

**A Study of the Relationship between Borderline-dysregulated Personality and Treatment-resistant Depression in the Course of the TADS Randomised Controlled Trial**

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### Abstract

**Aim:** This research project explored the relationship between borderline personality disorder, difficult to treat depression and treatment outcome. **Method:** The study used data collected in the course of the Tavistock Adult Depression Study (TADS). The TADS was a randomised controlled trial comparing Long Term Psychodynamic Psychotherapy (LTPP) to Treatment as Usual (TAU) for 129 patients whose depression has not been successfully treated by at least two previous interventions. The author of this project took part in rating all patients with the Shedler-Westen Assessment Profile (SWAP-II) – a 200-item personality measure – at the beginning and end of treatment. Statistical analysis explored the relationship between patients' borderline personality scores at the beginning and end of treatment, and their progress in treatment in terms of decrease in depression severity, and psychological distress, and increase in general functioning. In addition, changes in borderline personality features were also explored in relation to changes on the three main outcome measures. The author included the rest of the SWAP-II personality scales in the analysis, too, as the SWAP-II generates an overall personality profile. **Results:** Only a small number of patients reached cut-off scores for borderline personality disorder or features. When the SWAP-II borderline scale was entered in analysis as a continuous variable, no significant link was found between borderline personality scores at the beginning of treatment, and treatment outcome. The way borderline features changed in the course of treatment, however, was significantly related to the outcome measures. This was particularly the case for SWAP-II items indicating insecure attachment and affect dysregulation. In addition, patients in the LTPP group who presented with borderline personality features or disorder at the end of treatment were more likely to still experience severe or very severe depression at the end of therapy.

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*To my family.*

*“Words are instruments that people are free to adapt to any use, provided they make clear their intentions.”*

*Claude Lévi-Strauss*

This thesis is about treatment-resistant depression, borderline personality disorder and treatment outcome, and the link between the three. A neat and succinct introduction might start by providing a definition of each of these three terms, before launching into exploring if and how they are connected. Yet, as argued by the French anthropologist Claude Lévi-Strauss above, people use language as means to their varied goals.

The *raison d'être* of mental health practice and research is, as many would agree, to alleviate human suffering and provide support to those presenting with difficulties that stop them from living a fulfilling life. In order to devise and implement effective interventions in a climate of growing population and demand, and shrinking resources, clinicians and researchers need to go beyond the level of the individual narratives of their patients, and gain deeper knowledge about trends and patterns, processes and mechanisms, so that they can make reliable predictions on a bigger scale and ensure quality of services. A common language, allowing a multitude of patients and professionals to develop such body of knowledge, is inevitably needed for purposes of communication and sharing meaning. Such language is, however, the product of multiple and ever-evolving social and political processes, professional discourses and conflicting allegiances (McPherson & Armstrong, 2006). Furthermore, it is often tainted by interests and priorities different to what is, or at least should be, at the core of mental health practices – helping those in need of help (Horwitz, 2012).

In the first section of this chapter the author will present the main concepts related to depression and its treatment in mental health research and practice today. Whilst a detailed genealogy of all terms used in this thesis is far beyond its purpose and scope, the author considers it incumbent upon her to signpost the reader to the most relevant contradictions and debates related to

conceptualising depression and its treatment. Only after the broader setting is thus introduced, the lens can zoom in, in order to examine the definition and treatment of “treatment-resistant depression”, linking it to “borderline personality disorder features” and introducing the aims of the current research at the end of the chapter.

### **1.1. Major Depression – Current Definitions and Debates**

The World Health Organisation estimates that globally 350 million people of all ages suffer from depression (World Health Organization, 2015). Furthermore, it is estimated that 50% of those experiencing depression do not receive treatment for it, and that many people are misdiagnosed. Depression has been predicted to become the second largest cause for disability worldwide by 2020 (Murray & Lopez, 1997).

#### **1.1.1. Major depressive disorder – definition**

The psychiatric communities in Western societies (Europe and the USA) have adopted two main mental disorders classification and diagnostic manuals, both of which are updated periodically. These are the International Classification of Diseases (ICD), currently in its 10<sup>th</sup> Edition (World Health Organization, 1992) and the Diagnostic and Statistical Manual of Mental Disorders (DSM), currently in its 5<sup>th</sup> Edition (APA, 2013).

The term “major depression” was first introduced in the 3<sup>rd</sup> edition of the DSM (Spitzer, 1989), and has been in use in all subsequent editions of the Manual, but has not been adopted by the ICD. It has been argued that the DSM has been more accessible to psychiatric communities in the USA and beyond, than the ICD, due to the APA’s greater budget allowing for larger-scale production, dissemination and training in the use of the manual (McPherson & Armstrong, 2006). The term “major depression” has thus become widely accepted and used by mental health professionals, and has guided the clinical management of huge numbers of patients, and the creation of successive policies and guidelines in the field (e.g. National Collaborating Centre for

Mental Health (Great Britain) & Royal College of Psychiatrists, 2010).

The DSM-5 classifies Major Depressive Disorder (MDD) as the presence of five or more of a list of nine groups of symptoms, for a duration of at least two weeks, where a change to previous functioning is observed, the symptoms cause the individual significant distress and/or impairment of functioning, and the symptoms are not attributable to other psychiatric disorders, substance misuse or another medical condition (APA, 2013). In addition, for MDD to be diagnosed, the person should not have history of mania or hypomania, and, if significant loss such as a bereavement is experienced, the symptoms should be of an intensity beyond “the normal response to a significant loss” (APA, 2013, p. 161). The 12-month prevalence of MDD in adults has been estimated to be approximately 7% in the United States (ibid) and 6.9% in Europe (Bschor, Bauer, & Adli, 2014).

### **1.1.2. Current MDD debates and criticisms**

Key points of criticism of the DSM MDD diagnostic criteria are the seemingly arbitrary number of symptoms and their duration required to obtain diagnosis, and the lack of lifetime diagnostic stability of MDD (Demyttenaere, Van Oudenhove, & De Fruyt, 2005). In one study, for example, where 431 patients with an MDD diagnosis were followed for a period of up to 12 years, weekly analysis of their symptoms revealed a significant fluctuation of severity and duration of symptoms over time; furthermore, 23% of patients who were followed-up for at least two years were never symptom-free for more than a week, which suggests a marked MDD chronicity in the sample (Judd et al., 1998a).

Another critical argument highlights that, unlike medicine, where classification relies to a large extent on observable physical characteristics, psychiatry has to elicit and interpret patterns of symptoms for the purposes of deriving a classification; this renders all functional diagnoses (including MDD) “as problematic or mythological” (Pilgrim & Bentall, 1999). Despite attempts for standardisation through the use of standardised tools and interview schedules, significant

diagnostic heterogeneity is still evident among patients given the diagnosis of MDD (McPherson & Armstrong, 2006).

Moreover, the APA has been criticised for not acknowledging the role that social processes, mental health policies, advancements in the production of psychiatric drugs and power struggles between disciplines within the mental health field play in the development of its classification system (McPherson & Armstrong, 2006; Parker, 2000, 2005). Horwitz (2012), for example, points at a 600% increase in spending on anti-depressant medication in the 1990s in the USA and a \$7 billion annual expenditure by the year 2000. In his compelling book *“The loss of sadness: how psychiatry transformed normal sadness into depressive disorder”* (ibid), the author argues that there has been a deliberate medicalisation of “normal sadness” (conceptualised a reaction to one’s life circumstances), which served the interests of large pharmaceutical companies: “antidepressant medications such as Prozac, Paxil, Zoloft, and Effexor, are now among the largest selling prescription drugs of any sort” (Horwitz, 2012, p. 4).

Voicing similar concerns, the British Psychological Society (BPS), published a guideline document on the use of language in relation to functional psychiatric diagnosis, encouraging psychologists to avoid the use of diagnostic labels, and suggesting alternative descriptions instead (Division of Clinical Psychology Beyond Functional Psychiatric Diagnosis Committee, 2015). More specifically, the document suggests that terms like “emotional distress” and “severe mental distress” replace the use of “mental illness”, and “low mood” and “misery” replace the term “depressive disorder”. The BPS Division of Clinical Psychology argues that these changes are necessitated by a “growing body of evidence suggesting that the experiences described in functional diagnostic terms may be better understood as a response to psychological factors such as loss, trauma, poverty, inequality, unemployment, discrimination, and other social, relational and societal factors” (ibid, p.1). Another BPS document, issued in response to the publication of the DSM-5, goes as far as stating that both mental health service users and the general public are



negatively affected by the ongoing medicalisation of what can be thought of as a natural response to human experiences (British Psychological Society, 2011).

Another central criticism of the MDD diagnostic label is that it does not meet criteria for diagnostic validity and utility (Parker, 2005). Parker critiques the diagnostic criteria for creating a profile of high severity, whilst the descriptive criteria are seen as too broad and potentially leading to placing the inclusion bar too low. This is seen as compromising the clinicians' ability to make valid treatment response predictions on an individual level. Furthermore, Parker argues that "unless the classificatory system is underpinned by a valid explanatory model for the disparate depressive disorders, that system will be intrinsically flawed" (p.469).

