et al., 2012; Davidson et al., 2006; Feigenbaum et al., 2011; Linehan et al., 1994; Low et al., 2001), an obviously desirable outcome.

*Clinical comorbidity*<sup>3</sup>. Research has shown that mood disorders and substance misuse often co-occur with BPD (Afifi et al., 2011; Oldham et al., 1995; Skodol, Oldham & Gallagher, 1999; Widom, Czaja & Paris, 2009; Zanarini, Frankenburg, Hennen, Reich & Silk, 2006; Zanarini et al., 1998; Zimmerman & Mattia, 1999), as do other PD diagnoses (Afifi et al., 2011; Zanarini et al., 2006). This comorbidity has been shown to lead to higher dropout rates (Feigenbaum et al., 2011), worse outcomes for people with BPD over a ten year follow-up period (Zanarini et al., 2006) and worse outcomes for treatment of mood disorders and other clinical syndromes when PD pathology is present (Reich & Vasile, 1993). Moreover, patients with comorbid PDs are reported to have more severe forms of these syndromes (Tyrer et al., 1990).

Previous studies have showed that with successful treatment it is possible to reduce comorbidity, particularly depression and hopelessness, as well as symptoms characteristic of anxiety disorders (Freije et al., 2002; Koons et al., 2001). Links, Heslegrave and van Reekum (1998) found that 53% of patients followed-up for seven years no longer met diagnostic criteria for BPD and a large longitudinal study found at follow-up that two thirds of patients were clinically well with no clinical syndromes or PD diagnoses (Stone, Hurt & Stone, 1987). Following treatment, Sanislow and McGlashan (1998) reported fewer general PD symptoms and in patients treated with a combination of CBT and a systemic-based skills group for BPD, a reduction in BPD symptoms was noted (Blum, Pfohl, St. John, Monahan and Black, 2002; Freije et al., 2002), although a later Cochrane review found no change

---

<sup>3</sup> Clinical comorbidity was considered as a predictor variable in treatment completion analyses and as an outcome variable in treatment outcome analyses (see *Hypotheses* section, below).



in number of SCID-II-defined (First et al., 1997) PD diagnoses following six months of DBT treatment (Binks et al., 2006). It is likely that clinical comorbidity could have both an effect upon treatment completion as well as functioning as an outcome measure of treatment success.

Based on existing research and literature, the following hypotheses were examined:

### **Hypotheses**

#### *Hypothesis 1: Predictors of treatment completion*

1. Clinical comorbidity (number of clinical syndrome diagnoses and number of PD diagnoses at baseline), therapist expertise, and baseline substance misuse will be predictive of treatment completion.

Specifically,

- 1a) Higher therapist expertise will be predictive of treatment completers.
- 1b) Higher levels of clinical comorbidity will be predictive of non-completers.
- 1c) The presence of baseline substance misuse will be predictive of non-completers.

#### *Hypothesis 2: Predictors of treatment outcome*

2.1 Therapeutic dose, therapist expertise and baseline substance misuse will be predictive of changes in risk.

Specifically,

- 2.1a) Attending more sessions will be predictive of improvement in risk outcome.
- 2.1b) Having a more experienced therapist will be predictive of improvement in risk outcome.
- 2.1c) The presence of baseline substance misuse will be predictive of worse risk outcome.

2.2 Therapeutic dose, therapist expertise and baseline substance misuse will be predictive of change in PD clinical comorbidity (measured by change in number of PD diagnoses).

Specifically,

2.2a) Attending more sessions will be predictive of improvement in number of PD diagnoses.

2.2b) Having a more experienced therapist will be predictive of improvement in number of PD diagnoses.

2.2c) The presence of baseline substance misuse will be predictive of no improvement in or increase in number of PD diagnoses.

2.3 Therapeutic dose, therapist expertise and baseline substance misuse will be predictive of change in clinical comorbidity (measured by change in number of clinical syndrome diagnoses).

Specifically,

2.3a) Attending more sessions will be predictive of improvement in number of clinical syndrome diagnoses.

2.3b) Having a more experienced therapist will be predictive of improvement in number of clinical syndrome diagnoses.

2.3c) Baseline substance misuse will be predictive of no improvement in or increase in number of clinical syndrome diagnoses.

## **Method**

### **Design and setting**

The study used a pre- and post-treatment correlational design and took place in an NHS PD service. It was conducted as part of a larger naturalistic

investigation of outcomes for treatment of PD (Feigenbaum, in progress). The outpatient therapy service offers adults and older adults with a diagnosis of at least one PD either CBT or DBT treatment which typically lasts for one year. Individuals whose primary diagnosis is schizoid PD, antisocial PD, moderate to severe learning disability, personality change due to head injury, or florid psychotic disorder are excluded.

### **Sample size and statistical power**

It is conventional in psychological research to conduct a power analysis to determine the minimum sample size acceptable to achieve a given effect size. However, there are no generally agreed methods for relating the sample size to the number of predictor variables in a regression model and various 'rules of thumb' are used by different researchers (Field, 2009). Miles and Shevlin (2001) created a set of graphs which approximate sample size required to achieve different effect sizes with different levels of power based on number of predictors. Using the conventional power setting of 0.8 (Cohen, 1988), their graph recommends that a sample size of 80 would detect a medium effect with three predictors. The current study achieved a sample size of 231 which should be large enough to detect a small to medium effect with the three hypothesised predictors.

### **Participants**

Recruitment into the main study sample was conducted retrospectively. Sampling began by screening initial assessment reports, measures, online case records and correspondence of patients who were assessed by the service between 2008 and 2013 to determine their suitability for inclusion. Patients who did not have completed measures detailing their clinical and PD diagnoses, risk behaviour, substance misuse, attendance and demographic information at baseline assessment were removed from the final analysis ( $n = 4$ ). Six further patients were

not included in the study as they had not been diagnosed with PD at their initial assessment within the service. For patients who completed treatment the same process was used to extract data from discharge assessments and measures post-completion. One hundred and seventy patients (74% of total sample) were categorised by their therapist as treatment completers and had data available both pre-treatment at baseline assessment and post-treatment at discharge assessment. A further subset of patients (n = 61, 26% of total sample) failed to complete their assessment, or completed the assessment but declined or dropped out of treatment before it was complete<sup>4</sup>.

Table 1 presents demographic characteristics of treatment completers and non-completers at baseline assessment for the final sample of 231. Seventy eight per cent were female and 22% were male. Mean age at time of baseline assessment was 32.8 (s. d. = 10.6, range = 18-70). Patients in full-time college education or either part-time or full-time employment made up 23% of the total sample (n = 54). Seventy two per cent of the total sample classed themselves as White British, 9% as any other White background, 8% as Black or mixed White and Black, 8% as Asian or mixed White and Asian, and 3% as any other ethnicity.

Prior to testing for significant differences between treatment completers and non-completers, demographic variables were tested for each of the groups using standard normality checks (histograms, skewness and kurtosis statistics and Kolmogorov-Smirnoff tests) and all were found to be skewed and significantly deviated from a normal distribution. Removal of outliers by calculating z-scores and removing cases more than three standard deviations above or below the mean did not return normal distribution samples on any variables, therefore non-parametric

---

<sup>4</sup> All patients included in analyses had previously been diagnosed with PD at their first point of contact with the service. Assessments that were not completed related to assessing patients' suitability for therapy and further selection of either CBT or DBT treatment.

tests (Mann-Whitney U and Chi Square) were used to test for between-group differences.

There was no significant difference in age at time of baseline assessment between treatment completers and non-completers (treatment completers: mean age = 33.3 years, s. d. = 10.7, n = 170; non-completers: mean age = 31.2 years, s. d. = 10.2, n = 61;  $U = 4559$ ,  $p = 0.16$ , n. s.) or in gender (treatment completers: female n = 136, male n = 34; non-completers: female n = 45, male n = 16;  $\chi^2(1) = 1.03$ ,  $p = 0.31$ , n. s.). There was also no significant difference in ethnicity between the two groups (treatment completers: White British n = 130, any other ethnicity n = 40; non-completers: White British n = 38, any other ethnicity n = 23;  $\chi^2(14) = 16.47$ ,  $p = .29$ , n. s.) or in employment status at baseline (treatment completers: employed n = 40, unemployed n = 130; non-completers: employed n = 14, unemployed n = 47;  $\chi^2(1) = 0.008$ ,  $p = .93$ , n. s.).

Table 1  
*Demographic characteristics of treatment completers and non-completers at baseline*

Demographic	Treatment completers (n = 170)	Non-completers (n = 61)
Female	136 (80%)	45 (74%)
Male	34 (20%)	16 (26%)
Age	33.3 years $\pm$ 10.7 (range = 18-70)	31.2 years $\pm$ 10.2 (range = 18-54)
Employed	40 (23.5%)	14 (23%)
Unemployed	130 (76.5%)	47 (77%)
White British	130 (76%)	38 (63%)
White Other	10 (6%)	10 (16%)
Black or Black Mixed	12 (7%)	6 (10%)
Asian or Asian Mixed	13 (8%)	5 (8%)
Any Other Ethnic Origin	5 (3%)	2 (3%)

A final test was conducted to check for any difference between the two groups based on number of PD diagnoses at baseline assessment and on the treatment type they received (CBT or DBT) to ensure there was no significant variation. Again, Mann-Whitney and Chi Square tests were used. There was no significant difference on number of PD diagnoses (treatment completers: mean number of PD diagnoses = 1.3, s. d. = 0.5, n = 170; non-completers: mean number of PD diagnoses = 1.2, s. d. = 0.4, n = 61;  $U = 4895$ ,  $p = .36$ , n. s.). Not all non-

completers were allocated a treatment type due to disengagement before allocation of treatment but of those non-completers who were allocated a treatment condition (n = 16) there was no significant difference between the two groups based on treatment type received (treatment completers: CBT n = 84, DBT n = 86; non-completers CBT n = 8, DBT n = 8;  $\chi^2(1) = 0.002$ ,  $p = .96$ , n. s). Tables 2 and 3 show treatment type (Table 2) and frequencies of each PD diagnosis (Table 3) for completers and non-completers at baseline.

Table 2  
*Treatment type of treatment completers and non-completers at baseline*

Treatment type	Treatment completers (n = 170)	Non-completers (n = 61)
CBT	84 (49.4%)	8 (13.1%)
DBT	86 (50.6%)	8 (13.1%)
Not allocated treatment type	0 (0%)	45 (73.8%)

Table 3  
*Personality disorder profiles (SCID-II) of completers and non-completers at baseline*

PD diagnosis	Treatment completers (n = 170)	Non-completers (n = 61)
Borderline	162 (95.3%)	59 (96.7%)
Avoidant	17 (10%)	5 (8.2%)
Dependent	11 (6.5%)	1 (1.6%)
OCPD	8 (4.7%)	3 (4.9%)
Paranoid	6 (3.5%)	1 (1.6%)
Histrionic	5 (2.9%)	1 (1.6%)
Narcissistic	4 (2.4%)	0 (0%)
Schizotypal	1 (0.6%)	0 (0%)
Mean number of PD diagnoses	1.3 (s. d. = 0.5)	1.2 (s. d. = 0.4)

## Ethics

Ethical approval was sought from the local NHS Research Ethics Committee (REC) prior to the commencement of data collection. The study was given a favourable ethical opinion (REC Reference Number: 12/LO/0382; see Appendix B). Additionally, all patient correspondence from the service contains a phrase explaining that the service has an open file policy meaning that anonymised data may be used for research, service evaluation and audit purposes. All data was

treated in accordance with ethical guidelines pertaining to security and confidentiality of information.

## **Procedure**

Data was collected retrospectively from various sources: clinical notes, online case record systems, electronic correspondence and reports and measures that therapists administered as part of baseline and discharge assessments.

## **Measures<sup>5</sup>**

*Suicide Attempts Self-Injury Interview (SASII)* (Linehan et al., 2006b). The SASII was administered at baseline assessment (and post-treatment for treatment completers) to assess high risk behaviour (both DSH and suicide attempts). For non-completers, therapists were asked to complete the SASII based on their last contact with the patient prior to disengagement. The SASII is a semi-structured interview measure which breaks down the previous year into one month blocks, requiring the patient to recall the frequency of DSH and suicide attempts during each month of the last year. A calendar was used to point out significant dates, promoting optimum recall. All episodes of DSH reported were recorded as a single incident of DSH, regardless of how close together each incident occurred. Suicide attempts were asked about in some detail in order to clarify the patient's genuine intention to die (rather than representing a further incident of DSH), for example by asking, "did you leave a note?" and "did you expect to be found?" The SASII has been demonstrated to have good inter-rater reliability with high correlations across

---

<sup>5</sup> A number of other self-report measures not reported here were also administered at baseline and post-treatment as part of standard data collection per the service's open file policy, and as part of a larger research trial (Feigenbaum, unpublished). The SASII, SCID-I and SCID-II have not been included in the appendices due to copyright protection.

assessor-rated items and good validity with concurrent measures of self-injury such as medical records (median item correlation  $r = 0.956$ ; Linehan et al., 2006b).