The current debates about the validity and utility of MDD as a diagnostic category have important treatment implications, and are therefore relevant to the subject of treatment resistance. Parker (2005), for example, argues that the "non-specificity" of the MDD diagnosis leads to non-specificity in treatment options. Indeed, randomised controlled trials (RCTs) literature on the efficacy of various interventions has shown an overall improvement rate of 50-55%, which is also considered to be a possible over-estimate, as RCTs have been criticised for typically including patients who are on the less severe spectrum of the disorder and are more likely to remit spontaneously (ibid). The risk of unremitting depression in MDD populations has been reported to be as high as 29% (Keller et al., 1984) and the introduction of various treatments over the last few decades has been reported to have had little impact on the prevalence of chronic major depression (Scott, 1988). Parker (2005) warns against the danger of treating clinicians "fitting" the patient into the mode of therapy that the clinician is most familiar with.

Further understanding of treatment-resistance through advanced subtyping of depression has therefore been argued to be instrumental in the continuous improvement of treatment choice and delivery (Akiskal & McKinney, 1973).

The author of this thesis considers the criticisms outlined above to be of key importance to the ongoing conceptualisation and treatment of what is currently described as “major depressive disorder”; the reader will therefore be reminded of these on-going debates throughout the thesis. Yet, the author will continue to use the diagnostic terms “MDD” and “personality disorder” in this writing, as this has been the language adopted by the Tavistock Adult Depression Study (TADS), of which the current research formed part.

## **1.2. Treatment-resistant Depression**

### **1.2.1. Definition**

In light of the MDD ongoing debates presented above, it is not surprising that the definition of “treatment-resistant depression” (TRD) has also been marked by ongoing inconsistencies and lack of validation (Berlim & Turecki, 2007). Although a full review of the development and use of the term is beyond the scope of this chapter, the author considers it important to mark what appears to be common in current definitions of TRD across authors, where the main variations lie, and what the resulting limitations and controversies are. The latter, in particular, have important treatment implications.

It appears that most, if not all, authors agree that for a patient to be labelled as “treatment-resistant”, there has to be a preceding failed treatment attempt. Furthermore, this treatment, typically with anti-depressant medication, needs to have been administered at an adequate dose and for an adequate duration of time, and the patient must have complied with the prescription (Berlim & Turecki, 2007; Fava, 2003; Trivedi, Nieuwsma, & Williams, 2011). It is here that variations in TRD definitions begin, with authors presenting different ideas about what constitutes “adequate” treatment; how many previous failed treatment attempts are needed before TRD is diagnosed; and what is considered an “adequate treatment response”. Some authors consider a reduction of >50% of baseline depressive symptoms as significant improvement (Sackeim, 2001), whilst others suggest that TRD lies on a continuum, from “absolute” to

“partial” resistance (Berlim & Turecki, 2007) or partial response (Fava, 2003). A few authors warn against the more liberal definition of treatment response (e.g. >50% symptom reduction), and insist that a patient should be considered as treatment-resistant unless a full remission is achieved following treatment, for any residual symptoms have been shown to be a strong predictor of further relapse into depression and possible chronicity (Fava, 2003; Sackeim, 2001).

There are two further major problems with the above definitions: first, non-pharmacological treatments are largely neglected in the literature; second, treatment “success” is operationalised as a reduction of the number of observed or reported symptoms of depression. If “depression” is thought of as “misery”, “sadness” or “unhappiness”, “deserving of help and intervention” (British Psychological Society, 2011; Division of Clinical Psychology Beyond Functional Psychiatric Diagnosis Committee, 2015), the use of medication as a main (and often sole) line of treatment becomes hugely questionable. The treatment “failure”, could therefore be easily attributed to the treatment (i.e. medication) not being the right type of intervention needed, as opposed to the condition (i.e. sadness, misery) being “resistant” to the “right” treatment.

Furthermore, TRD definitions also pay little, if any, attention to the “embodied conscious experience” of sadness (Parnas, Sass, & Zahavi, 2013). Parnas et al. (ibid) critique the exclusive focus of symptoms in psychiatry, for they are devoid of “intrinsic sense or meaning” (p.275), whilst attempting to capture and describe a subjective human experience. TRD definitions and the related literature generally fail to make any references to personal experiences and meaning of depression, and how those might change as a result of a person being subjected to numerous unsuccessful pharmacological interventions.

Some further limitations of the current TRD definitions include: the evident heterogeneity of those considered to have TRD; the confusion of TRD with “pseudo-resistance”, defined as resistance due to initial misdiagnosis or the delivery of inadequate treatment; and the blurred line between TRD and chronic depression (Bschor et al., 2014; Greden, Riba, & McInnis, 2011;

Scott, 1988).

Heterogeneity and chronicity pose particular challenges to treatment, as clinicians are left with little insight into what might work for a particular patient, labelled as treatment-resistant. Indeed, TRD indicates only what had not worked in the past (e.g. a particular class of anti-depressant) but reveals virtually nothing about subgroup characteristics and differences on the basis of which to justify treatment choice (Fava, 2003). Likewise, there is no clear distinction between chronic depression (e.g. MDD that has persisted for 2 or more years (Scott, 1988)) and TRD – a problem which some have addressed by offering further sub-classifications, such as “non-chronic TRD” and “chronic TRD” (ibid). The DSM-5 (APA, 2013) had, however, merged chronic depression and dysthymia into the label of “characterological or chronic minor depression”. These differences in classification pose a major challenge to clinical interventions, as depression has been manifested to change its course and severity over periods of time (Judd et al., 1998a). It might therefore appear that whether one is given a label of “dysthymia”, “chronic”, “characterological” or “treatment-resistant” depression might be more dependent on the time one is psychiatrically assessed, rather than on the individual’s actual history of depression and the underlying subjective experiences. Such an approach might perpetuate, rather than alleviate TRD.

The current thesis uses the TRD definition adopted by the TADS (Fonagy et al., 2015), as it uses the RCT’s data. The TADS considered patients to be “treatment-resistant” if they met current DSM-IV MDD diagnostic criteria (APA, 1994), with a minimum two-year duration of the current depressive episode at the time of assessment. In addition, patients had to score a minimum of 14 on the Hamilton Depression Rating Scale (Hamilton, 1960) and have history of at least two previously unsuccessful treatments, at least one of which had been with antidepressant medication.

The TADS results will be considered in more detail in the following section of this chapter,

which reviews current treatments for TRD and their efficacy.

### **1.2.2. Treatment**

#### ***1.2.2.1. Pharmacological interventions***

Antidepressant medication generally remains the first line of treatment for MDD, both in terms of guidelines recommendations (National Collaborating Centre for Mental Health (Great Britain) & Royal College of Psychiatrists, 2010), as well as in actual clinical practice in primary care in the UK (McPherson & Armstrong, 2012). This has been despite ongoing critical reviews of medication's actual benefits (Kirsch, 2014) and claims that the medical profession has to a large degree "pathologised sadness" (Speed, Moncrieff, & Rapley, 2014).

A number of reviews of the effectiveness of antidepressants in treating TRD have been published (e.g. Berlim & Turecki, 2007; Greden et al., 2011). It is beyond the scope of this thesis to present a summary of their findings. However, it is important to note that these publications focus predominantly on three main areas: what is considered an "effective treatment", what factors (patient-, treatment-specific and other) are related to treatment outcome, and what treatment alternatives are recommended, when a patient does not respond to an antidepressant. Longer courses of pharmacological treatment, labelled as "maintenance" treatment, as well as treatment augmentation or substitution with another class anti-depressant appear to be common recommendations. However, these approaches have been criticised for lacking compelling empirical support and validation (Berlim & Turecki, 2007 in the case of prolonged treatment; Bschor et al., 2014, in the case of medication substitution). Furthermore, some authors go as far as recommending a "life-time approach" to pharmacological treatment (Greden et al., 2011), whilst significantly downplaying the short- and long-term side-effects of anti-depressants and the impact of those on patients' functioning and overall quality of life (Trivedi et al., 2011). To name a few, these are known to include weight gain, insomnia and disruptions in sexual functioning (N. H. S. Choices, 2015). Pharmacogenetic testing (looking into how the individual

metabolises medication and what side-effects are likely to develop) is recommended for all patients diagnosed with MDD, not just those deemed “treatment-resistant” (Greden et al., 2011), but it is unclear to what extent this is adopted in routine clinical practice.

Thase (2013) reported a general estimate of 50% remission rate, following treatment with antidepressants. It does appear, however, that for a large proportion of patients, pharmacological treatments remain ineffective, which necessitates the exploration of alternative treatment options, such as psychological interventions.

#### ***1.2.2.2. Psychological therapies***

Talking therapies have been recommended as a second line of treatment for TRD (Bschor et al., 2014; Rush et al., 2006), despite a number of studies having reported similar overall treatment effectiveness for medication and psychological interventions. Bschor et al. (2014), for example, concluded that all interventions appear to lead to clinically significant improvements in roughly half of the patients, and that medication and psychotherapy should be given equal weighting in the treatment of TRD. The observed dominance of medication is attributed by some authors to funding being more readily available for pharmaceutical trials (Thase, 2013; Trivedi et al., 2011). Furthermore, carrying out well-designed RCTs is considered methodologically more difficult for psychotherapies, because there is no “placebo” condition in therapy and larger-scale studies are therefore less feasible, leading to limited statistical power and less likelihood that true differences between conditions are detected (Thase, 2013).

The results of a number of studies on the effectiveness of psychological interventions for TRD have nonetheless been published in the last couple of decades. These have focused mainly on Cognitive, Behavioural and Cognitive-Behavioural Therapy, as well as Interpersonal Therapy.

*1.2.2.2.1. Cognitive-behavioural therapy (CBT), Interpersonal therapy (ITP) and Behavioural Therapy*

A systematic review of the existing literature on the effectiveness of psychological treatments for TRD was carried out by McPherson et al. (2005) and included five controlled and eight uncontrolled clinical trials. TRD was defined as failure to respond to at least one course of antidepressant treatment and MDD severity of HDRS score >14. All but one study examined the effectiveness of CBT, as compared to either medication, being on a waiting list or using self-help materials. One trial looked at the effectiveness of psychoeducation as an intervention for TRD. A significant decrease in depressive symptoms was reported in two of the controlled and five of the uncontrolled trials. However, the authors pointed at a number of definition- and methodology limitations in these studies, and concluded that there is an urgent need for more well-designed trials testing the effectiveness of various psychological therapies for TRD.

Trivedi et al. (2011) carried out a systematic review of Randomised Controlled Trials looking at the effectiveness of CBT, IPT and/or behavioural therapy for TRD. TRD was defined as failure to achieve remission or remitting only partially, following treatment with antidepressant medication for six or more weeks. The authors were interested in treatment in primary care settings and therefore applied stringent exclusion criteria. This led to only 13 articles, representing 6 trials, being included in the review. The majority (78%) of the total number of participants (592) came from only two trials. All but one trial studied Cognitive Therapy (CT) (the exception was Dialectical Behavioural Therapy) and the number of sessions was 12-16, with one trial offering 23 sessions. The average depression severity at baseline in the studies varied. It was below 14 on the Hamilton Depression Rating Scale (Hamilton, 1960) in three of the trials and between 16.2 and 17.8 for the other three. Finally, the review excluded studies where patients presented with any other psychiatric conditions “unlikely to be treated by primary care clinicians (e.g. suicidal ideation, severe substance abuse)” (p.644). The results of the review

were mixed, with some studies reporting similar benefits of psychological interventions, compared to medication (e.g. CT in the STAR\*D trial, Rush et al., 2006). One study reported lithium augmentation to be superior to CT, whilst another study concluded that CT was superior to medication.

This review has a number of limitations. First, the sample of participants across the studies could be considered as “diagnostically clean”, due to the stringent exclusion criteria. Second, although participants were followed-up for 8 weeks to 2 years, little information was provided on the extent to which treatment benefits were sustained over longer follow-up periods. Finally, no reference was made to the aetiology of depression in the population included. The importance of a thorough assessment and formulation in the treatment of major depression has been highlighted as key to effective treatment planning and delivery (Wakefield, 1998).

Bschor et al. (2014) carried out a review of the existing literature on treating chronic and treatment-resistant depression. The authors recommend a systematic, step-wise approach to diagnosis and treatment and argued that following a “scientifically grounded treatment algorithm” (p.772) yields better outcomes than providing treatments based on clinical judgement. Yet, in the proposed algorithm psychological therapies were presented right at the bottom of what would appear a treatment hierarchy. Furthermore, the authors themselves noted that psychotherapy appears to yield more sustained longer-term benefits for the patients and improves compliance with medication. The authors gave very brief summaries of the main psychotherapeutic approaches (CBT, IPT, psychodynamic psychotherapy, and CBASP), and provided no reference to trials looking at these therapies’ effectiveness in treating TRD. Furthermore, it is unclear if the authors used the term “chronic depression” interchangeably with TRD.



#### *1.2.2.2.2. Psychodynamic psychotherapy for TRD*

Far fewer studies appear to have been conducted on the effectiveness of psychodynamic psychotherapy for TRD. The author carried out a systematic search of the following databases on 16<sup>th</sup> January 2016: PsycINFO, Education Source, ERIC, PEP Archive, SocINDEX, Psychology and Behavioral Sciences Collection, eBook Collection (EBSCOhost), PsycARTICLES, PsycBOOKS, MEDLINE, Health Business Elite, and CINAHL, using the key phrases "psychodynamic therapy", OR "psychodynamic psychotherapy", OR "psychoanalytic therapy", OR "psychoanalytic psychotherapy" to denote the treatment modality, and "treatment resistant depression" OR "treatment refractory depression" to specify TRD. This search yielded 13 results, but after their titles and abstracts were screened, only two publications were deemed to be relevant (actually looking at the effectiveness of psychodynamic psychotherapy for TRD): Abbas (2006) and Fonagy et al. (2015). Each is reviewed below.

Abbass (2006) reported significant symptomatic and functional improvements in patients with TRD, who had completed a course of Intensive Short-Term Dynamic Psychotherapy. This treatment was described by the author as focusing on helping patients tolerate difficult emotions, by placing emphasis on affect or cognition, followed by supporting the patient in processing emotional experiences linked to past or current events and interpersonal relationships. The author did not comment on the similarities and differences of this intervention with other psychodynamic approaches. The study was not controlled and there were only 10 patients in the sample, with the treatment delivered by one clinician. Significant improvements in TRD were reported, as measured through both self-report and clinician-rated measures. Based on post-treatment HDRS scores, 8 of the 10 patients are reported to have remitted. No information was provided on the follow-up period. The author reported that all of the patients presented with complex difficulties at the start of treatment (personality disorders, HDRS mean score of 22.6 indicating severe depression, and major interpersonal problems). He concluded that "The

observation that depression and interpersonal problems improved simultaneously suggests that personality changes may be necessary in the TRD for depression to lift” (p.452).

It is surprising that the only other study on psychodynamic psychotherapy for TRD was not published until 2015, particularly given that a meta-analysis by Driessen et al. (2010) concluded that short-term psychodynamic psychotherapy appears to be an effective treatment for major depression, with its benefits sustained over 3-, 6- and 12-month follow-up periods. No studies on the efficacy of longer-term psychodynamic therapy (LTPT) for major depression have been carried out prior to the Tavistock Adult Depression Study (Fonagy et al., 2015), although Luyten & Blatt (2012) reported the results of an RCT comparing LTPT to brief psychodynamic therapy (BPT) and solution-focused therapy (SFT) for mood and anxiety disorders, where LTPT was initially inferior to BPT, but demonstrated superior results to both BPT and SFT in the third year of the follow-up period.

The Tavistock Adult Depression Study (TADS) (Fonagy et al., 2015) was carried out in response to these gaps in the evidence base for TRD. It was a pragmatic randomised controlled trial, where patients were allocated either to 18-month weekly psychodynamic psychotherapy or to Treatment as Usual (TAU). The methodology of the TADS will be presented in detail in the methods chapter of this thesis, as the current project forms part of the TADS and uses its data. For the purposes of the current review of treatments for TRD, it is sufficient to say that few of the participants achieved full remission at the end of treatment (9.4% in the treatment condition and 6.5% in the control group), and only some achieved partial remission (32.1% and 23.9% respectively). The differences between the two conditions at 18 months were not statistically significant. Significant between-group differences emerged, however, at 24-months of the follow-up period with 30% of the treatment group participants sustaining partial remission, compared to only 4.4% of the TAU group, indicating that long-term psychodynamic psychotherapy can lead to long-term improvements in some of the patients considered as

“treatment-resistant”. It remains unclear, however, what differentiates those who benefitted (albeit partially) from the LTPP treatment, from those who did not.

#### ***1.2.2.3 TRD psychological interventions - conclusion***

As evidenced in the psychological therapies reviews presented above, significant variations in the definition of TRD make the comparison of trials difficult (Gaynes et al. 2011). Furthermore, studies of the effectiveness of psychological (and pharmacological) interventions tend to use predominantly the HDRS (Hamilton, 1960) and the Beck Depression Inventory (Beck, Steer, & Brown, 1996) as outcome measures. These two measures focus solely on depressive symptoms and provide no information about the individuals’ quality of life or global functioning.