*Structured Clinical Interview for DSM-IV-II (SCID-II)* (First et al., 1997). The SCID-II is a structured clinical interview conducted at baseline (and at discharge assessments for treatment completers) to ascertain number and type of PD diagnoses. All therapists were fully trained in the use of SCID-II and random reliability checks were conducted monthly by the service's clinical lead in order to assess inter-rater agreement. For non-completers, therapists completed the SCID-II based on the patient's psychopathology at their last contact with the patient. Diagnostic criteria for each PD diagnostic category are established by the SCID-II using a range of trait-based questions and each trait is rated by the clinician as either "absent", "possible" or "definitely present". Test-retest and inter-rater reliability of the SCID-II have been extensively researched. Dreessen and Arntz (1998) produced high reliability scores in most subscales of the SCID-II using an outpatient population.

*Structured Clinical Interview for DSM-IV-I (SCID-I)* (First, Spitzer, Gibbon & Williams, 2002). The SCID-I is a structured interview administered as part of baseline assessment (and discharge assessment for completers) in order to assess the number and type of clinical syndromes. All therapists received training in the use of SCID-I and again, random reliability checks were conducted at regular intervals by the clinical lead of the service. For non-completers, therapists completed the SCID-I based on the patient's psychopathology at their last contact with the patient. The SCID-I interview assesses symptomology across a number of domains (including mood disorders, anxiety disorders and eating disorders) using questions to assess different traits of each diagnostic disorder which the clinician rates as either "absent", "possible" or "definitely present". The SCID-I is a lengthy and thorough diagnostic tool which research has shown to have high inter-rater reliability and high test-retest reliability for most subscales (Zanarini et al., 2000).



*Therapeutic dose.* This was quantified by recording the number of therapy sessions attended (which included both group and individual therapy sessions) as identified on the NHS RiO electronic notes system.

*Therapist Expertise.* Therapists' expertise was assessed by the clinical lead of the service (an international expert trainer in both CBT and DBT for PD), who rated therapists on a four point Likert scale (novice, experienced, adherent and very experienced, adherent and highly skilful) based on perceived level of expertise observed in supervision. This was corroborated using a combination of the following: scores on the CBT adherence rating scale (the Cognitive Therapy Scale-Revised (CTS-R); Blackburn et al., 2001), putative ratings of adherence using knowledge of the DBT competencies framework (available at [www.ucl.ac.uk/CORE](http://www.ucl.ac.uk/CORE)) and the DBT Expert Rating Scale (Linehan, Lockard, Wagner, & Tutek, 1996), the number of months the therapist had been working within the service, clinical supervision notes and consideration of professional development trainings attended.

*Christo Inventory for Substance-misuse Services (CISS).* Christo, Spurrell & Alcorn, 2000). The CISS was used both pre-treatment at baseline assessment and post-treatment at discharge assessment for treatment completers. For non-completers, therapists were asked whether or not patients were abusing substances at their point of disengagement with the service. This is a clinician-rated measure asking specifically about type and amount of substance use (including alcohol) in the 30 days prior to questioning only. Christo et al. (2000) report CISS test-retest and inter-rater reliability coefficients of 0.82 and 0.82, respectively. Information on patients' substance misuse was also available for corroboration from the SCID-I. See Appendix C for copy of the CISS.

## **Analysis**

Analysis was conducted using SPSS Statistics for Windows, Version 22.0 (IBM, 2013). The sample was divided into two groups: those who completed

treatment and those who did not. Demographic variables were checked for normality and non-parametric tests were conducted to ensure there were no significant differences between completers and non-completers, as described above (see *Participants* section). Outcome variables were recoded and change between baseline and completion of, or dropout from, treatment was computed (see *Computation of variables*, below). All variables were checked to ensure parametric assumptions of normal distribution were met. Where this was not the case, transformations were considered (see *Results*, below). Using data collected both pre-treatment and at the point of dropout or treatment completion, the main analysis consisted of simple, logistic and multiple hierarchical regressions to test hypotheses. Given the large number of hypotheses generated, the number of analyses was kept to a minimum by combining hypotheses regarding separate predictors into the same regression for each outcome variable.

## **Results**

### **Preliminary analysis**

*Computation of variables.* New variables were computed for risk, clinical comorbidity and therapeutic dose. Following collection of DSH and suicide attempt data from the SASII, each patient's risk score was coded using the following system: *severe risk* (DSH weekly and more than one suicide attempt in the past 12 months), *high risk* (DSH at least monthly and one suicide attempt in past 12 months), *moderate risk* (DSH monthly or more frequently but no suicide attempts in past 12 months), *low risk* (infrequent DSH no suicide attempts in past 12 months and *no risk* (no DSH and no suicide attempts in past 12 months). Change between pre- and post-treatment risk scores was then calculated (risk score at baseline minus risk score at treatment completion/dropout) to create one unique risk change variable where a higher score denoted a greater improvement in risk. Where patients' risk

was higher at completion of treatment or point of dropout this was indicated with a negative score. In order to measure all scores on a positive scale, a value of three was added to every risk change score (minus three being the lowest score obtained).

Number of PD diagnoses was calculated at both baseline assessment and at post-treatment/dropout. Change between the two was calculated for all patients (number of PD diagnoses at baseline minus number at treatment completion/dropout) to produce a final PD diagnosis change variable where a higher score indicated a greater improvement in number of diagnoses. Again, to avoid the issue of negative values where patients' number of diagnoses had increased, a new variable was computed which added a value of two to every risk change score (minus two being the lowest score obtained). The same process was followed for number of clinical syndrome diagnoses. In order to create one overall clinical comorbidity variable for the purpose of investigating the predictive value of comorbidity on treatment completion outcome, number of PD diagnoses was added to number of clinical syndrome diagnoses for all cases at baseline and post-treatment/dropout.

A final variable was computed for therapeutic dose/treatment completion which assigned a rank from one to five based on how many sessions patients completed and therapist identification of agreed completion of treatment (*did not complete assessment, assessed and offered treatment but declined or referred elsewhere, began treatment but attended less than eight sessions, began treatment and attended eight or more sessions but did not complete and completed treatment*).

*Normality checks of variables.* All variables were checked for basic assumptions of normality both within the sample as a whole and separately for completers and non-completers. The independent variables *therapeutic dose, therapist expertise* and *overall clinical comorbidity* as well as the dependent variable

*change in risk* were found to be skewed in all analyses with significant deviations from normality noted on the Kolmogorov-Smirnov test. The demographic variable *age* also showed negative skewness, marking a distribution significantly different from that of a normal distribution. For this reason, despite a small loss of power, it was decided that non-parametric tests would be used in correlational analyses involving these variables.

*Transformations and outliers.* Due to the significant skewness described above, square root and log transformations were tested, albeit without success. Outliers were examined by calculating standardised z-scores for all skewed variables and even after removal of three extreme cases, data was still found to represent a significant deviation from the normal distribution for most variables. The sample therefore remained complete for regression analyses and normality checks on residuals were conducted following analysis.

*Normality checks within regression models.* Hypothesis 1: logistical regression: standardised residuals were all within three standard deviations of the mean indicating no significant outliers so transformations or removal of outliers were unnecessary (n = 231). Hypothesis 2.1: multiple hierarchical regression: standardised residual z scores revealed two outliers both with a value less than minus three. Both cases were removed and the regression re-run (n = 229). This produced greater accounting of the variance by the predictors and a better fit of the model as well as a histogram more closely representing a normal distribution. Hypothesis 2.2: multiple hierarchical regression: standardised residual scores revealed no outliers greater than two standard deviations from the mean and the histogram of standardised residual scores looked broadly normal, therefore no outliers were removed, or transformations performed (n = 231). Hypothesis 2.3: simple regression: two outliers were revealed in standardised residual z scores, both more than three standard deviations above the mean. Following removal, a histogram appeared more normally distributed and the simple regression was re-run

(n = 229). When hypotheses 2.1, 2.2 and 2.3 were analysed using a sample consisting of treatment completers only (n = 170), again two outliers were revealed in standardised residual z scores, both more than three standard deviations above the mean. Following removal of these two outliers, a histogram appeared more normally distributed and the three multiple regressions were re-run (n = 168).

### **Hypothesis 1: Predictors of treatment completion**

Number of sessions attended ranged from zero to 165 and as can be seen in Table 4, clinical comorbidity was very similar between completers and non-completers. Baseline substance misuse was higher in non-completers than in completers.

Table 4  
*Mean number of sessions attended, clinical comorbidity and substance misuse for treatment completers and non-completers*

	Treatment completers (n = 170)	Non-completers (n = 61)
Mean number of sessions attended	56.7 (s. d. = 34.1)	5.5 (s. d. = 3.8)
Clinical comorbidity mean (number of PD diagnoses plus number of clinical syndrome diagnoses)	2.7 (s. d. = 1.4)	2.9 (s. d. = 1.4)
Percentage of patients using substances at baseline	46%	59%
Percentage of patients using substances at treatment completion/dropout	23%	54%

To test the hypothesis that baseline clinical comorbidity, therapist expertise and baseline substance misuse would predict treatment completion (completed verses did not complete), initial correlation of all variables was first explored to ensure that any significant relationships could be controlled as covariates. Table 5 shows a correlation matrix which illustrates a high correlation between baseline

Table 5

*Hypothesis 1: Predictors of treatment completion. Correlation matrix (Spearman's  $r$ ,  $n = 231$ )*

	Patient variables			Demographics		
	Baseline clinical comorbidity	Baseline substance misuse	Treatment completion	Age	Gender	Employment
Patient variables						
Baseline clinical comorbidity	—					
Baseline substance misuse	.152*	—				
Treatment completion	-.071	-.111	—			
Demographics						
Age	.084	-.072	.092	—		
Gender	.066	-.023	.067	-.016	—	
Employment at baseline	-.099	-.039	.006	-.051	-.033	—
Therapist variables						
Therapist expertise	-.081	.092	.138*	-.001	.010	-.119

*Note:* Treatment completion = completed (1)/ did not complete (2). Baseline clinical comorbidity = number of clinical syndrome diagnoses + number of PD diagnoses at baseline. Therapist expertise = adherent and highly skilful (4)/adherent and very experienced (3)/experienced (2)/novice (1).

\* $p < .05$ . \*\* $p < .01$ .

clinical comorbidity and baseline substance misuse ( $r = 0.15, p < .05$ ) which is to be expected because the SCID-I measure of clinical syndromes contains separate diagnostic categories which include substance misuse diagnoses. No association was found between either baseline clinical comorbidity and treatment completion ( $r = -.07, p = n. s.$ ), nor between baseline substance misuse and treatment completion ( $r = -.11, p = n. s.$ ), although an inverse relationship between both predictors and treatment completion existed confirming the hypotheses that clinical comorbidity and substance misuse form a negative relationship with completion outcome. Therapist expertise and treatment completion, however revealed a significant positive association ( $r = 0.14, p = .36$ ). There were no significant associations between any of the demographic variables and treatment completion, nor between any of the remaining predictors so these were not controlled as covariates in any further analyses.

The multicollinearity between the two predictors, baseline clinical comorbidity and baseline substance abuse is likely to pose a threat to the validity of multiple regression (Field, 2009) and the lack of significant association between both of these variables and treatment completion means that they are unlikely to contribute to the model's ability to accurately predict treatment completion. Therefore, a logistic regression was conducted using therapist expertise only which produced a model that accurately predicted treatment completion category 74% of the time (given that 74% of patients did in fact complete treatment). Table 6 presents the regression coefficients (beta values), their standard errors and the model's general statistics.

Table 6  
*Logistical regression exploring the role of therapist expertise in treatment completion*

	B (SE)	95% CI for exp <i>b</i>		
		Lower	exp <i>b</i>	Upper
Included				
Constant	-0.097 (0.59)	0.995	1.42	2.025
Therapist expertise	0.35 (0.18)			

*Note:*  $R^2 = .014$  (Hosmer & Lemeshow, calculated by *final* model Chi-Square divided by *original* model -2LL), .016 (Cox & Snell), .023 (Nagelkerke). Model  $\chi^2 (1) = 3.73$ . \* $p < .05$

The model showed that therapist expertise doesn't predict treatment completion well enough to significantly improve on a 74% prediction rate (Wald  $\chi^2(1) = 3.74$ ,  $p = .053$ , n. s.). Calculating an R-statistic<sup>6</sup> produced a partial correlation of  $R = 0.08$  between therapist expertise and treatment completion which suggests that although a positive relationship exists between therapist expertise and treatment completion (such that as therapist expertise increases, so does the likelihood of completing treatment), therapist expertise makes a very minor contribution to the model, representing a small effect size (Cohen, 1988) and falling just below the threshold of a significant Wald statistic. Further exploration of this association was warranted and due to non-normality of the therapist expertise variable, a Mann-Whitney U test was conducted, revealing a significant difference in therapist expertise between the two groups ( $U = 4321$ ,  $p = .036$ ), suggesting, as was evident from the Spearman's correlation, that therapist expertise has an impact on whether or not patients completed treatment.