Furthermore, TRD appears to comprise a heterogeneous group of individuals with likely varying depressive aetiologies and comorbidities. It is therefore not surprising that all available treatments (psychological and other) appear to fare similarly overall, as it is unlikely that one size would fit all. Wijeratne & Sachdev (2008) indeed argued that “treatment resistance may be more usefully conceived within the context of well-defined cohorts such as patients with specific subtypes of depression” (p.751). Some of the factors that might assist in such further refinement of definition and classification, are presented next.

#### **1.2.3. Risk factors predicting treatment resistance**

A number of authors have attempted to establish what predicts treatment-resistance. Illness-specific factors (relating to the MDD history, the nature and severity of symptoms and the duration of depressive episodes) and person-specific factors (relating to personal characteristic, cognition and personality traits/personality disorder) appear to be most often referred to in the literature. Overall, these investigations have remained largely medical and diagnosis-driven, which is a major limitation to understanding what contributes to the recurrent and persistent experiences of depression, as outlined earlier in this chapter.

### ***1.2.3.1. Depression-specific factors***

The number of previous depressive episodes was linked by a few authors to rapid treatment relapse and chronicity (e.g. Judd et al., 1998b; Kessing, Andersen, Mortensen, & Bolwig, 1998). One study estimated that a history of three or more previous major depression episodes is associated with 70-80% likelihood of relapse (Segal, Pearson, & Thase, 2003). Kessing et al. (1998) concluded that the higher the number of episodes, the shorter the time to recurrence, and that severe unipolar depressive disorder is recurrent and progressive in nature, despite the effect of treatment.

In addition, both the length of previous major depressive episodes (Scott, 1988) and the duration of the current episode (Sackeim, 2001) have been found to be predictive of chronicity and treatment-resistance, respectively. Sackeim (2001) acknowledges, however, that subjecting the patient to an ineffective treatment for a prolonged period of time might actively contribute to the duration of the depressive episode and therefore might be precipitating “treatment-resistance”.

Partial-recovery and sub-syndromal depressive symptoms were also found to be reliable predictors of rapid relapse into MDD, even when adequate treatment had been delivered (Scott, 1988; Judd et al., 1998b; Rush, Thase, & Dubé, 2003). Rush et al. (2003) suggest that temperament and personality disorder might predispose individuals to incomplete recovery and that the link between MDD and personality should therefore be investigated further.

### ***1.2.3.2. Personality traits – neuroticism***

Howland & Thase (2005) stated that there are two main ways of classifying personality: categorically (in line with the DSM Axis II clusters) and in terms of traits and temperaments, which lie on a continuum “and span normal and pathological populations, differing only by a matter of degree of severity” (p.158-159). Neuroticism is one such trait. It is defined as “a tendency to respond to distress in emotional, anxious and somatic ways” (p.159).

Kay, Garside, Beamish, & Roy, (1969), Hirschfeld, Klerman, Andreasen, Clayton, & Keller (1986) and Segal et al. (2003) all link neuroticism to MDD chronicity and treatment resistance, whilst also highlighting hypotheses that the prolonged experience of major depression might exaggerate certain personality traits, including neuroticism. It is therefore recommended by these authors that personality assessments are ideally carried out pre-morbidly (i.e. before the patient becomes depressed, or during an episode of remission). Kay et al. (1969) and Scott (1988) emphasise the importance of premorbid personality assessment in the formulation of prognosis and the choice and course of treatment, but consider such assessments problematic, not least because depression and personality traits are thought to interact, making causal relationships difficult to ascertain. Personality appears to play an important role in treatment chronicity and resistance, however, despite these conceptual challenges.

The remaining part of this chapter will focus on the link between personality disorders in general, and borderline personality disorder, in particular, and treatment-resistant depression, as well as on the potential implications that this has for treatment.

### **1.3. Depression and Personality Disorders**

The relationship between depression and personality has been most widely studied in terms of the links between the different personality *disorders* and depressive illness. A review of the literature on depression and personality disorders is beyond the scope of this thesis. The author will therefore give a brief summary of what is known about the prevalence of personality disorders in treatment-resistant depression and the implications that the former are thought to have for the treatment of the latter. The author will then examine more closely the literature on borderline personality disorder (BPD) and TRD. BPD is chosen as the focus of this thesis, as it has been reported to be commonly diagnosed in outpatient and forensic psychiatric settings, and to be associated with a number of other psychiatric diagnoses (MDD being one of them), incurring significant personal and social costs (Bateman & Fonagy, 2016). The thesis's research

questions, which address the links between BPD, TRD and treatment outcome, will be presented at the end of the chapter.

The reader is again reminded of the limitations of psychiatric diagnosis, this time in relation to the term “personality disorder”; the BPS recommends the use of terms like “complex trauma” or “relationship or attachment difficulties”, instead (Division of Clinical Psychology Beyond Functional Psychiatric Diagnosis Committee, 2015). The link between personality disorder and trauma will be explored in more detail later in this chapter. The author continues to use the term “personality disorder”, however, as this was the language adopted by the TADS.

### **1.3.1 Treatment-resistant depression and personality disorders – prevalence, comorbidity and treatment implications**

A number of research studies have reported high comorbidity rates of depression and personality disorders (PD) (Farmer & Nelson-Gray, 1990; Keown, Holloway, & Kuipers, 2002; Rao, 2003).

A review by Hirschfeld (1999) found that comorbidity of PD across the different types of depression ranges from 41% to 81%. Shea & Yen (2005) provided an overview of existing theories, but focused more broadly on the role of personality traits, in addition to personality disorders, in relation to depression. The authors concluded that there is not a single theoretical model that could explain the complex links between depression and personality in general.

Petersen et al. (2002) found no differences in the prevalence of PD in a sample of TRD patients compared to non-TRD patients. In fact, more patients in the latter group initially appeared to meet criteria for Obsessive Compulsive personality disorder, but this difference became statistically insignificant after adjustments were made in statistical analysis. The authors pointed at the large body of literature that indicates that poor response to treatment of depression is associated with “personality dysfunctions” (expressed in shorter periods of remission, poorer social support, and earlier age of depression onset). Yet, they also argued that the exact role of

personality disturbance in treatment-resistant depression has been difficult to study, because of TRD being historically poorly defined by clinicians and researchers.

Howland & Thase (2005) argued that the link between TRD and personality is not simply one of co-morbidity, but rather a reciprocal and interactive one, where stress is likely to be also linked to and have an impact on both. The authors presented several different ways of conceptualising the relationship between depression and personality: the two have common causes; personality is an early manifestation of depression; personality predisposes one to depression; personality influences the expression/course of depression; personality features are amplified in the course of depression and can therefore be seen as the effect of depression. Furthermore, the authors reported mixed findings in terms of the impact of personality on antidepressant treatment outcome, but a few studies have been carried out to address this issue. Moreover, there have been very few studies looking at personality implications for psychotherapy treatment outcome in chronic depression (none reported for TRD). The authors nonetheless concluded that personality difficulties and disorder have negative impact on treatment, although treatment (medication and psychotherapy) has also been found to improve personality functioning in depressed individuals in some studies. The authors pointed at the importance of comprehensive initial assessment in chronic and refractory depression, which makes use of both the patient and collateral sources in order to build up a formulation and gather information about possible personality pathology. When Axis II comorbidity is clearly established a wide range of treatment options (medication and/or therapy) should be considered (ibid).

### **1.3.2. Psychoanalytic conceptualisation of depression, its links to personality and the related treatment implications**

The DSM-5 (APA, 2013) is a classification and diagnostic tool which, at least in theory, has been designed as a-theoretical. This means that each disorder listed in the Manual is described in terms of its presenting symptoms but with no reference to its aetiology.

The aetiology of depression is conceptualised differently by psychiatry and psychoanalysis (Bell, 2010). Psychiatry, argues Bell (2010), makes a clear distinction between personality and mental illness and between normal and abnormal (as expressed through the DSM's definition of Axis I and Axis II disorders and the categorical approach to diagnosis). More specifically, personality difficulties, or disorders, are thought to be associated with enduring, long-term dysfunctions across a number of life domains, and therefore are considered to require long-term treatment. Mental illnesses, like depression, however, are seen as episodic, and therefore susceptible to localised, shorter-term treatments. Different disorders are then seen as co-morbid, and, as presented earlier in this chapter, the implications of such comorbidities for treatment outcome are studied empirically in RCTs.

Psychoanalytic theories, conversely, view mental illness as lodged into personality development, under the stress of external and internal conditions. Symptoms are viewed as “the outer expression of deeper [personality] structures” (Bell, 2010, p. 148). The term co-morbidity does not exist, consequently, as disorders like depression are understood and treated through an understanding of how the person functions as whole.

Furthermore, psychoanalysis takes a dimensional, rather than categorical view with regards to what is “normal” and what is “abnormal”:

“Disorder manifests aspects of mental life which at a different developmental phase might have been normal. Although we never completely lose earlier ways of functioning, when these more archaic forms come to dominate mental life they become the basis of psychopathology.” (Bell, 2010, p. 146)

The concept of depression was first explored by Freud, as the concept of *melancholia* in his seminal paper *Mourning and Melancholia* (Freud, 1917). There, he argued that the loss of important objects (e.g. a loved person, one's country, liberty or an idea) leads the person to experience a range of complex feelings, including sadness and internally directed anger



(expressed through self-denigration, for example). According to Freud, loss is an inevitable part of life. When the person navigates successfully through the process of loss, they are thought to have “mourned” the lost object, or in other words to have “fully relinquished” what has been lost, which in turn leads to a resolution of the painful feelings and to developmental progress. When this process is arrested, however, the individual is left in a state of melancholia, which Freud described as characterised by:

“(…) a profoundly painful dejection, cessation of interest in the outside world, loss of the capacity to love, inhibition of all activity, and a lowering of the self-regarding feelings to a degree that finds utterance in self-reproaches and self-revilings, and culminates in a delusional expectation of punishment.” (Freud, 1917, p. 244)

Freud further made links between melancholia and personality (he used the word “character” to denote this), by hypothesizing that melancholia was marked by an identification with the lost object and a narcissistic object-choice, as well by narcissistic regression. It is important to note that the term “narcissism” was not used in the same way as what psychiatry today classifies as narcissistic personality disorder. The scope of this thesis does not allow for a detailed analysis of Freud’s theory on melancholia, but it suffices to say that Freud, for the first time, tried to link the experience of real or perceived loss, to depressive states and the structure of the personality as understood through object-identification, object-choice and the nature of regression into earlier modes of psychic functioning.

Freud, in that sense, established the psychoanalytic tradition of viewing depression, and mental illnesses in general, not as what the person “has” (in terms of symptoms and complaints), but as who he or she is, “his or her way of being in the world”, with the current problems being “woven into the fabric of their lives” (Shedler, 2010).

Psychoanalytic authors since Freud have criticised the symptom-based approach to diagnosing and treating depression and the lack of attention paid to the overall personality an individual

experiencing depression. Parker (2005), for example, argued that the term “major depression” aims at having a “cachet value”, whilst circumscribing a set of different conditions, homogenising them and thus misconstruing the individual patient’s experiences and treatment needs. He pointed to the importance of linking the different depression subtypes to “predisposing personality style and clinical phenotype” (p.472).

In line with Parker’s (2005) critique, recent psychoanalytic writings on depression stress the importance of focusing on the individual’s internal experience of depression, such as the “feelings of loss and of being abandoned and unloved, on the one hand, and feelings of worthlessness, failure, and guilt on the other” (Blatt, 2015, p. 131). Furthermore, it is suggested that depression can be further understood in relation to two main dimensions: the “quality of interpersonal relatedness” (e.g. feelings of being unloved, abandoned, and uncared for by others) and the “experiences of self-definition” (e.g. feeling worthless and a failure) (ibid). These two dimensions are further labelled as “anaclitic” (or “dependent”) and “introjective” (or “self-critical”), respectively. Blatt (2015) argues that the expression of either is determined by a heterogeneous group of early life experiences, leading to different symptomatic manifestations and therefore to different responsiveness to treatments. For example, those individuals who present as predominantly anaclitic, are thought to be more dependent on others for support, and to be very fearful of losing important others, which in turn leads them to be more submissive and placating. Suicidality and substance misuse are thought to be an attempt to elicit others’ support, within an anxious-avoidant attachment context, as opposed to a serious intent to harm or kill oneself (i.e. the individual’s strategies to elicit interpersonal support are limited to eliciting care through, for example, self-harming, whilst they do not intend to actually end their life). Individuals who are more introjected, on the other hand, are thought to be highly-critical of self and others and therefore rejecting of support and generally more hostile in their interpersonal

interactions. The differences between these two dimensions are argued to be of huge relevance to the treatment of depression. Blatt (ibid) concluded that:

“(...) significant differences in sustained therapeutic change might occur, in a variety of treatment interventions, as a function of interaction between differences in patients’ pre-treatment personality organisation and aspects of the treatment process, especially the quality of the therapeutic relationship. Including a differentiation among patients in research designs and data analytic strategies could provide a fuller understanding of the processes that lead to sustained therapeutic change.” (p.138).

The TADS LTPP Manual (Taylor, 2003) maintained the general psychoanalytic view of depression by postulating that all types of depression “involve needs and dependency, separation, loss, and disappointment, and internally directed aggression, arising in relation to these important primary object-relation wishes” (p. 4). Taylor further argued that in ‘treatment resistant’ depression the more destructive parts of the personality have taken over the healthy, functioning parts of the self, through a complex constellation of factors, which operate “powerfully to keep the individual concerned in a state of chronicity in a way that is not the case in ‘ordinary’, self-limiting depressions”.

Long-term psychoanalytic psychotherapy for depression is thus not primarily aimed at alleviating symptoms, although the symptoms might be the focus of conversation between the therapist and patient in the initial stages of the treatment. Rather, the therapist attempts to support the patient in gradually changing key aspects of his/her personal functioning, aspects “often connected with developmentally early experiences of loss, to reduce an underlying depressive diathesis” (ibid). Of note, different aspects of the personality are considered to be in constant interaction both with the external environment, as well as with one another (Taylor, 2003). Furthermore, object relations, personality structure and the experience of symptoms are all regarded as “dimensional rather than categorical, and dynamic rather than static in nature”.

(Taylor, 2003, p. 28). This point is of particular importance to the subject of the current project and will be considered in more detail in the discussion chapter of the thesis.

The LTPP approach to depression and the way it is intrinsically linked to personality appears further in line with Parker's (2000) argument for linking, what is in DSM terms, Axis I and Axis II conditions, in order to obtain a more refined classification of different types of depression. The author discusses the role that clinical observation has to play in identifying clinical patterns, or clinical “syndromes”. This is demonstrated through examples from existing studies, where participants’ experiences of depression and the way in which the condition is manifested are qualitatively different, based on the person’s personality characteristics, as well as on the presence of other Axis I conditions, such as anxiety. The author further argues that temperament is likely to play an important role in the expression of depressive symptoms. He does not comment on the link between personality and past life experiences, such as one’s early attachment relationships, however.

In conclusion, the experience of loss, and feelings of abandonment, loneliness, worthlessness and self-criticism appear to be central features of depression in the psychoanalytic literature. These features are in fact not dissimilar to the more symptom-focused DSM conceptualisation. What distinguishes psychoanalytic theories from psychiatric ones, however, is the importance that personality plays in understanding any mental illness, depression included, as symptoms are seen as the expression of deeper psychic structures. Exploring and understanding the personality structure in theory, as well as in the consulting room, is therefore seen as an integral part of facilitating treatment management and good treatment outcome.

#### **1.4. Borderline Personality Disorder and Treatment-resistant Depression**

This section will present a summary of the psychiatric and psychoanalytic conceptualisation of borderline personality disorder, followed by a discussion of how this relates to depression and treatment outcome.

### 1.4.1. Definition

#### 1.4.1.1. DSM-5 definition

The DSM-5 defines personality disorder 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.”

(APA, 2013, p. 645)

Furthermore, this enduring pattern needs to manifest itself in at least two of the following areas:

*cognition, affectivity, interpersonal functioning, impulse control.* In terms of diagnosing personality disorder, the DSM-5 recommends that the diagnosing clinician carries out a number of interviews with the patient, spread over time and focusing on the individual’s functioning across different situations, as well as gathering information from “other informants” (ibid). This is contrasted to the assessment and diagnosis of “more transient mental states”, such as anxiety or depressive disorders (APA, 2013, p.647).

The main diagnostic features of borderline personality disorder (BPD), as described in the DSM-5, include instability of self-image and interpersonal relationships, as well as unstable affect and marked impulsivity. Nine BPD criteria are listed in the manual, at least five of which need to be met in order for a diagnosis to be justified. In summary, the criteria are: efforts to avoid real or imagined abandonment; unstable and intense interpersonal relationships, marked by an oscillation between denigrating or idealising others; unstable self-image/sense of self; impulsivity that leads to risk to oneself; recurrent suicidal or self-harm behaviour or threats; affective instability; chronic feelings of emptiness; inappropriate, intense anger, and difficulties managing it; transient dissociation and paranoid ideation, triggered by stress.

The DSM-5 further highlights that individuals diagnosed with BPD are highly sensitive to environmental circumstances and experience intense fear of abandonment and strong reaction (e.g. panic, anger) to endings, even when those had been planned in advance. Furthermore, new relationships (with a care-giver or an intimate partner) tend to follow a pattern of initial idealisation and high hopes, followed by disillusionment and denigration in the face of perceived abandonment and expected punishment. The individual's self-image is described as likewise unstable, with shifting values, interests and goals. Strong feelings of not-existing are linked to lack of supportive relationships and it is argued that individuals diagnosed with BPD struggle particularly in unstructured activities (e.g. work or school situations).