To confirm this association, a Pearson's Chi-Square test was conducted which is designed to analyse two categorical variables. This test revealed a significant Pearson Chi-Square statistic demonstrating that therapist expertise and treatment completion are not independent and are therefore related in some way ( $\chi^2 = 8.144(3)$ ,  $p = .043$ ). In smaller samples where more than 20% of the expected frequencies are less than five (25% were less than five in the current analysis), it is conventional to report the Likelihood Ratio as well as the Pearson Chi-Square statistic, and accordingly, this Likelihood Ratio represents a slightly more significant effect ( $\chi^2 = 8.455(3)$ ,  $p = .037$ ), confirming the association between therapist expertise and treatment completion. In order to examine this relationship more closely, the treatment completion category variable (see *Computation of variables*, above) was tabulated

---

<sup>6</sup> R-Statistic calculated using the formula:  $R = \pm \sqrt{\frac{\text{Wald} - (2 \times df)}{-2LL(\text{original})}}$  (Field, 2009)



against therapist expertise, demonstrating that the greater the therapist's expertise, the more likely patients were to attend more sessions and complete treatment (Table 7).

Table 7  
*Frequencies (percentages) of treatment completion category by therapist expertise level*

Treatment completion category	Therapist Expertise			
	Novice	Experienced	Adherent and very experienced	Adherent and highly skilful
Did not complete assessment	0 (0%)	4 (1.7%)	2 (0.9%)	3 (1.3%)
Assessed and offered treatment but declined or referred elsewhere	0 (0%)	3 (1.3%)	10 (4.3%)	6 (2.6%)
Began treatment but attended less than 8 sessions	0 (0%)	3 (1.3%)	2 (0.9%)	6 (2.6%)
Began treatment and attended 8 or more sessions but did not complete	0 (0%)	4 (1.7%)	10 (4.3%)	8 (3.5%)
Completed treatment	1 (0.4%)	37 (16%)	43 (18.6%)	89 (38.5%)

Using overall baseline clinical comorbidity (number of clinical syndrome diagnoses plus number of PD diagnoses) as a proxy for patient complexity/clinical severity, it can be safely assumed that it was not the case that more complex patients were allocated to more expert therapists as a Spearman's correlation (see correlation matrix, Table 5) revealed that there was no significant association between baseline clinical comorbidity and therapist expertise ( $r = -.081$ ,  $p = n. s.$ ) and that in fact this was a negative relationship suggesting that actually as therapist expertise increased, patients' baseline clinical comorbidity decreased.

## **Hypothesis 2: Predictors of treatment outcome (whole sample)**

An initial Spearman's correlation was conducted to test associations between all variables. The correlation matrix in Table 8 shows a high correlation between gender and therapeutic dose ( $r = .179$ ,  $p < .01$ ) which is not surprising given the heavy weighting of the sample in favour of females. Females attended a mean of 47 sessions compared to a mean of 29 for males. Providing initial support for the hypotheses, strong associations were revealed between all three predictors and change in risk

Table 8

*Hypothesis 2.1, 2.2 and 2.3: Prediction of treatment outcome. Correlation matrix (Spearman's  $r$ ,  $n = 231$ )*

	Patient variables				Demographics				
	Therapeutic dose	Substance misuse	Comorbidity change–CS	Comorbidity change–PD	Risk change	Age	Gender	Employment	
Patient variables									
Therapeutic dose	—								
Baseline substance misuse	-.052	—							
Comorbidity change – C	.273**	.050	—						
Comorbidity change – PD	.336**	-.137*	.320**	—					
Risk change	.482**	-.150*	.106	.358**	—				
Age	.087	-.072	.043	.052	-.025	—			
Gender	.179**	-.023	.073	.070	.093	-.016	—		
Employment at baseline	-.026	-.039	-.038	.009	.009	-.051	-.033	—	
Therapist variables									
Therapist expertise	.083	.092	-.029	.147*	.167*	-.001	.010	-.119	

*Note:* Comorbidity change – CS = number of clinical syndrome diagnoses at baseline - number at treatment completion/drop out. Comorbidity change – PD = number of PD diagnoses at baseline – number at treatment completion/drop out. Risk change = risk score at baseline - risk score at completion of treatment/drop out. Therapist expertise = adherent and highly skilful (4)/adherent and very experienced (3)/experienced (2)/novice (1).

\* $p < .05$ . \*\* $p < .01$ .

(therapeutic dose  $r = .482$ ,  $p < .01$ ; therapist expertise  $r = .167$ ,  $p < 0.5$ ; baseline substance misuse  $r = -.150$ ,  $p < .05$ ) and all three predictors and change in PD diagnoses (therapeutic dose  $r = .336$ ,  $p < .01$ ; therapist expertise  $r = .147$ ,  $p < .05$ ; baseline substance misuse  $r = -.137$ ,  $p < .05$ ) including, as expected, a negative relationship between both of these outcome variables and baseline substance misuse, suggesting that as substance misuse increased, risk score and PD diagnosis change score both decreased, indicating greater risk and more PD diagnoses. Only therapeutic dose was found to be significantly associated with change in clinical syndrome diagnoses, however ( $r = .273$ ,  $p < .01$ ). No demographic variables were found to correlate significantly with change in risk, change in PD diagnoses or change in clinical syndrome diagnoses. Change in PD diagnoses and change in clinical syndrome diagnoses correlated significantly with each other ( $r = .320$ ,  $p < .01$ ) which is not surprising given that it is well-documented that many features of clinical syndrome diagnoses are characteristic in PD. Table 9 shows the changes in risk, PD diagnoses and clinical syndrome diagnoses for treatment completers and for the sample as a whole between baseline and treatment completion/dropout.

Table 9  
*Frequencies (percentages) of change in risk, change in PD diagnoses and change in clinical syndrome (CS) diagnoses for treatment completers and sample as a whole.*

	Treatment completers (n = 170)	Whole sample (n = 231)
Risk decreased from baseline	141 (83%)	144 (63%)
Risk remains the same from baseline	22 (13%)	77 (33%)
Risk increased from baseline	7 (4%)	10 (4%)
PD diagnoses decreased from baseline	84 (49%)	89 (39%)
PD diagnoses remained the same from baseline	81 (48%)	137 (59%)
PD diagnoses increased from baseline	5 (3%)	5 (2%)
CS diagnoses decreased from baseline	66 (39%)	100 (43%)
CS diagnoses remained the same from baseline	85 (50%)	108 (47%)
CS diagnoses increased from baseline	19 (11%)	23 (10%)

### *Hypothesis 2.1: Predictors of change in risk*

To test the hypothesis that therapeutic dose, therapist expertise and baseline substance misuse are predictive of change in risk, a multiple hierarchical regression was performed to see how much each predictor accounted for the variance in risk change. The correlation matrix (Table 8) shows that the highest correlation between change in risk and any of the predictor variables is with therapeutic dose ( $r = .482$ ,  $p < .01$ ) and this predictor was therefore entered first into the model before adding in therapist expertise and baseline substance misuse which were also both shown to be significantly correlated with risk change (substance misuse negatively as expected). After removal of outliers (see *normality checks within regression models*, above), the first model with just therapeutic dose produced an  $R^2$  of .151 (accounting for 15% of the variation in risk change), demonstrating a significant change from zero ( $F(1, 227) = 40.32$ ,  $p < .001$ ). When therapist expertise and baseline substance misuse were added to the model, the variance in risk change explained by the three predictors together increased to 19% ( $R^2 = .192$ ), representing a significant improvement on the previous model ( $F(2, 225) = 5.085$ ,  $p < .01$ ).

The first model using just therapeutic dose was significantly better at predicting outcome than the mean ( $F(1, 227) = 40.32$ ,  $p < .001$ ) as was the second model. All three predictors made a significant contribution to the model with therapeutic dose having the greatest impact ( $t(225) = 6.29$ ,  $p < .001$ ) followed by therapist expertise ( $t(225) = 2.52$ ,  $p = .012$ ) and baseline substance misuse ( $t(225) = -2.52$ ,  $p = .012$ ). It can therefore be concluded that although all variables did significantly predict change in risk, therapeutic dose was by far the greatest predictor explaining 15% of the total 19% variance. Table 10 shows the regression coefficients (beta values), their standard errors and standardised beta values for both models, highlighting significant values.

Table 10

*Multiple hierarchical linear regression exploring the role of therapeutic dose, therapist expertise and baseline substance misuse in the prediction of risk change outcome*

	B	SE B	$\beta$
Model 1			
Constant	3.92	0.16	
Therapeutic dose	0.02	0.003	.39**
Model 2			
Constant	3.16	0.44	
Therapeutic dose	0.02	0.003	.38**
Therapist expertise	0.32	0.13	.15*
Baseline substance misuse	-0.52	0.21	-.15*

Note:  $R^2 = .15$  for Model 1.  $\Delta R^2 = .04$  for Model 2. \* $p < .05$  \*\* $p < .001$

### *Hypothesis 2.2: Predictors of change in clinical comorbidity (PD diagnoses)*

To investigate the hypothesis that therapeutic dose, therapist expertise and baseline substance misuse predict change in number of PD diagnoses, a multiple hierarchical regression analysis was performed. As was the case with change in risk, the highest correlation between change in PD diagnoses and any predictor variable was with therapeutic dose ( $r = .336$ ,  $p < .01$ ) and this predictor was therefore entered first into the model before adding in therapist expertise and baseline substance misuse which were both also significantly associated with change in PD diagnoses (again, substance misuse showing a negative association, as expected). The first model using just therapeutic dose accounted for 8.5% of the variance in change in PD diagnoses ( $R^2 = .085$ ), a significant change from zero ( $F(1, 229) = 21.18$ ,  $p < .001$ ). Adding therapist expertise and baseline substance misuse to the model increased the variance in PD outcome explained by the predictors to 10.5% ( $R^2 = .0105$ ), not a significant improvement on the first model ( $F(2, 227) = 2.57$ ,  $p = .079$ , n. s.). Although both models were significantly better predictors of change in PD outcome than the mean (Model 1:  $F(1, 229) = 21.18$ ,  $p < .001$ ; Model 2:  $F(3, 227) = 8.87$ ,  $p < .001$ ), only therapeutic dose made a significant contribution to the model ( $t(227) = 4.51$ ,  $p < .001$ ) and neither therapist expertise ( $t(227) = 1.59$ ,  $p = .11$ , n.s.) nor baseline substance misuse ( $t(227) = -1.77$ ,  $p = .08$ ,

n. s.) did. Again, although all three variables were indeed predictors of change in PD diagnoses, therapeutic dose was by far the greatest predictor explaining 8.5% of the total 10.5% variance. Table 11 shows the regression coefficients (beta values), their standard errors and standardised beta values for both models, highlighting significant values.

Table 11  
*Multiple hierarchical linear regression exploring the role of therapeutic dose, therapist expertise and baseline substance misuse in the prediction of change in PD diagnoses*

	B	SE B	$\beta$
Model 1			
Constant	2.20	0.07	
Therapeutic dose	0.005	0.001	.21**
Model 2			
Constant	2.009	0.18	
Therapeutic dose	2.20	0.07	.28**
Therapist expertise	0.08	0.05	.10
Baseline substance misuse	-0.15	0.09	-.11

Note:  $R^2 = .085$  for Model 1.  $\Delta R^2 = .02$  for Model 2. \* $p < .05$  \*\* $p < .001$

*Hypothesis 2.3: Predictors of change in clinical comorbidity (clinical syndrome diagnoses)*

As noted, the correlation matrix in Table 8 shows that of the three predictor variables, only therapeutic dose was significantly associated with change in clinical syndrome diagnoses ( $r = .273$ ,  $p < .01$ ). A simple regression was therefore conducted to assess the impact of this single predictor on change in clinical syndrome diagnoses. After removal of outliers (see *normality checks within regression model*, above), the regression showed that therapeutic dose accounted for 7% of the variance in change in clinical syndrome diagnoses, a significantly better predictor than the mean ( $F(1, 227) = 18.24$ ,  $p < .001$ ) and a significant contributor to the model ( $t(225) = 4.27$ ,  $p < .001$ ). Although therapeutic dose was the only predictor associated with outcome in this hypothesis, it did not have as

great an impact on change in clinical syndrome diagnoses as it did on change in risk and change in PD diagnoses.