Suicidality is considered a particular feature of BPD and the Manual quotes an 8-10% completed suicide rate in individuals diagnosed with BPD. Self-harm and attempted suicide are argued to be common reasons for borderline patients to come in contact with health care services. Co-occurring depressive disorder is considered to increase the risk of premature death due to suicide.

Finally, the Manual lists "features" that are associated with BPD diagnosis, but not included in the main diagnostic criteria. These are: sabotaging oneself before the attainment of an important goal (e.g. completing a degree or staying in a supportive relationship); physical handicaps resulting from risk-taking and suicidal behaviours; recurrent occupational, educational and interpersonal disturbances and losses; early life history of neglect, abuse and parental loss; high comorbidity with other disorders such as mood, substance misuse and eating disorders, as well as post-traumatic stress disorder.

Of note, the Manual states that whilst considered to be stable and enduring over time, some personality disorders, such as anti-social and borderline personality disorders, tend to remit or become less expressed as the individual advances in age and might therefore be more difficult to identify in later life. Borderline personality disorder in particular is considered as most acutely

expressed during early adulthood, with individuals arguably becoming more settled in their vocational and interpersonal functioning during the third and fourth decade of life.

The DSM-5 acknowledges the high rates of personality disorders' comorbidity, pointing at the poor discriminant validity of its classification system, and in response discusses an alternative model for personality disorders, which places the degree of impairment in functioning and the presence of specific personality traits at the core of making a diagnosis. The alternative criteria for BPD, in particular, include an impairment in two of the following four areas: identity, self-direction, empathy, and intimacy; and the presence of at least four of the following seven personality traits: emotional lability, anxiousness, separation insecurity, depressivity, impulsivity, risk taking, and hostility. *Depressivity*, which bears particular relevance to this thesis' subject, is described as: "Frequent feelings of being down, miserable, and/or hopeless; difficulty recovering from such moods; pessimism about the future; pervasive shame; feelings of inferior self-worth; thoughts of suicide and suicidal behaviour" (APA, 2013, p. 767).

As with other mental disorders described in the DSM-5, no reference is made to aetiology or treatment implications. Trauma is only mentioned briefly in terms of higher rates of victimisation and parental loss in early life for those diagnosed with BPD, as specified above. This latter point will be returned to later in this chapter, as well as in the discussion chapter of this thesis.

#### ***1.4.1.2. Psychoanalytic definitions***

Psychoanalytic theorists define personality disorders as marked by "chronic, long-standing patterns of responding to distress, which are often limited in variability and rigidly applied regardless of appropriateness of context" (Meehan & Levy, 2015, p. 313).

Clarkin, Fonagy, Levy, & Bateman (2015) define borderline personality disorder as "(...) a chronic, serious disorder involving suicidal and self-destructive behaviour, affective lability and

dysregulation, intense interpersonal conflict, and incoherent internal representations of self and others” (p.353).

The term “borderline” was first used in 1938 by the psychoanalyst Adolph Stern when he described patients, whom he considered to be neither neurotic, nor psychotic, but fell somewhere “in-between” these two categories and thus posed a significant challenge to treatment (Stern, 1938). Stern did not consider psychoanalysis a recommended treatment for this group of patients in general, unless they presented with what he called “neurotic” symptoms, such as depression and/or anxiety (ibid). He argues that a psychoanalytic treatment was not a suitable intervention for the more psychotic parts of these patients’ personalities.

Since Stern, the “borderline patient” and issues related to the classification, diagnosis and treatment of borderline personality, have been the subject of many theoretical writings in the psychoanalytic world. These include, to name but a few, Helene Deutsch (Deutsch, 1942), Robert Knight (Knight, 1953), Donald Winnicott (Winnicott, 1969), Andre Green (Andre Green, 1977), Otto Kernberg (Kernberg, 1978), and Judy Gammelgaard (Gammelgaard, 2010). The sheer volume of these works would not allow for a synthesis of their ideas here, but the interested reader is guided to collections which attempt to accomplish this task (e.g. Hartocollis, 1977; Stone, 1986).

For the purpose of the current thesis, the author has chosen some key points from the past and current psychoanalytic ideas about the “borderline patient”, which are presented next.

“Ego weakness” and the use of primitive defences such as denial, splitting and projection/projective identification, have been described as central borderline personality characteristics by Green (1977) and Kernberg (1978). Kernberg (1975, 1978) described borderline patients as lacking integration in representations of self and others, leading to the reliance on more primitive psychic defences, such as splitting between “good” and “bad”, as well as identity diffusion, and difficulties with reality-testing. Positive and negative aspects of the self



and others cannot thus be integrated as part of a coherent whole. This in turn compromises the ability of the developing mind in early childhood to differentiate between self and other, inside and outside, somatic and psychic, fantasy and reality (Green, 1977).

Attachment theorists, starting with John Bowlby (Bowlby, 1975) and more recently Levy and Blatt (1999), as referred to in Meehan & Levy (2015), made links between anxious-avoidant and anxious-preoccupied attachment patterns of relating, and personality difficulties and disorder. Early attachment relationships (i.e. to the primary caregiver), and the process of mirroring in early life, have been hypothesised as key to the development of a stable sense of self, regulation of affect and the capacity for relatedness (e.g. Lyons-Ruth & Jacobvitz, 2008; Target, 2015).

Clarkin et al. (2015) further linked the lack of a stable and integrated sense of self to severe disruptions in the interpersonal relationships of borderline patients, which research has found to be marked by “disagreements, confusion, hostility, emptiness, and ambivalence” (p.356). The authors also listed impulsivity and negative affect, which includes experiences of depression, anxiety, anger and irritability, as the other two main features of borderline personality disorder. The emotional experiences, in particular, are marked by instability and fluctuations that are linked to environmental stimuli.

Finally, the ability to perceive and understand one’s own and others’ mental states and their link to behaviour, was coined as “the capacity to mentalize” (Fonagy, Gergely, Jurist, & Target, 2002). Deficits in mentalizing have been linked to early disruptions in the relationship between the child and the care-giver, and have been identified as particularly prevalent in people diagnosed with borderline personality disorder (ibid).

#### **1.4.2. The role of trauma and early adversity**

In her book “Trauma and Recovery” Judith Herman (1997) draws attention to the complex psychological processes that take place in the context of repeated trauma, and the impact trauma

has on the development of the personality. Throughout the book she comments on humans' innate need to relate to others. This can both protect an individual psychologically during traumatic experiences (e.g. the development of camaraderie in war context; of a "buddy" relationship in concentration camps) or can perpetuate the individual staying in an abusive relationship where the person who perpetrates the abuse is also the only person available to provide support, closeness and guidance. Furthermore, she points at the close link between trauma and depression:

"These staggering psychological losses can result in a tenacious state of depression.

Protracted depression is the most common finding in virtually all clinical studies of chronically traumatized people. Every aspect of the experience of prolonged trauma works to aggravate depressive symptoms" (Herman, 1997, p. 94).

Herman (ibid) further links experiences of abuse, particularly during childhood, to disrupted early attachments, which – as noted above – have been known to be characteristic of many patients diagnosed with BPD. She describes how for abused children, caregivers, who in any non-abusive parent-child relationship, are the source of protection, care and comfort, become a source of grave danger, too. In order to protect the attachment relationship with the parent or care-giver, the child develops a belief that he/she is causing the abuse, and a related sense of inner badness. This, Herman argues, allows the child to preserve some sense of hope, power and control – if only the child can become better, the abuse will stop. It is not difficult to the understand how a distorted representation of self- and others results, and how very little of what Bion (2003) describes as the "container" function of the parent – to process raw affect and reflect it back to the child in a more manageable form – can take place, in order to facilitate the young mind's development of the capacity for thought.

The experience of trauma, single or multiple, does not in itself lead to a borderline personality organisation. A number of authors have written, however, about the links between the two.

Green (1977), for example, linked a precocious and therefore fragile ego development to the experiences of early trauma, which he stated is often observed with borderline patients, and carry along the threat of object loss. Feelings of inner badness, fragmented representations of self- and other, and deficits in thinking and mentalizing, are all characteristic of patients diagnosed with BPD and, as just described, can be traced to experiences of early and often repeated abuse.

More recent studies point at the link between abuse and borderline pathology, too. Clarkin et al. (2015), for example, are in agreement about attachment disruptions being “robust predictors of later BPD pathology”, and link such disruptions to prospective studies of abused and neglected children. They argue, however, that the experience of early neglect, more than sexual and physical abuse, is predictive of borderline personality pathology in adult life.