Table 12 shows the mean number of PD diagnoses and clinical syndrome diagnoses for completers and non-completers at baseline and treatment completion/dropout. Paired sample t-tests showed that the differences in mean number of diagnoses between baseline and treatment completion/dropout were significant for both completers (PD diagnoses:  $t(169) = 7.34, p < .001$ ; clinical syndrome diagnoses:  $t(169) = 9.88, p < .001$ ) and non-completers (PD diagnoses:  $t(60) = 2.19, p < .05$ ; clinical syndrome diagnoses:  $t(60) = 2.73, p < .05$ ), suggesting that there was some improvement for those who did not complete treatment, although it was not as great as for those who did.

Table 12  
*Change in clinical comorbidity. Mean number of PD and clinical syndrome diagnoses at baseline and at treatment completion/dropout for completers and non-completers*

Clinical comorbidity	Treatment completers (n = 170)	Non-completers (n = 61)
Mean number of PD diagnoses at baseline assessment	1.3 (s. d. = 0.5)	1.2 (s. d. = 0.4)
Mean number of PD diagnoses at treatment completion/dropout	0.7 (s. d. = 0.6)	1.1 (s. d. = 0.4)
Mean number of clinical syndrome diagnoses at baseline assessment	1.4 (s. d. = 1.2)	1.7 (s. d. = 1.3)
Mean number of clinical syndrome diagnoses at treatment completion/dropout	0.75 (s. d. = 0.9)	1.4 (s. d. = 1.0)

## **Hypothesis 2: Predictors of treatment outcome (treatment completers only)**

Exploratory analyses were re-run using treatment completers only to assess differences in prediction of outcome.

### *Change in risk*

A multiple regression showed that therapeutic dose, therapist expertise and baseline substance misuse together accounted for only 3.6% of the variance in risk scores in completers and that the model was not significantly better at predicting

change in risk than the mean ( $F(3, 164) = 2.07, p = .11, n.s.$ ). None of the three predictor variables made a significant contribution to the model (therapeutic dose:  $t(164) = 1.49, p = 0.14, n.s.$ ; therapist expertise:  $t(164) = 1.72, p = .09, n.s.$ ; baseline substance misuse:  $t(164) = -1.49, p = .014, n.s.$ ).

#### *Change in PD diagnoses*

A multiple regression showed that all three predictors accounted for only 3.8% of the variance in change in PD diagnoses and that the model was not significantly better at predicting change in risk than the mean ( $F(3, 164) = 2.19, p = .09, n.s.$ ). Therapeutic dose was the only predictor which made a significant contribution to the model ( $t(164) = 2.22, p < .05$ ) with neither therapist expertise ( $t(164) = 0.89, p = .38, n.s.$ ) nor baseline substance misuse ( $t(164) = -1.29, p = .2, n.s.$ ) having a significant impact.

#### *Change in clinical syndrome diagnoses*

A multiple regression showed that all three predictors together accounted for 6.3% of the variance in change in clinical syndrome diagnoses. The model was significantly better at predicting risk than the mean ( $F(3, 164) = 3.68, p < .05$ ) and again, therapeutic dose was the only predictor which made a significant contribution to the model ( $t(164) = 2.83, p < .01$ ) with neither therapist expertise ( $t(164) = -1.47, p = .14, n.s.$ ) or baseline substance misuse ( $t(164) = 0.34, p = .74, n.s.$ ) contributing significantly.

A sample consisting of just treatment completers, therefore, revealed that none of the variables accurately predicted change in risk and only therapeutic dose contributed to a regression model predicting change in number of PD diagnoses and change in number of clinical syndrome diagnoses. The model was slightly better at predicting change in clinical syndrome diagnoses than the other two outcome variables but still accounted for only a very small proportion of the variance in



change in number of clinical syndrome diagnoses. Table 13 presents the regression coefficients (beta values), their standard errors and standardised beta values for all three outcomes in treatment completers only, with significance values and R<sup>2</sup> figures incorporated.

Table 13

*Multiple linear regression exploring the role of therapeutic dose, therapist expertise and baseline substance misuse in the prediction of change in risk, change in PD diagnoses and change in clinical syndrome diagnoses in treatment completers only.*

	B	SE B	β
<b>Change in risk</b>			
Constant	4.35	0.55	
Therapeutic dose	0.005	0.003	.12
Therapist expertise	0.25	0.15	.13
Baseline substance misuse	-0.36	0.24	-.12
<i>Note: R<sup>2</sup> = .036. *p &lt; .05 **p &lt; .01</i>			
	B	SE B	β
<b>Change in PD diagnoses</b>			
Constant	2.22	0.26	
Therapeutic dose	0.004	0.002	.17*
Therapist expertise	0.06	0.07	.07
Baseline substance misuse	-0.02	0.11	-.10
<i>Note: R<sup>2</sup> = .038. *p &lt; .05 **p &lt; .01</i>			
	B	SE B	β
<b>Change in clinical syndrome diagnoses</b>			
Constant	2.75	0.41	
Therapeutic dose	0.007	0.003	.22**
Therapist expertise	-0.16	0.11	.14
Baseline substance misuse	0.06	0.18	.74
<i>Note: R<sup>2</sup> = .063. *p &lt; .05 **p &lt; .01</i>			

## Discussion

In agreement with previous similar studies (Kilem et al., 2010; Linehan et al., 2006a), the current study reported an overall drop out rate of 26%. Results showed that, as hypothesised, more experienced therapists were able to retain their patients in treatment longer than less experienced therapists. Although this finding is in agreement with previous anecdotal evidence and opinion (Andrews, 2001; Feigenbaum et al., 2011), similar research did not reveal the same effect (Franklin

et al., 2003) and there has been little evidence to date documenting the role of therapist expertise in treatment completion or outcome. One hypothesis for the current study's finding is that more expert therapists were better able to manage difficulties arising in early sessions, forging a more useful therapeutic alliance than less expert therapists. Quantification of therapist expertise will inevitably vary between researchers and studies as there is no universal method of defining this construct. Franklin et al. (2003) simply based their measure on number of years of experience as a practising therapist but other factors are likely to contribute such as quality of supervision, frequency and quality of training and level of difficulty attained in previous therapy cases. The current study attempted to incorporate all of these factors by using the clinical lead's observations from clinical supervision and knowledge of each therapist's achievements. Specifically, the use of the CTS-R (Blackburn et al., 2001) and expert knowledge of the DBT adherence rating scale (Linehan et al., 1996) and the DBT core competencies (available at [www.ucl.ac.uk/CORE](http://www.ucl.ac.uk/CORE)) reduced the possibility of subjective bias by introducing objective, validated rating scales. However, subjectivity was still an issue because of the clinical lead's prior involvement with the service and its therapists (see *Limitations*, below).

Patients who are defined as having more severe symptoms are likely to be allocated to more expert therapists which could potentially mask any effects of expertise (Franklin et al., 2003) although a non-significant association between baseline clinical comorbidity and therapist expertise revealed this not to be the case in the current study. In fact, although not a strong association, a negative relationship was revealed showing that more experienced therapists were actually more closely associated with patients with lower levels of clinical comorbidity. Moreover, due to the large geographical spread of the service, patients were allocated to therapists based on location which additionally mitigated against allocations based on expertise. In any case, there was still a large enough effect to

observe that patients stayed in treatment longer with more expert therapists. This is encouraging, demonstrating that even in this difficult-to-treat population, the best therapists are still able to retain patients long enough for them to attend as many as 165 sessions and in the majority of cases, to complete treatment. Given that therapeutic dose was revealed to be the primary predictor in outcomes of risk, PD diagnoses and clinical syndrome diagnoses, keeping patients engaged with and regularly attending the therapeutic process is extremely crucial in order for them to achieve the best outcomes (McMurrin et al., 2010).

Although previous research has shown that comorbid substance misuse leads to higher tendency to drop out from treatment (Karterud et al., 2003; Linehan et al., 1999) as does PD comorbidity (Feigenbaum et al., 2011) and that clinical comorbidity in general leads to worse outcomes for BPD treatment (Zanarini et al., 2006), the current study found that neither clinical comorbidity nor baseline substance misuse was significantly associated with treatment completion outcome. There was a higher percentage of baseline substance misuse in non-completers (59%) than in completers (46%) although this was not great enough to represent a significant statistical effect. Mean number of PD diagnoses, clinical syndrome diagnoses and overall clinical comorbidity was very similar between completers and non-completers at baseline so any pre-treatment differences were unlikely to have affected completion outcome. This suggests that other variables are more likely to play a role in whether or not patients complete treatment.

After establishing that patients receiving treatment with a more expert therapist are more likely to complete treatment, ascertaining how helpful completing treatment is was the next logical step. Using data for the whole sample, therapeutic dose, therapist expertise and baseline substance misuse were all found to be significant predictors of change in risk and change in PD diagnoses. Eighty three per cent of treatment completers and 63% of the whole sample had fewer DSH incidents and suicide attempts at the point of completion/dropout and 49% of completers saw

an improvement in their number of PD diagnosis following treatment. Therapeutic dose was the single predictor found to be responsible for most of the variance in these outcomes suggesting that the more sessions a patient attends the more likely they are to see an improvement in their incidents of DSH and suicide attempts and number of PD diagnoses. Again, therapeutic dose was found to be a significant predictor of change in clinical syndrome diagnoses, confirming the importance of retaining patients in treatment. As with treatment completion, having a more expert therapist predicted better outcomes in risk and PD diagnoses and in agreement with previous research (Ryle and Golyukina, 2000; Zanarini et al., 2003) baseline substance misuse functioned as a negative predictor contributing to increases in risk and number of PD diagnoses. These findings confirm the importance of facilitating patients' attendance and compliance with treatment. Replication of substance misuse as a factor which leads to worse risk and PD outcomes for patients confirms the need to routinely screen patients for this at initial assessment in order to ensure that it can be well-monitored and if necessary treated prior to beginning treatment to ensure treatment offered has the best chance of good outcomes.

Neither therapist expertise or baseline substance misuse were found to be significant predictors of change in clinical syndrome diagnoses and results showed that for both completers and non-completers, the majority of patients' number of clinical syndrome diagnoses did not change, echoing previous findings that these conditions are resistant to treatment in the presence of PDs (Reich & Vasile, 1993). Linehan et al.'s (1999) study of BPD treatment reported a baseline number of just over two and a half clinical syndrome diagnoses which was approximately one diagnosis more than the current study found. This suggests that clinical syndrome comorbidity was less severe in this sample than in previous samples and higher baseline levels might have been required to see a greater change with treatment. Therapists are trained explicitly in treating PD and although clinical syndromes are often comorbid (Afifi et al., 2011; Widom et al., 2009; Zanarini et al., 2006;

Zimmerman & Mattia, 1999), they were not the focus of treatment which is targeted at decreasing risk behaviour and symptoms characteristic of PD diagnoses.

There is some overlap between clinical syndrome diagnoses and substance misuse as substance misuse can be accurately defined as a clinical syndrome diagnoses in its own right. Substance misuse halved from baseline to end of treatment for treatment completers and reduced by about 20% for the sample as a whole which, although promising, does not represent major change, therefore it is not surprising that this did not have a significant effect on change in clinical syndrome diagnoses.

All three analyses were re-run using just treatment completers which revealed that neither regression model using all three predictors was significantly better at prediction than the mean for both change in risk and change in PD diagnosis outcomes. Standardised beta values were significant only in the models that included non-completers for these outcomes. This suggests that the model needs the variance in outcome in order to more accurately predict these changes and that a sample skewed more towards improvement makes it difficult to detect changes using therapeutic dose, therapist expertise and baseline substance misuse as predictors. Further research investigating outcome in just those who complete treatment would be worthwhile in order to replicate the importance of these predictors in risk and PD outcomes. Using completers only reduced the sample size by more than one quarter to 170 which may have compromised power although previous findings indicate that a sample size above 80 should still have had enough power to detect medium effects (Miles & Shevlin, 2001). Analysing treatment completers only did produce a model that was able to predict variance in clinical syndrome diagnosis outcome using therapeutic dose, therapist expertise and baseline substance misuse as predictors although therapeutic dose was the only predictor significantly contributing to the model. In treatment completers therefore, it

was possible to observe a greater effect of number of sessions attended on change in clinical syndrome diagnoses.

A noteworthy secondary finding was that in both completers and non-completers, a small proportion of patients' risk, PD diagnoses and clinical syndrome diagnoses increased. In non-completers this can be easily explained by the fact that they either dropped out before receiving treatment or received a dose so small that it did not produce a useful effect; being left untreated exposed them to increased risk and symptoms. However, across all three outcomes there were patients who completed treatment yet effectively got worse in terms of risk and number of diagnoses. It is well-documented that treatment can destabilise patients, increasing their risk behaviour as they struggle to find an appropriate outlet to express affect relating to distressing early experiences (e.g. Bateman & Fonagy, 2003). This could also result in an increase in symptoms of clinical syndromes such as anxiety and depression. It would not be surprising for patients to also then meet criteria for other PDs in these circumstances, especially given the fact that it is not uncommon to meet criteria for more than one PD diagnosis at a time (Shedler and Westen, 2007), or to possess enough traits of other PDs to just sit below the threshold for diagnosing that PD. It is also possible that by discharge assessment, therapists had better understanding and knowledge of their patients (including patients who attended only a few sessions before dropping out) and were therefore more able to accurately assess their PD and clinical diagnoses, and risk, than they were at baseline.

### **Limitations**

This study was limited by significant skewness in many variables, restricting its generalisability to wider samples. Age was skewed towards younger patients, ethnicity towards White British populations and gender heavily weighted in favour of females (although this is typical of BPD (American Psychiatric Association, 2013) which unsurprisingly made up the majority (~95%) of the current sample). There

was a significant association between gender and therapeutic dose and further examination revealed that females attended a third more sessions than males. It is well-documented that females engage more in help-seeking behaviour than males (e.g. Addis & Mahalik, 2003; Oliver, Pearson, Coe & Gunnell, 2005) so this is not unusual.

It was also a limitation that a larger sample size could not be obtained given the amount of data available spanning many more years than it was possible to include. This was largely due to missing and incomplete data which additionally meant that several measures were not included in the final analysis as patient data for them was too sparse. This resulted in reliance on clinician-rated measures and a lack of self-report information which would have contributed a more subjective dimension to the data (Uher et al., 2012). Dichotomising the sample into those who completed treatment and those who did not has been criticised for contributing to further loss of power due to losing variability in the data set (DeCoster, Iselin & Gallucci, 2009) which was perhaps an issue contributing to the null findings that were discovered when only treatment completers were analysed. Additionally, it is possible that the multiple statistical tests conducted on this data inflated the likelihood of Type I error and replication of these findings with a larger sample could address this, as well as possibly reducing skewness in several variables.

As mentioned, when carrying out a Pearson Chi-Square test (as was conducted to analyse the relationship between treatment completion and therapist expertise), if more than 20% of the expected frequencies are less than five, the test loses some of its power which can only be rectified by re-running the test using a larger sample. As this was not possible, the Likelihood Ratio was also reported which did reveal a more significant effect but it is likely that a stronger effect would have been exposed had a larger sample been obtained. This, again, highlights the need for replication with a greater sample size.

Computing the suicide attempt and DSH risk variable into a five point scale (*severe, high, moderate, low and no risk*) assumes that these categories form an interval scale and this variable was indeed treated as interval data for the purpose of calculating a risk change score and conducting subsequent regression analyses. However, it is possible that splitting the variable this way might be more representative of ordinal data and this may therefore have influenced the validity of the regression model.

Like other studies of treatment for PD, attrition was a problem and although this creates variation in the data that allowed for useful comparison between different levels of treatment completion, it was problematic to obtain reliable and valid estimates of substance use, risk and clinical comorbidity in patients who dropped out of treatment without warning. One way of addressing this would be to take measures every session rather than just pre- and post-treatment, however, this could interfere with development of the therapeutic alliance which has been shown to be an important mechanism of change in PD treatment (Bedics, Atkins, Comtois & Linehan, 2012; Turner, 2000), as well as a factor likely to predict better BPD treatment outcome (Barnicot et al., 2012). Therapeutic alliance also possibly contributes to retaining patients in treatment long enough to receive beneficial effects, as described. Additionally, completing measures regularly is a time-consuming process, particularly as the current study did not use self-report measures which could be independently completed in patients' own time.

Finally, an element of subjectivity was introduced in the ratings of therapist expertise because these ratings were completed by the clinical lead of the service whom, of course, had prior knowledge of each therapist's approximate dropout rate before providing expertise scores. It is possible that this information affected ratings, even if only subconsciously. This could have been avoided by using a blind assessor with no prior knowledge of the service and its therapists. This independent rater could have scored therapist expertise by listening to session recordings and



using core competency charts and standardised, validated expert rating scales. Unfortunately this was not possible within the limits of this project, however, upon replication, this should be considered.

### **Clinical implications**

Based on the finding that therapist expertise predicted treatment completion and that treatment completion (better attendance) as well as therapist expertise led to improved clinical outcomes, providing therapists with good supervision and relevant training and professional development opportunities is important. Findings from the first analyses present a problem in terms of using the data to predict which patients are more likely to complete treatment as neither of the patient variables were significantly related to completion outcome although it is noteworthy that both baseline clinical comorbidity and baseline substance misuse were negatively related to treatment completion suggesting that, albeit weakly, they were associated with non-completion. Should more convincing data be produced regarding these patient predictive factors, clinically, it could assist in decision-making regarding patients' suitability for long-term therapy or prior allocation to alternative interventions such as substance addiction programs to target comorbidity. The significance of good attendance on outcomes of risk and clinical diagnoses confirms the importance of promoting patients' engagement with their therapy in order that they have the best possible chance of achieving symptom improvement.

### **Future research**

Future replication clarifying the role of therapist expertise in completion and outcome of treatment would add to its sparse evidence base. It would be useful to clarify the role of comorbidity and particularly substance misuse in dropout as most studies have focused on treatment outcomes rather than completion. Previous research (Barnicot et al., 2011; McMurrin et al., 2010) has noted the role of patient

demographic variables in dropout and it would be useful to expand upon this. Replication with a larger sample would alleviate the potential for statistical errors and ensuring more equal groups of completers and non-completers would avoid introduction of further bias.

Findings demonstrated by the current study illustrate the importance of identifying factors which can predict not just treatment completion but treatment outcome. Importantly, therapeutic dose and substance misuse were demonstrated to be useful predictors in risk and PD diagnosis change and it would be worthwhile to investigate a variety of other patient factors involved in outcome such as employment status and resilience factors. Given the existing evidence base regarding clinical comorbidity and substance misuse it would be worthwhile to conduct more detailed investigations into the different diagnostic categories and how they interact with different PD diagnoses during the course of therapy, to alter outcome. The current sample was heavily weighted in favour of BPD which is widely accepted as the most common PD diagnosis (Coid et al., 2006; de Ruiter & Greeven, 2000), however it would be valuable to obtain larger samples of patients with primary PD diagnoses other than BPD in order to see if the findings revealed herein still stand. Finally, patients in the current study were equally distributed between CBT and DBT treatment and although research to date has not demonstrated differences in outcomes between these treatments (Brazier et al., 2006), using a variety of predictor variables among a larger sample may yield interesting results with regards to predicting what patient factors predict who is more likely to achieve better outcomes with which treatment.

## **Conclusion**

Although the current study did not identify any patient variables associated with treatment completion, results did demonstrate the importance of therapist expertise in retaining patients in therapy. Attending a higher number of sessions, having lower substance misuse at baseline and having a more experienced therapist predicted better outcomes with regards to reductions in DSH and suicide attempts and number of PD diagnoses. Better attendance predicted a reduction in number of comorbid anxiety, mood and other clinical disorders. There are several patient variables which future research should address in order to add to this evidence in predicting treatment completion and treatment outcome for personality disorder.

## References

Addis, M. E. & Mahalik, J. R. (2003). Men, masculinity and the contexts of help-seeking. *American Psychologist*, 58, 5-14.

Afifi, T. O., Mather, A., Boman, J., Fleisher, W., Enns, M. W., MacMillan, H., & Sareen, J. (2011). Childhood adversity and personality disorders: Results from a nationally representative population-based study. *Journal of Psychiatric Research*, 45, 814- 822.

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5<sup>th</sup> Edition). Arlington, VA: American Psychiatric Publishing.

American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders (Revised 4<sup>th</sup> Edition – Text Revision)*. Washington DC: American Psychiatric Publishing.

Andrews, H. B. (2001). Back to basics: Psychotherapy is an interpersonal process, *Australian Psychologist*, 36, 107-114.

Barnicot, K., Katsakou, C., Bhatti, N., Savill, M., Fierns, N. & Priebe, S. (2012). Factors predicting the outcome of psychotherapy for borderline personality disorder: a systematic review. *Clinical Psychology Review*, 32, 400-412.

Barnicot, K., Katsakou, C., Marougka, S., & Priebe, S. (2011). Treatment completion in psychotherapy for borderline personality disorder — A systematic review and meta-analysis. *Acta Psychiatrica Scandinavica*, 123, 327–338.

- Bateman, A.W. & Fonagy, P. (2008). 8-year follow-up of patients treated for borderline personality disorder: Mentalization-based treatment versus treatment as usual. *American Journal of Psychiatry* 165, 631-638.
- Bateman, A. W. & Fonagy, P. (2004). Psychotherapy for borderline personality disorder: Mentalization-based treatment. Oxford: Oxford University Press.
- Bateman, A. W. & Fonagy, P. (2003). The development of an attachment-based treatment program for borderline personality disorder. *Bulletin of the Menninger Clinic*, 67, 181-211.
- Bateman, A. W. & Fonagy, P. (1999). Effectiveness of partial hospitalization in the treatment of borderline personality disorder: a randomised controlled trial. *American Journal of Psychiatry*, 156, 1563-1569.
- Beck, A.T., Freeman, A. & Davis, D.D. (2004). *Cognitive therapy of personality disorders*. (2nd edition). New York: Guilford
- Bedics, J. D., Atkins, D. C., Comtois, K. A. & Linehan, M. M. (2012). Treatment differences in the therapeutic relationship and introject during a 2-year randomised controlled trial of dialectical behaviour therapy versus non-behavioural psychotherapy by experts for borderline personality disorder. *Journal of Consulting and Clinical Psychology*, 80, 66-77.
- Binks, C., Fenton, M., McCarthy, L. Lee, T., Adams, C. E. & Duggan, C. (2006). Psychological therapies for people with borderline personality disorder (Review). *Cochrane Database of Systematic Reviews*. Issue 1. Art. No.: CD005652. DOI: 10.1002/14651858.CD005652.

- Black, D. W., Allen, J., St John, D., Pfohl, B., McCormick, B. & Blum, N. (2009). Predictors of response to Systems Training for Emotional Predictability and Problem Solving (STEPPS) for borderline personality disorder: an exploratory study. *Acta Psychiatrica Scandinavia*, 120, 53–61.
- Blackburn, M., James, I. A., Milne, D. L., Baker, C., Standart, S., Garland, A. and Reichelt, K. (2001). The Revised Cognitive Therapy Scale (CTS-R): Psychometric properties. *Behavioural and Cognitive Psychotherapies*, 29, 431-446.
- Bloom, J. M., Woodward, E. N., Sasmaras, T., Pantalone, D. W. (2012). Use of dialectical behaviour therapy in inpatient treatment of borderline personality disorder: a systematic review. *Psychiatric Services*, 63, 881–888.
- Blum, N., Pfohl, B., St. John, D. S., Monahan, P. & Black, D. W. (2002). STEPPS: A cognitive behavioural systems-based group treatment for outpatients with borderline personality disorder—a preliminary report. *Comprehensive Psychiatry*, 43, 301–310.
- Bohus, M., Haaf, B., Stiglmayr, C., Pohl, U., Bohme, R. & Linehan, M. (1999). Evaluation of inpatient dialectical behaviour therapy for borderline personality disorder: A prospective study. *Behaviour, Research and Therapy*, 38, 875-887.
- Bowen R, South M, Fischer D, Looman T. (1994). Depression, mastery and number of group sessions attended predict outcome of patients with panic and agoraphobia in a behavioural/medication program. *Canadian Journal of Psychiatry*, 39, 283-288.

- Brazier, J. E., Tumor, I., Holmes, M., Ferriter, M. Parry, G., Dent-Brown, K. & Paisley, S. (2006). Psychological therapies including dialectical behaviour therapy for borderline personality disorder: a systematic review and preliminary economic evaluation. *Health Technology Assessment*, 10, 1-138.
- Butler, A., Chapman, J. E., Forman, E. M. & Beck, A. T. (2006). The empirical status of cognitive-behavioural therapy: A review of meta-analyses. *Clinical Psychology Review*, 26, 17-31.
- Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported treatments. *Journal of Consulting and Clinical Psychology*, 66, 7–18.
- Christo, G., Spurrell, S. & Alcorn, R. (2000). Validation of the Christo Inventory for Substance-misuse Services (CISS): a simple outcome evaluation tool. *Drug and Alcohol Dependence*, 59, 189–197.
- Clarkin, J., Levy, K. N., Lenzenweger, M. F., & Kernberg, O. F. (2007). Evaluating three treatments for borderline personality disorder: A multiwave study. *American Journal of Psychiatry*, 164, 922–928.
- Cohen, J. (1988). *Statistical power analysis for the behavioural sciences* (2<sup>nd</sup> ed). Hillsdale, New Jersey: Laurence Erlbaum Associates.
- Coid J., Yang M, Tyrer, P., Roberts, A., & Ullrich, S. (2006). Prevalence and correlates of personality disorder in Great Britain. *British Journal of Psychiatry*, 188, 423-431.