Fonagy & Luyten (2016), in their comprehensive conceptualisation of the development of BPD, further point at the multifaceted links between early life experiences in general, and trauma, abuse and neglect in particular, and the development of BPD. More specifically, the authors consider the negative impact that early life adversity, especially as expressed through emotional neglect, has on the development of the “full mentalizing capacities” of the individual (ibid). The theory proposed, suggests that a child develops the ability to think about and understand its own mind, and the mind of others, in the context of a relationship with a care-giver who is attuned and responsive to its needs and emotional experiences. A care-giver who is abusive and/or neglectful creates an environment where the child learns, as an adaptive maneuver, to suppress, or dissociate from its emotional experiences, and/or elicit contingent care-giver responses through extreme behaviours (e.g. aggression, self-harm). Difficulties with regulating one’s emotions have been found to also ensue from abusive and neglectful early life relationships. These attachment patterns are replicated in later-life relationships and situations, but in a less adaptive, or even harmful ways. For example, those individuals diagnosed with borderline-personality disorder, are thought to experience more extreme negative affects, particularly under stress, which leads to an

activation of their attachment relationships (i.e. they experience a stronger need to seek closeness to and support by others); at the same time the very experience of heightened affect compromises their ability to accurately perceive their own and others' subjective worlds, which leaves them vulnerable to further interpersonal difficulties and stress. Fonagy & Luyten (2016) further link developmental (particularly attachment) theories to recent neuroscience research and the development of BPD. A comprehensive summary of their model is beyond the scope of this chapter, but the reader is encouraged to refer to the original text.

The link between early adversity, borderline personality and depression is likely to be even more complex, but this does not mean that these different factors should not be thought about and investigated both in clinical work, as well as in research. This point will therefore be returned to again in the following chapters of this thesis.

#### **1.4.3. A prototype approach to describing and diagnosing borderline personality disorder**

The two approaches to assessing and diagnosing borderline personality disorder, described above, each have their strengths and limitations. More specifically, the psychiatric approach has been argued to enable communication by providing common language for mental health professionals and patients, and to be well-fitted for the purposes of research and defining discrete population groups. Its main disadvantages have been argued to include poor reliability and validity, and limited usefulness in guiding management or treatment (McLean, 2016). Moncrieff (2007) further argues that psychiatric diagnosis, compared to a more detailed formulation of one's individual difficulties and their context, predict treatment response poorly.

The psychoanalytic approach, on the other hand, has been argued to be a better guide to intervention, due to its narrative nature, which allows for encompassing greater clinical complexity. The psychoanalytic assessment, however, is also critiqued for providing poor shared language between professionals and lay people; it is also considered lacking

indicators of severity or dimension of the “disorder” in question (McLean, 2016).

A prototype-approach to personality disorders has been suggested as superior to the current DSM classification by a number of authors (e.g. PDM Task Force, 2006; Shedler, 2015; Westen & Shedler, 1999a; Westen, Shedler, Bradley, & DeFife, 2012). A personality “prototype” is considered to be a distinct pattern of “thinking, feeling, behaving, and relating to others” (Meehan & Levy, 2015).

Jonathan Shedler and Drew Westen introduced a prototype-based approach to assessing personality disorder, which they argued combines the strengths of a quantitative approach to assessment and classification, whilst harnessing clinical judgement and experience (Shedler, 2015; Shedler & Westen, 2010; Westen & Shedler, 1999a, 1999c). The Shedler-Western Assessment Procedure (SWAP) will be presented and discussed in more detail in the method chapter of this thesis, as the tool was used as one of the main measures in the current research project.

Shedler & Westen (2007) describe a *borderline-dysregulated personality disorder* prototype, which they argue to be parallel to the DSM borderline personality disorder construct. The borderline-dysregulated PD is assessed through the SWAP tool and consists of a list of 24 descriptive statements. These are organised under seven sub-groupings as follows: *affect dysregulation*, *splitting*, *projective identification*, *identity diffusion*, *insecure attachment*, *self-harm*, and *chaotic life-style* (Shedler, 2015). These sub-headings of the borderline-dysregulated descriptive items appear to capture and combine some of the key aspects of borderline personality disorder described by psychiatry, psychoanalysis and attachment theory, as summarised earlier in this chapter. Shedler & Westen (2007) argued that their approach to assessing and categorising personality disorders allowed for the description of “complex intrapsychic processes” (p.154), like splitting and projective identification, in addition to listing more easily observable patterns of behaviour and relating to self and others (e.g. self-harm,

unstable relationships).

Appendix A presents the full list of the SWAP-II 24 items considered to encapsulate the borderline-dysregulated personality disorder profile in its pure form. Appendix B presents the narrative description of the borderline-dysregulated personality (Westen et al., 2012).

#### ***1.4.3.1. Personality “disorder” vs. personality “features”***

The DSM-5 (APA, 2013) differentiates between personality *disorders*, in general, and personality *traits* or *features*. Features are described as separate parts of the personality disorders criteria, such as “dependency” or “insensitivity”. The manual states that personality disorder should be diagnosed only when its features originate in adolescence or early adulthood, are persistent over time and lead to significant impairment in the individual’s functioning.

Meehan & Levy (2015) point at psychodynamic theories taking a continuum approach to personality conceptualisation. Individuals on the healthy end of the personality spectrum are characterised by using a wide range of defence and coping strategies in response to stressors, which in turn enables them to form and maintain stable, mutual and intimate relationships with others. Higher personality pathology, in turn, is marked by difficulties in functioning in one or more areas of life. The pervasiveness of such difficulties across different areas (e.g. occupational, interpersonal) is what distinguishes those with personality disorder *features* from those with more severe pathology – or personality *disorder*.

Westen, Shedler, & Bradley (2006) took a similar approach to distinguishing between personality *disorder* and personality disorder *features*, as measured by the SWAP. More specifically, an assessed patient’s personality is compared in the SWAP to each of the personality disorders’ prototypes in their “pure” form. If the degree of match between the patient’s presentation and the specific PD prototype is very high, the patient is considered to “exemplify” the disorder or, in high but not perfect match, to “have” the disorder. If there is a

“significant” match, the patient is considered to present with “significant features” of the disorder. The meaning of “features” vs. “disorder” in the SWAP and how this is measured will be discussed in further detail in the method chapter of this thesis.

Researchers and clinicians have now long argued that personality traits should be measured on a continuum, as opposed to categorically (Johnson & Bornstein, 1991; Perry, 1993; Shedler & Westen, 2007). It is therefore likely that there would be a group of people who fall on the BPD *spectrum* without meeting the diagnostic criteria for BPD (Blagov, Bradley, & Westen, 2007). Such individuals might be more likely to seek treatment for other co-existing problems, such as depression. It is not clear at present how BPD features might affect the treatment and outcome of depression for that group, especially for people who have never been formally diagnosed with a personality disorder (or would not meet the criteria for such diagnosis). It is even more unclear what might be of therapeutic benefit for those individuals, who also fall under the umbrella of “treatment resistant depression”.

#### **1.4.4. Conceptualisation of the Links Between Depression, BPD and the Related Treatment Implications**

The DSM-5 notes that “Borderline personality disorder often co-occurs with depressive or bipolar disorders, and when criteria for both are met, both may be diagnosed” (p.666). Bateman and Fonagy (2001) argue, however, that “depression in borderline personality disorder is an indication of affect dysregulation rather than part of an affective disorder” (p.41). This view is also supported by Trull, Stepp, and Solhan (2006), who conceptualise BPD as “primarily characterized by disturbances in emotional regulation, impulse control and identity” (p.299). The affect disturbance is thought to be expressed by emotional shifts (in response to environmental stimuli) “between different types of negative affect (e.g. anger, depression, anxiety)” (ibid).

These different theoretical standpoints pose the question of whether borderline personality disorder and depression should be viewed as separate “comorbid conditions”, or whether the two are underpinned by common intra- and interpersonal processes and experiences. This question is a very important one when treatment planning and delivery is considered. As already stated earlier in this chapter, the answer depends, at least to an extent, on the theoretical framework within which it is answered (e.g. psychiatric vs. psychoanalytic).

Furthermore, the National Collaborating Centre for Mental Health guidelines for treating BPD and those for treating depression differ in the treatments they recommend. For example, a combination of anti-depressant medication and Cognitive Behavioural Therapy (CBT)/Interpersonal Therapy (IPT) is recommended for those with moderate to severe depression (National Collaborating Centre for Mental Health (Great Britain) & Royal College of Psychiatrists, 2010), whilst the recommendation for BPD is for a minimum of three months of (ideally) twice-weekly therapy and no use of medication for unstable mood, where the latter is considered as part of the BPD presentation (National Collaborating Centre for Mental Health (UK), 2009). There is also no clear guidance currently for what course of treatment to follow when both disorders are present. Furthermore, current personality diagnostic tools (e.g. the DSM Structured Clinical Interview, SCID) have been criticized for their poor validity and reliability, as well as for placing individuals into categories, rather than on a spectrum with regards to their personality traits (Clark, Livesley, & Morey, 1997; Westen & Shedler, 1999b, 1999c).