- Davidson, K., Norrie, J., Tyrer, P., Gumley, A. I., Tata, P., Murray, H., & Palmer, S. (2006). The effectiveness of cognitive behaviour therapy for borderline personality disorder: Results from the BOScot Trial. *Journal of Personality Disorders, 20*, 450–465.
- DeCoster, J., Iselin, A., & Gallucci, M. (2009). A conceptual and empirical examination of justifications for dichotomization. *Psychological Methods, 14*, 349-366.
- De Panfilis, C., Marchesi, C., Cabrino, C., Monici, A., Politi, V., Rossi, M. & Maggini, C. (2012). Patient factors predicting early dropout from psychiatric outpatient care for borderline personality disorder. *Psychiatry Research, 200*, 422-429.
- de Ruiter, C. & Greeven, P. J. G. (2000). Personality disorders in a Dutch forensic psychiatric sample: Convergence of interview and self-report measures. *Journal of Personality Disorders, 14*, 162-170.
- Dreessen, L. & Arntz, A. (1998). Short-interval test-retest inter-rater reliability of the Structured Clinical Interview for DSM-III-R personality disorders (SCID-II) in outpatients. *Journal of Personality Disorders, 12*, 138-48.
- Dulit, R. A., Feyer, G. R., Haas, M. L., Sullivan, T. & Frances, A. J. (1990). Substance use in borderline personality disorder. *American Journal of Psychiatry, 147*, 1002-1007.
- Feigenbaum, J. (unpublished). Cognitive behavioural and dialectical behavioural therapy for personality disorder: A naturalistic study of outcome. On-going research. North East London NHS Foundation Trust.



Feigenbaum, J., Fonagy, P., Pilling, S., Jones, A., Wildgoose, A. & Bebbington, P. (2011). A real-world study of the effectiveness of DBT in the UK National Health Service. *British Journal of Clinical Psychology*, 51, 121-141.

Feigenbaum, J. (2007). Dialectical behaviour therapy: An increasing evidence base. *Journal of Mental Health*, 16, 51-68.

Field, A. (2009). *Discovering statistics using SPSS*. 3<sup>rd</sup> Edition. London: SAGE.

First, M. B., Spitzer, R. L., Gibbon, M., and Williams, J. B. W. (2002). Structured Clinical Interview for DSM-IV-TR Axis I disorders, research version, patient edition. New York: Biometrics Research, New York State Psychiatric Institute.

First, M.B., Gibbon M., Spitzer, R.L., Williams, J.B.W. & Benjamin L.S. (1997). Structured Clinical Interview for DSM-IV Axis II personality disorders, (SCID-II). Washington, D.C.: American Psychiatric Press, Inc.

Franklin, M. E., Abramowitz, J. S., Furr, J. M., Kalsy, S. & Riggs, D. S. (2003). A naturalistic examination of therapist experience and outcome of exposure and ritual prevention for OCD. *Psychotherapy Research*, 13, 153-167.

Frieje, H., Dietz, B. & Appelo, M. (2002). Treatment of borderline personality disorder with the VERSE: skills emotion regulation disorder. *Directive Therapy*, 4, 367-378.

- Fonagy, P. & Bateman, A. (2006). Progress in the treatment of borderline personality disorder. *British Journal of Psychiatry*, 188, 1-3.
- Fonagy, P. (1999). Process and outcome in mental health care delivery: A model approach to treatment evaluation. *Bulletin of the Menninger Clinic*, 6, 288–304.
- Grapow, M. T. R., von Wattenwyl, R., Guller, U., Beyersdorf, F. & Zerkowski, H. (2006). Randomised controlled trials do not reflect reality: Real-world analyses are critical for treatment guidelines! *Journal of Thoracic and Cardiovascular Surgery*, 132, 5-7.
- Howard, K. I., Kofta, S. M., Krause, M. S. & Orlinsky, D. E. (1986). The dose-effect relationship in psychotherapy. *American Psychologist*, 41, 139-164.
- IBM Corp. (Released 2013). IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp.
- Kabat-Zinn, J. (1994). *Wherever You Go, There You Are: Mindfulness Meditation in Everyday Life*. New York: Hyperion.
- Karterud, S., Pedersen, G., Bjordal, E., Brabrand, J., Friis, S., Haaseth, Ø., Haavaldsen, G., Irion, T., Leirvåg, H., Tørum, E. & Urnes, Ø. (2003). Day treatment of people with personality disorders: Experiences from a Norwegian treatment research network. *Journal of Personality Disorders*, 17, 243–262.

- Kellogg, S. H. & Young, J. E. (2006). Schema therapy for borderline personality disorder. *Journal of Clinical Psychology*, 62, 445-458.
- Kliem, S., Kröger, C. & Kosfelder, J. (2010). Dialectical behaviour therapy for borderline personality disorder: A meta-analysis using mixed-effects modelling. *Journal of Consulting and Clinical Psychology*, 78, 936-951.
- Koons, C. R., Robins, C. J., Tweed, J. L., Lynch, T. R., Gonzalez, A. M., Morse, J. Q., Bishop, G. K., Butterfield, M. I., & Bastian, L. A. (2001). Efficacy of dialectical behaviour therapy in women veterans with borderline personality disorder. *Behaviour Therapy*, 32, 371 – 390.
- Leichsenring, F. (2004). Randomised controlled versus naturalistic studies: A new research agenda. *Bulletin of the Menninger Clinic*, 68, 137 – 151.
- Leichsenring, F. & Leibing, E. (2003). The effectiveness of psychodynamic therapy and cognitive behaviour therapy in the treatment of personality disorders: A meta-analysis. *American Journal of Psychiatry*, 160, 1223–1232.
- Linehan, M. M., Comtois, K. A., Murray, A. M., Brown, M. Z., Gallop, R. J., Heard, H. L., Korslund, K. E., Tutek, D. A., Reynolds, S. K., Lindenboim, N. (2006a). Two-year randomised controlled trial and follow-up of dialectical behaviour therapy verses therapy by experts for suicidal behaviours and borderline personality disorder. *Archives of General Psychiatry*, 63, 757-766.

- Linehan, M. M., Comtois, K. A., Brown, M. Z., Heard, H. L. & Wager, A. (2006b).  
Suicide Attempt Self-Injury Interview (SASII): Development, reliability, and  
validity of a scale to assess suicide attempts and intentional self-injury.  
*Psychological Assessment*, 18, 303-312.
- Linehan, M. M., Dimeff, L. A., Reynolds, K., Comtois, K. A., Shaw Welch, S.  
Heagerty, P. & Kivlahan, D. R. (2002). Dialectical behavior therapy versus  
comprehensive validation therapy plus 12-step for the treatment of opioid  
dependent women meeting criteria for borderline personality disorder. *Drug  
and Alcohol Dependence*, 67, 13-26.
- Linehan, M. M., Schmidt, H. Dimeff, L. A. Craft, J. C. Kanter, J. & Comtois, K. A.  
(1999). Dialectical Behaviour therapy for patients with borderline personality  
disorder and drug-dependence. *American Journal of Addictions*, 8, 279-292.
- Linehan, M. M., Lockard, J. S., Wagner, A. W., & Tutek, D. (1996). DBT expert  
rating scale. Unpublished manuscript, University of Washington, Seattle,  
WA.
- Linehan, M. M., Tutek, D. A., Heard, H. L & Armstrong, H. E. (1994). Interpersonal  
outcome of cognitive behavioural treatment for chronically suicidal borderline  
patients. *American Journal of Psychiatry*, 151, 1771-1776.
- Linehan, M. M. (1993). Cognitive-behavioural treatment of borderline personality  
disorder. New York: The Guilford Press.

- Links, P. S., Heslegrave, R. & van Reekum, R. (1998) Prospective follow-up study of borderline personality disorder: prognosis, prediction of outcome, and Axis II comorbidity. *Canadian Journal of Psychiatry* 43, 265– 270.
- Low, G., Jones, D., Duggan, C., Power, M. & Macleod, A. (2001). The treatment of deliberate self-harm in borderline personality disorder using dialectical behaviour therapy: A pilot study in a high security hospital. *Behavioural and Cognitive Psychotherapy*, 29, 85–92.
- Luborsky, L., Auerbach, A. H., Chauder, M., Cohen, J. & Bachrach, H. M. (1971). Factors influencing the outcome of psychotherapy: A review of quantitative research. *Psychological Bulletin*, 75, 145–185.
- McMurrin, M., Huband, M., & Overton, E. (2010). Non-completion of personality disorder treatments: A systematic review of correlates, consequences and interventions. *Clinical Psychology Review*, 30, 277–287.
- Miles, J. & Shevlin, M. (2001). *Applying regression and correlation: A guide for students and researchers*. London: Sage.
- National Institute for Health and Clinical Excellence. (2009). *Borderline Personality Disorder: Treatment and management*. CG78. London: National Institute for Health and Clinical Excellence.
- Oldham, J., Skodol, A., Kellman, H., Hyler, S., Doidge, N., Rosnick, L., & Gallagher, P. (1995). Comorbidity of Axis I and Axis II disorders. *American Journal of Psychiatry*, 152, 571-578.

- Oliver, M. I., Pearson, N., Coe, N. and Gunnell, D. (2005). Help-seeking behaviour in men and women with common mental health problems: Cross-sectional study. *British Journal of Psychiatry*, 186, 297-301.
- Öst, L. (2008). Efficacy of the third wave of behavioural therapies: A systematic review and meta-analysis. *Behaviour, Research and Therapy*, 46, 296-321.
- Panos, P., Jackson, J., Hasan, O. & Panos, A. (2013). Meta-analysis and systematic review assessing the efficacy of dialectical behaviour therapy. *Research on Social Work Practice*, 24, 213-223.
- Pistrang, N., Barker, C. & Elliott, R. (2002). Research methods in clinical and counselling psychology. An introduction for students and practitioners. West Sussex: John Wiley and Sons Ltd.
- Reich, J. H. & Vasile, R. G. (1993). Effect of Personality Disorders on the Treatment Outcome of Axis I Conditions: An Update. *Journal of Nervous and Mental Disease*, 181, 475-484.
- Robins, C. & Chapman, A. (2004). Dialectical behaviour therapy: Current status, recent developments and future directions. *Journal of Personality Disorders*, 18, 73-89.
- Roth, A. & Fonagy, P. (2005). What works for whom? A critical review of psychotherapy research. New York: Guilford.
- Rothwell, P. (2005). External validity of randomised controlled trials: To whom do the results of this trial apply?, *Lancet*, 365, 82–93.

- Ryle, A. & Golyukina, K. (2000). Effectiveness of time-limited cognitive analytic therapy of borderline personality disorder: Factors associated with outcome. *British Journal of Medical Psychology*, 73, 197-210.
- Sanislow, C. & McGlashan, T. (1998). Treatment outcome of personality disorders. *Canadian Journal of Psychiatry*, 43, 237-250.
- Seligman, M. (1995). The effectiveness of psychotherapy. The consumer reports study. *American Psychologist*, 50, 965-974.
- Shedler, J., & Westen, D. (2007). The Shedler–Westen Assessment Procedure (SWAP): Making personality diagnosis clinically meaningful. *Journal of Personality Assessment*, 89, 41-55.
- Skodol, A. E., Oldham, J. M. & Gallaher, P. E. (1999). Axis II comorbidity of substance use disorders among patients referred for treatment of personality disorders. *American Journal of Psychiatry*, 156, 733-738.
- Stoffers, J. M., Völlm, B. A., Rucker, G., Timmer, A., Huband, N. & Lieb, K. (2012). Psychological therapies for people with borderline personality disorder. *Cochrane Database of Systematic Reviews*, Issue 8. Art. No.: CD005652. DOI: 10.1002/14651858.CD005652.pub2.
- Stone, M. H., Hurt, S. W. & Stone, D. K. (1987) The PI-500: Long term follow-up of borderline in-patients meeting DSM-III criteria. I: Global outcome. *Journal of Personality Disorders*, 1, 291–298.

- Tarrier, N., Pilgrim, H., Sommerfield, C., Faragher, B., Reynolds, M., Graham, E. & Barrowclough, C. (1999). Randomized trial of cognitive therapy and imaginal exposure in the treatment of chronic posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 67, 13-18.
- Turner, R. M. (2000). Naturalistic evaluation of dialectical behaviour therapy-oriented treatment for borderline personality disorder. *Cognitive and Behavioural Practice*, 7, 413-419.
- Tyrer, P. & Ferguson, B. (2000). Classification of personality disorder. In P. Tyrer (Ed.) *Personality disorders: Diagnosis, management and course*. Oxford: Butterworth Heinemann.
- Tyrer, P., Seivewright, N., Ferguson, B., Murphy, S., Darling, C., Brothwell, J., Kingdon, D. & Johnson, A. L. (1990). The Nottingham Study of Neurotic Disorder: relationship between personality status and symptoms. *Psychological Medicine*, 20, 423-431.
- Uher, R., Perlis, R. H., Placentino, A., Dernovsek, M. Z., Henigsberg, N., Mors, O. Maier, W., McGuffin, P. & Farmer, A. (2012). Self-report and clinician-rated measures of depression severity: Can one replace the other? *Depression and Anxiety*, 29, 1043-1049.
- Verheul, R., van den Bosch, L. M. C., Koeter, M. W. J., de Ridder, M. A. J., Stijnen, T. & van den Brink, W. (2003). Dialectical behaviour therapy for women with borderline personality disorder: A 12-month, randomised clinical trial in the Netherlands. *British Journal of Psychiatry*, 182, 135-40.



Weinberg, I., Gunderson, J. G., Hennen, J. & Cutter, C. J. (2006). Manual assisted cognitive treatment for deliberate self-harm in borderline personality disorder patients. *Journal of Personality Disorders*, 20, 482–492.

Westen, D., Novotny, C. & Thompson-Brenner, H. (2004). The empirical status of empirically supported psychotherapies: Assumptions, findings, and reporting in controlled clinical trials. *Psychological Bulletin*, 130, 631-663.

Widom, C. S., Czaja, S. J., & Paris, J. (2009). A prospective investigation of borderline personality disorder in abused and neglected children followed up into adulthood. *Journal of Personality Disorders*, 23, 433-446.

Winston, A. P. (2000). Recent developments in borderline personality disorder. *Advances in Psychiatric Treatment*, 6, 211-217.

Zanarini, M. C., Frankenburg, F. R., Hennen, J., Reich, D. B., & Silk, K. R. (2006). Prediction of the 10-year course of borderline personality disorder. *American Journal of Psychiatry*, 163, 827-832.

Zanarini, M. C., Frankenburg, F. R., Hennen, J. & Silk, K. R. (2003). The longitudinal course of borderline psychopathology: 6-year prospective follow-up of the phenomenology of borderline personality disorder. *American Journal of Psychiatry*, 160, 274-283.

Zanarini, M.C., Skodol, A.E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., Morey, L.C., Grilo, C.M., Shea, M.T., McGlashan, T.H. and Gunderson, J.G. (2000). The collaborative longitudinal personality disorders study: Reliability of Axis I and II diagnoses. *Journal of Personal Disorders*, 14, 291-299.

Zanarini, M. C., Frankenburg, F. R., Dubo, E. D., Sickel, A. E., Trikha, A., Levin, A. & Reynolds, V. (1998). Axis I comorbidity of borderline personality disorder. *American Journal of Psychiatry*, 155, 1733-1739.

Zimmerman, M. & Mattia, J. I. (1999). Axis I diagnostic comorbidity and borderline personality disorder. *Comprehensive Psychiatry*, 40, 245-252.

## **Part 3: Critical Appraisal**

## **Introduction**

This critical review intends to identify and reflect upon some of the key issues that arose during the planning and implementation of this major research project. The main aspects to be considered are categorised under the following headings: joining an established research project, managing missing and incomplete data, the challenges of conducting research in a 'real-world' NHS personality disorder (PD) service and some general reflections on current issues within the field of PD research. Some recommendations and conclusions follow. It is hoped that discussion of these issues will aid future researchers investigating PD, highlighting where procedural improvements may be made to ensure the effectiveness and efficiency of future research.

### **Joining an established research project**

This project was appealing not just because of its subject matter which is of great interest, but because of the obvious benefits of joining a project for which large amounts of data had already been collected over a number of years. By the time of data collection, data was available for over 2,000 patients which is invaluable in terms of providing strong statistical power and revealing solid relationships from which to make clinically important inferences. Given the timescale of most doctoral research projects, collecting and accessing data over such a large time period would not have been possible unless much of the data was already collected.

This did mean, however, that the measures utilised by the service to collect data about its service users were fixed and non-negotiable, shaping the focus of the project towards specific research questions. This is not necessarily a disadvantage as measures were scientifically sound and reliable, comprising a battery which covered all aspects of behavioural change one might wish to investigate in a longitudinal study of PD therapy outcomes. The measures included in the wider

study struck a good balance between self-report questionnaires and clinician-rated scales and interviews, in accordance with recent guidance suggesting that it is important to obtain both subjective and objective ratings of psychopathology (Uher et al., 2012). However, due to missing data, no self-report measures were included in the final analyses meaning that the study relied solely on clinician-rated measures, although interviews did of course rely on patients' responses. The decision to exclude all self-report measures was made based upon how much data was missing for each measure and in all cases it was decided that incomplete data sets would cause too many difficulties with analysis, even with the aid of statistical techniques such as multiple imputation (Rubin, 2004; Sterne et al., 2009). There was scope to obtain rich information without the addition of other (mainly self-report) measures, although they doubtlessly would have added valuable input.

Unfortunately, joining an established project meant that there was sometimes little that could be done to rectify problems with incomplete data. This will be considered in more detail below, but did represent a significant challenge in terms of obtaining complete and reliable data for a satisfactory number of patients. This highlighted the importance of following recent best-practice guidance regarding keeping good research records (Schreier, Wilson & Resnick, 2006), detailing my role in the data collection and collation process in order that searching for missing data need not be a lengthier process than necessary and so that at the end of my involvement, future researchers are able to understand clearly what has already been established.

Although the benefits of having such a large amount of data readily available for analysis were clear, the challenge of transforming this data into something that could be usefully entered and analysed within the available time constraints meant that I often wondered whether it would have been easier to collect the data myself from the outset and this is a decision I would struggle to make should I begin the

project again with the benefit of hindsight. Collecting my own data would have avoided the problem of missing and incomplete information, but at the great expense of limiting the amount of data it would be possible to obtain, therefore reducing the validity of the clinical conclusions that could be drawn from the research.

### **Missing and incomplete data**

Missing data was without doubt the largest problem encountered during the research process and although this is by no means unusual or disastrous in social, behavioural and health science research (Graham, 2012), the majority of statistical techniques assume (or require) complete data, and in its absence most commonly default to the least desirable option: deletion of the entire case from the analysis (Osborne, 2013). Collecting data for the current study was a time-consuming and laborious process so not including valuable data was disappointing.

Although the disorganisation of the data was made clear at the commencement of my involvement in the project, it became apparent throughout the course of the research that in some cases the location of the data required for certain analyses was actually unknown. In terms of disorganisation, the first difficulty that occurred was that of questionnaires and interview measures that had not been correctly filed or labelled with a patient name or date and were thus unusable. Some data was labelled but not all measures from the standard test battery were present and complete. In this instance, it was possible in many cases to obtain the missing data by asking clinicians to search in other workspaces where data had been securely filed at earlier stages of the project. Where hard copies of the data could not be found, clinicians were often able to remember specific outcomes (for example, if patients had self-harmed following treatment completion or dropout, or whether they continued to meet diagnostic criteria for certain clinical syndromes and PDs). Where this was not an option, information could often be found on an

excellent electronic folder system on the services' shared drive which contained a file for each patient complete with assessment/discharge summaries and correspondence. In many cases it was fairly easy to obtain missing data this way, albeit a lengthy process. For the most part, baseline data was present and usually fairly complete; post-treatment data however, was harder to obtain, and is obviously crucial for calculating change. With strategic approaches, it was possible in most cases to obtain some post-therapeutic missing data but was nevertheless a time-consuming and tedious process.

In some cases it was difficult to ascertain whether data available in hard files was collected pre- or post-treatment and in these instances, and where demographic information had not been properly entered onto measures prior to completion of the assessment, the electronic care records system (RiO) was utilised to match dates to measures. Although this system contains a wealth of information, finding the specific information required was again a lengthy process, taking up valuable time that could have been put to better use collecting and entering data for a larger number of patients. The issue of missing data was the major factor responsible for the collection of a significantly lower amount of complete data sets than anticipated meaning that the total sample ultimately contained about seventy fewer data sets than anticipated. Additionally, as described above, several (mostly self-report) measures had to be removed from the analysis altogether as they were missing for too large a proportion of the final sample. Ultimately, it was possible to collect complete pre- and post-treatment data for six variables for a total of 231 patients and although interesting results were produced, there was potential for a much vaster and richer data set to be produced. A sample size of 600 or more would have been achievable with more relaxed time constraints and this would have had the power to detect small effects with three predictors (Miles and Shevlin, 2001) which might have made a difference, particularly where null findings were concerned.

## **Conducting research in an NHS ‘real-world’ personality disorder service**

A primary factor contributing to the difficulty in collecting a larger sample of complete data related to the challenge of collecting research within a ‘real-world’ NHS PD service. This service was set up for the treatment of people with PD and does not exist solely as a research unit. Its primary goal is therefore to ensure patients receive the best support and treatment for their needs. Any data that can be collected during this process is of course extremely valuable, but remains secondary to providing effective treatment. The service does, however recognise that in order to provide the best treatments, evidence documenting their effectiveness is crucial. Additionally, research provides the potential to be able to identify which patients are less likely to remain engaged and committed to the therapeutic process as well as who is likely to achieve better outcomes. This is vital information that can be used to economise resources by allocating patients to treatments they are most likely to benefit from and by facilitating improvements for those who are likely to dropout from treatment or achieve poor outcomes.

For this reason, research is valued highly by the service and recent NHS initiatives such as Payment by Results (Fairbairn, 2007) highlight the importance for therapists at an individual level to ensure that their work can be consistently and reliably outcomed. While clinical settings are the very best option for producing field validity in ‘real-world’ services, they also mean that therapists working in the service are employed primarily as clinicians, and although they may recognise their skills as scientist practitioners who are partly responsible for the evaluation of their treatments, they may have felt quite threatened by a research presence analysing data which has the ability to assess individual therapists’ outcomes. Of course, this highlights strengths but it might also have felt intrusive and anxiety-provoking, possibly resulting in some therapists’ lack of cooperation with the research process.

Unsurprisingly, not all therapists working at the service remained there for the duration of the research period and a busy clinical setting meant that multiple



researchers and therapists were involved with the project at different times, working differently and inconsistently and thus impacting the quality of the data. Additionally, busy therapists rightly prioritised writing thorough and useful clinical notes, reports and correspondence meaning that scoring measures and interviews was neglected. Rating and interpreting data therefore added to the lengthy process of translating patient information and measures into useful, usable data.

As is typical within a population of patients receiving treatment for PD, many patients dropped out before the assessment or treatment was complete (Fonagy & Bateman, 2006), thus inflating gaps in the data that could not always be reliably filled. Attrition was therefore a problem although it is fairly common in longitudinal research involving follow-up of patients who entered treatment several months previously and is very common in treatment for PD (Barnicot, Katsakou, Marougka & Priebe, 2011; Clarkin, Levy, Lenzenweger & Kernberg, 2007; Feigenbaum et al., 2011). Those who dropped out of treatment made up one quarter of the total sample and further exploratory analyses revealed that without their data to provide variation for comparisons, many statistical effects were lost, demonstrating that it was worthwhile to continue to seek further data even for those patients who dropped out prematurely.

Roth and Fonagy (2005) refer to the laboratory versus naturalistic research distinction as *research efficacy* versus *clinical effectiveness*. The strengths and weaknesses of both methods contribute to great debate in psychotherapy research (Fishman, 2000). Despite the aforementioned difficulties encountered when conducting research in a 'live' clinical setting, the benefits are great. As mentioned, this provides the most ecologically valid setting for collecting information about how services work, who they treat, how they treat and what the outcomes are. A wealth of information exists on a vast number of patients which can be analysed according to the service's interests and hypotheses. In terms of research efficacy, a trial set up purely for research purposes may boast tighter control of extraneous variables,

fewer incomplete data sets and closer adherence to important ethical procedures but would lack field validity. It is this ability to investigate what actually happens when treatments are implemented in a 'real-world' service exposed to the pitfalls and challenges of a tightly-resourced NHS that is most valuable (Binks et al., 2006; Feigenbaum et al., 2011). The validity of the data obtained in the current project is therefore extremely high and this goes some way towards offsetting the aforementioned limitations.

### **Current issues in PD research**

One of the major factors affecting research and practice within the field of PD currently is how PD is classified, and this was evident when conducting the literature review and empirical project. In categorising patients with PD for research trials, comorbidity within PDs becomes problematic. Twenty per cent of the current study's sample met criteria for more than one PD at baseline assessment. This has been a major criticism of the categorical approach to classifying PD (Shedler & Westen, 2007; Westen & Shedler, 1999) with estimates of PD comorbidity using the Structured Clinical Interview for DSM-IV-Axis II (SCID-II; First, Gibbon, Spitzer, Williams & Benjamin, 1997) ranging between an additional four to six PD diagnoses per patient (Bell & Jackson, 1992; Morey, 1988; Oldham et al., 1992). PD comorbidity is important as it has been reported to be associated with poorer treatment outcomes (Zanarini, Frankenburg, Hennen & Silk, 2006) and poses difficulties when treatment effects need to be analysed separately for different PDs. It therefore remains a challenge for future researchers to clarify complex comorbid relationships within PD (Links, 2007).

Very recently, the Diagnostic and Statistical Manual of Mental Disorders (DSM-5, American Psychiatric Association, 2013) underwent major changes. The new DSM-5 retained the ten subtypes of PD classified under separate categorical

definitions, deeming them easiest to use in clinical practice (American Psychiatric Association, 2013). However, prior to its publication, much speculation existed as to whether or not DSM-5 would switch to a more dimensional or combined dimensional/categorical approach where PDs are additionally considered on a spectrum or continuum of 'normal' behaviour (Rounsaville et al., 2002). This was thought to be preferable to a purely categorical approach which uses arguably arbitrary cut-off points to define a PD (for example, a diagnosis of BPD may be made if someone meets five out of nine of the BPD traits listed in the diagnostic criteria, but not if they only meet four). A considerable amount of evidence exists to support a more dimensional approach (Clark, 2007; Livesley, 2007; Widiger, Livesley & Clark, 2009; Widiger & Trull, 2007) and Morey and Hopwood (2013) argue that a dimensional approach has the potential to capture PD traits more succinctly using transdiagnostic dimensions that straddle the different possible PD subtypes, such as the impulsivity that is seen in both borderline and antisocial PD (e.g., Clark 2007; Krueger et al. 2011). A dimensional approach would also go some way towards rectifying the aforementioned problems associated with clinical comorbidity.

The DSM-5 (American Psychiatric Association, 2013) also combined DSM-IV-TR's (American Psychiatric Association, 2000) Axes I and II into one single Axis, grouping PD alongside other clinical syndromes such as anxiety and depression. By contrast, this change could be viewed in a positive light, meaning that PD may no longer be viewed as markedly different from other mental disorders, meeting the aim of the National Institute for Mental Health in England's (2003) guidance that it need not be a diagnosis of exclusion. In time, this may help patients to cope with the stigma of their diagnosis. The diagnosis 'personality disorder' is not considered to be a particularly helpful term (Robertson & Coccia, 2007) and a reduction in the negative associated effects of this label may be useful in facilitating patients' compliance with their treatment which the current study has proved leads to

improvements in risk and diagnostic outcomes. Moreover, classifying PDs on one Axis along with clinical syndromes suggests that they are now considered treatable. This is in stark contrast to their previous grouping on the former Axis II along with the more untreatable category of 'mental retardation'.

### **Implications and recommendations**

The Improving Access to Psychological Therapies for Severe Mental Illness initiative (IAPT SMI, 2013) makes this a particularly salient time for research into PD treatment outcomes and it is crucial that this research continues to promote its importance within the field of mental health. Identifying mechanisms of change, not just within DBT and CBT treatment, but also in newer treatments for PD such as Mentalization Based Therapy (MBT; Bateman & Fonagy, 2008; Bateman & Fonagy, 2004) and Schema Therapy (Kellogg & Young, 2006) is vital in educating therapists about why their treatments work so that they can target and emphasise those mechanisms that have been evidenced to produce the greatest therapeutic change. Continued evidence documenting the specific mechanisms that result in the effectiveness of CBT, DBT and newer treatments for PD will assist in securing their rightful place among the next edition of NICE guidance for PD, attracting the funding required to continue providing such valuable services.

It is recommended in this difficult financial time that future researchers focus their attentions on identifying those patients that are less likely to complete treatment or to achieve positive outcomes so that more effective alternatives to existing interventions can be sought. The current study highlighted factors associated with positive treatment outcomes as therapist expertise, good attendance and lower levels of substance misuse and this is an important step, not only because previous findings were confirmed (e.g. the role of substance misuse in treatment outcome) but because novel associations were revealed that warrant

further investigation: although it is a limitation that the current study was unable to identify patient factors involved in the completion of treatment, it did reveal the role of therapist expertise in retaining patients in treatment. Services should offer regular training and encourage professional development of therapists to ensure they have the chance to improve their skills. Replication of current findings would strengthen the case for services to focus resources on promoting these factors in order to be more confident of achieving positive outcomes for those most likely to benefit.

The current project found no significant effect of gender on whether or not treatment was completed (although women did attend more sessions than men). Gender was also not associated with change in risk outcome, change in number of PD diagnoses or change in number of clinical syndrome diagnoses. The current National Institute for Health and Clinical Excellence (NICE; 2009) guidance recommends DBT for women only and with further evidence documenting the effectiveness of both CBT and DBT for men too it is possible that future recommendations could be reconsidered to include evidence regarding the effectiveness of treatment for both genders.

## **Conclusions**

It is of course, extremely important for PD treatment services to be based on the most up to date evidence, and for this evidence to be widely disseminated for maximum advantage. Despite a lower than desired final sample size and difficulties with incomplete data meaning that some analyses were not possible, this project makes a vital contribution to the current PD evidence base, providing 'real-world' information on the factors that influence early disengagement with treatment as well as the factors that predict successful and unsuccessful clinical outcomes. This information means that resources can be most usefully deployed where they are most likely to have a positive impact on treatment completion and outcome.

## References

- American Psychiatric Association (2000). *Diagnostic and Statistical Manual of Mental Disorders (Revised 4<sup>th</sup> Edition – Text Revision)*. Washington DC: American Psychiatric Publishing.
- American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders (5<sup>th</sup> Edition)*. Arlington, VA: American Psychiatric Publishing
- Barnicot, K., Katsakou, C., Bhatti, N., Savill, M., Fierns, N. & Priebe, S. (2012). Factors predicting the outcome of psychotherapy for borderline personality disorder: a systematic review. *Clinical Psychology Review*, 32, 400-412.
- Bateman, A.W. & Fonagy, P. (2008). 8-year follow-up of patients treated for borderline personality disorder: Mentalization-based treatment versus treatment as usual. *American Journal of Psychiatry* 165, 631-638.
- Bateman, A. W. & Fonagy, P. (2004). *Psychotherapy for borderline personality disorder: Mentalization-based treatment*. Oxford: Oxford University Press.
- Bell, E. & Jackson, D. The structure of personality disorders in DSM-III. *Acta Psychiatrica Scandinavica*, 85, 279–287.
- Binks, C., Fenton, M., McCarthy, L. Lee, T., Adams, C. E. & Duggan, C. (2006). Psychological therapies for people with borderline personality disorder (Review). *Cochrane Database of Systematic Reviews*. Issue 1. Art. No.: CD005652. DOI: 10.1002/14651858.CD005652.

- Clark, L. A. (2007). Assessment and diagnosis of personality disorder: Perennial issues and an emerging reconceptualization. *Annual Review of Psychology*, 57, 277-257.
- Clarkin, J., Levy, K. N., Lenzenweger, M. F., & Kernberg, O. F. (2007). Evaluating three treatments for borderline personality disorder: A multiwave study. *American Journal of Psychiatry*, 164, 922–928.
- Fairbairn, A. (2007). Payment by results in mental health: the current state of play in England. *Advances in Psychiatric Treatment*, 13, 3–6.
- Feigenbaum, J., Fonagy, P., Pilling, S., Jones, A., Wildgoose, A. & Bebbington, P. (2011). A real-world study of the effectiveness of DBT in the UK National Health Service. *British Journal of Clinical Psychology*, 51, 121-141.
- First, M.B., Gibbon M., Spitzer, R.L., Williams, J.B.W. & Benjamin L.S. (1997). Structured Clinical Interview for DSM-IV Axis II personality disorders, (SCID-II). Washington, D.C.: American Psychiatric Press, Inc.
- Fishman, D. B. (2000). Transcending the efficacy versus effectiveness research debate: Proposal for new, electronic “Journal of pragmatic case studies”. *Prevention and Treatment*, 3, No Pagination Specified.
- Fonagy, P. & Bateman, A. (2006). Progress in the treatment of borderline personality disorder. *British Journal of Psychiatry*, 188, 1-3.
- Graham, J. W. (2012). Missing Data: Analysis and Design. [Statistics for Social and Behavioural Sciences]. E-books: Springer. Retrieved from [www.springer.com](http://www.springer.com)

IAPT (2013). How do we make psychological therapies more available and effective for people with severe mental illness? SMI Conference. 7<sup>th</sup> March 2013. London.

Kellogg, S. H. & Young, J. E. (2006). Schema therapy for borderline personality disorder. *Journal of Clinical Psychology*, 62, 445-458.

Krueger, R. F., Eaton, N. R., Clark, L. A., Watson, D., Markon, K. E., Derringer, J. & Livesley, W. J. (2011). Deriving an empirical structure of personality pathology for DSM-5. *Journal of Personality Disorders*, 25, 170–191.

Links, P. S. (2007). Impact of Recent Research on Borderline Personality Disorder. *Current Psychiatry Reports*, 9, 1-2.

Livesley, W. L. (2007). A framework for integrating dimensional and categorical classification of personality disorder. *Journal of Personality Disorders*, 21, 199-224.

Miles, J. & Shevlin, M. (2001). Applying regression and correlation: A guide for students and researchers. London: Sage.

Morey, L. C., & Hopwood, C. J. (2013). Stability and change in personality disorders. *Annual Review of Clinical Psychology*, 9, 499-528.

Morey, L. C. (1988). Personality disorders in DSM-III and DSM-III-R: Convergence, coverage, and internal consistency. *American Journal of Psychiatry*, 145, 573–577.



National Institute for Health and Clinical Excellence. (2009). *Borderline Personality Disorder: Treatment and management*. CG78. London: National Institute for Health and Clinical Excellence.

National Institute for Mental Health in England (2003). *Personality disorder: No longer a diagnosis of exclusion. Policy implementation guidance for the development of services for people with personality disorder*. Gateway Ref: 1055.

Oldham, J. M, Skodol, A. E, Kellman, H. D, Hyler, S. E, Rosnick, L & Davies, M. (1992). Diagnosis of DSM-III-R personality disorders by two structured interviews: patterns of comorbidity. *American Journal of Psychiatry*, 149, 213–220.

Osborne, J. W. (2013). *Best Practices in Data Cleaning: A Complete Guide to Everything You Need to Do Before and After Collecting Your Data*. Louisville, KY: Sage.

Robertson, K. & Coccia, F. (2007). Personality disorder – a stigmatising diagnosis? *Psychiatric Bulletin*, 31, 194.

Roth, A. & Fonagy, P. (2005). *What works for whom? A critical review of psychotherapy research*. New York: Guilford.

Rounsaville, B. J., Alarcon, R. D., Andrews, G., Jackson, J. S., Kendell, R. E., & Kendler, K. (2002). Basic nomenclature issues for DSM–V. In D. J. Kupfer, M. B. First, & D. E. Regier (Eds.). *A research agenda for DSM–V* (p. 1–29). Washington, DC: American Psychiatric Association.

Rubin, D. B. (2004). Multiple imputation for non-response in surveys. Hoboken, New Jersey: John Wiley and Sons.

Schreier, A. A., Wilson, K. & Resnik, D. (2006). Academic research record-keeping: best practices for individuals, group leaders and institutions. *Academic Medicine: Journal of the Association of American Medical Colleges*, 81, 42-47.

Shedler, J., & Westen, D. (2007). The Shedler–Westen Assessment Procedure (SWAP): Making personality diagnosis clinically meaningful. *Journal of Personality Assessment*, 89, 41-55.

Sterne, J. A. C., White, I. R., Carlin, J. B., Spratt, M., Royston, P., Kenward, M. G., Wood, A. M. & Carpenter, J. R. (2009). Multiple imputation for missing data in epidemiological and clinical research: potential and pitfalls. *British Medical Journal*, 338:b2393.

Uher, R., Perlis, R. H., Placentino, A., Dernovsek, M. Z., Henigsberg, N., Mors, O., Maier, W., McGuffin, P. & Farmer, A. (2012). Self-report and clinician-rated measures of depression severity: Can one replace the other? *Depression and Anxiety*, 29, 1043-1049.

Westen, D. & Shedler, J. (1999). Revising and assessing Axis II, part I: Developing a clinically and empirically valid assessment method. *American Journal of Psychiatry*, 156, 258–272.

Widiger, T. A., Livesley, W. L. & Clark, L. A. (2009). An integrative dimensional classification of personality disorder. *Psychological Assessment*, 21, 243-255.

Widiger, T. A. & Trull, T. J. (2007). Plate tectonics in the classification of personality disorder: Shifting to a dimensional model. *American Psychologist*, 62, 71-83.

Zanarini, M. C., Frankenburg, F. R., Hennen, J., Reich, D. B., & Silk, K. R. (2006). Prediction of the 10-year course of borderline personality disorder. *American Journal of Psychiatry*, 163, 827-832.

# Appendices

# **Appendix A**

## **Criteria for Critical Appraisal of Studies**

**(Downs and Black, 1998)**

## **Appendix B**

### **Ethical Approval Letters**

## **Appendix C**

### ***Christo Inventory for Substance- misuse Services***

**(CISS; Christo, Spurrell & Alcorn, 2000)**