When depression and borderline personality features (particularly in the absence of a personality disorder diagnosis) are not taken into consideration by the clinician from the intervention onset and as part of the initial formulation, it could be argued that the clinician remains blind to potential therapeutic obstacles. It is insufficient to attend to the depressive symptoms alone. Furthermore, there are implications for the therapeutic relationship and treatment duration. For example, Gabbard & Simonsen (2007) explored the link between childhood trauma, BPD and



the implications for treatment of depression and suggested that “neurobiological changes may account for some of the difficulty in treating patients who are comorbid for BPD and depression” (p.168). The authors further postulated that BPD patients might ascribe (wrongly) negative intentions/misreading facial expressions to others, including the therapist and therefore might experience the clinician as a “bad object” in the transference, which in turn creates significant obstacles to building a therapeutic alliance. This latter point is supported by recent research, presented by Fonagy & Luyten (2016), where patients with a BPD diagnosis have been found to ascribe negative emotions and intent to others’ facial expressions that are in reality “neutral”. It is easy to understand how this might pose challenges to fostering a positive therapeutic relationship, in turn.

Gabbard & Simonsen (2007) further argued that patients with BPD and depression need to be in treatment for at least 12 months in order for therapist and patient to allow time for the therapeutic alliance to develop. Following a case example, the authors stated that “treating the depression alone without a psychotherapeutic treatment specifically tailored to BPD was simply not sufficient” (p.171). Furthermore, the process of learning for individuals with BPD diagnosis and history of trauma was argued to take longer, despite the person accumulating new evidence and insight. This was linked to neurobiological changes associated with repeated exposure to trauma (also noted in Fonagy & Luyten (2016)). The treatment approach should therefore be tailored to take into consideration the impact of trauma. The longer time needed in treatment was also related by the authors to research showing that procedural or implicit memories need more time to alter (e.g. via multiple repetition of new experiences). Gabbard and colleagues (ibid) finally stressed the importance of nonetheless maintaining hope with this patient group, as they believe that change is possible.

The idea that treatments for patients with BPD diagnosis should be altered is not new. A number of authors over the past few decades have suggested different modifications and new treatment

approaches in order to respond to these patients' presentation and the challenges it creates for the therapeutic relationship (Clarkin, Fonagy, & Gabbard, 2010; Clarkin et al., 2015; Knight, 1953; Milner, 2010; Winnicott, 1969). Winnicott (1969), for example, argued that "borderline patients" can engage very skilfully in analysis by inviting the analyst to collude with their "false" neurotic self and thus to never engage with the more psychotic parts of the personality. He linked this to the "borderline patient" not being able to experience the transference as a transitional space - the analyst as a representation of the mother, but rather analyst *is* the mother in the patient's mind.

More recently, manualised treatment packages have been developed for patients with borderline personality disorder, and these new, "tailored" therapies have been tested through Randomised Controlled Trials and subsequently included in national guidelines in the UK (National Collaborating Centre for Mental Health (UK), 2009). Three main examples of such treatment approaches are Mentalization-Based Therapy (MBT) (Bateman & Fonagy, 2006, 2013; Daubney & Bateman, 2015), Dialectical Behavioural Therapy (DBT) (Linehan, 2014), and Transference-focused Psychotherapy (TFP) (Arntz et al., 2015; Kernberg, 2016; Yeomans, Clarkin & Kernberg, 2015). Despite the theoretical differences that underpin them, all three types of treatments are characterised by a relatively focused approach (the therapist being more active and guiding), compared to other "talking therapies" such as psychodynamic psychotherapy, as well as by the higher intensity of treatment engagement – for example, typically a combination of individual and group work, as well as other structured activities within a therapeutic community (DBT) or day centre setting (MBT). Patients who have not been formally diagnosed with borderline personality disorder do not typically "qualify" for the receipt of MBT, DBT or TFP.

The question thus remains of what the treatment implications are for those individuals who might present to services with depression, when there is also a degree of personality difficulties, too, which could be described as, for example, borderline personality features. In such cases

there seems to be a risk of clinicians and researchers labelling patients as “treatment-resistant” when it is the mental health professionals’ failure to develop a more sophisticated formulation of the patient’s presenting difficulties, and to offer treatment in accordance with this initial formulation.

## **1.5. Rationale and Aims of the Current Study**

### **1.5.1. Rationale**

Treatment-resistant depression is experienced by a heterogeneous group of individuals and no single aetiological conceptualisation has been proposed to date. The high comorbidity of depression and personality disorders in general, and depression and borderline personality disorder in particular, suggests that for at least some individuals experiencing persistent depression, intra- and interpersonal difficulties may be linked to the experiences of low mood. There is little research, however, that explores the link between borderline personality features and treatment-resistant depression. It is therefore not known what the prevalence of BPD is amongst individuals who experience difficult to treat depression, neither is there any empirical data on the impact that such comorbidity might have on treatment outcome.

Having a clearer idea about the potential impact that borderline personality disorder or borderline personality features might have on treatment outcome is likely to have important implications for a number of reasons.

First, healthcare professionals might consider more carefully what type of intervention they offer. For example, treatments recommended for major depression in general, such as CBT, might be counter-indicated for people with a borderline personality profile; or the same intervention might be offered, but with a different duration and in combination with another intervention (e.g. medication and psychotherapy).

Second, offering better tailored treatments to people who experience depression in the context of a BPD diagnosis might reduce the likelihood of these individuals being labelled “treatment resistant”. This in itself shifts the “responsibility” for improvement from the person to the type of support offered, as it demonstrates that not all interventions work for all patients experiencing depression, and interventions need to be more carefully matched to the overall presentation of the individual and their idiosyncrasies.

Finally, a focus on borderline *features*, as opposed to borderline *disorder* and the relation of the former to treatment outcome in difficult-to-treat depression is likely to aid the tailoring of interventions, as it shifts clinical decision-making from a categorical diagnostic approach which focuses on symptoms and comorbidities, to a conceptualisation that seeks to understand depressive experiences in the context of personality organisation and functioning.

### **1.5.2. Aim**

The main aim of this research project is to test initial hypotheses about the link between borderline personality features and treatment outcomes of difficult-to-treat depression. In order to achieve this aim, the research will focus on the following research questions:

1. Do high scores on a measure for borderline personality disorder at baseline predict treatment outcomes amongst patients with treatment-resistant depression who have been randomly allocated to either Treatment as Usual (TAU) or to Long-term (18-month) Psychodynamic Psychotherapy (LTPP)?
2. Are changes in borderline personality organisation in the course of therapy related to changes in depression and treatment outcome?

## **2. Method**

### **2.1. Epistemology**

Critical-realism postulates that there is a “reality” existing independently of the process of observation, whilst each observation and attempt to make sense of this reality is a form of social practice. This includes science and social science. Consequently, the study of the social world is constructed through language, meaning and concepts (Danermark, 2002). Pilgrim & Bentall (1999) further state that “our theories of reality, and the methodological priorities we deploy to investigate it” are also a form of social construction (p.262).

“Conceptual abstraction” is the process of isolating aspects of certain events or phenomena in the social world in order to study the underlying generative mechanisms (Danermark, 2002). Furthermore, Danermark (ibid) argues that “abstraction” is necessary, for it allows us to differentiate an object or groups of objects from everything else.

The current project uses theoretical concepts from the fields of psychology, psychiatry and psychoanalysis. “Borderline personality disorder and features” and “treatment-resistant depression” can be considered such conceptual abstractions as they refer to specific characteristics and subjective states that can be attributed to an individual. The researcher aims to link these two concepts in order to build hypotheses about the inter- and intrapersonal processes in operation for those experiencing difficult to treat depression. These hypotheses can in turn be tested further and hopefully used to inform the design of more effective treatments for depression.

At the same time, the reader is reminded of some of the limitations of psychiatric diagnosis and language, which have been noted in the Introduction chapter. Psychiatry as a discipline has been subjected to very different pressures over the last century: from needing to “prove” itself as being “a science” in its own right in the second half of the 20<sup>th</sup> century, to more recently being

criticised for: its focus on individual “pathology” (as opposed to context), having a technological (as opposed to ethical) stance to clinical practice, and for using coercive practices more often than collaborating with patients in a reciprocal manner (Bracken & Thomas, 2001).

The author of this thesis is aware that by choosing to use psychiatric terms (such as “BPD” and “TRD”) she is inadvertently adopting psychiatry’s position in relation to the experiences of low mood and interpersonal difficulties. At the same time, an attempt is being made throughout this thesis to remain attuned of the ongoing conceptual and clinical debates in the field of mental health, and to thus sustain a more critical and reflective position. The discussion chapter of this thesis will give a closer consideration to the limitations of the concepts of “borderline personality features” and “treatment resistant depression”.

A central aim of the research, as outlined in the previous chapter, is to establish whether borderline personality features at the start of an intervention are in any way associated with its outcome, and whether changes in borderline features are associated with changes in depression and treatment outcome. The issue of studying causality has been discussed from an epistemological point of view by a number of philosophers. David Hume, for example, stated that “causation” is not something that exists “out there” in the world but is something that we, as human species, add in order to “make sense” of impressions we perceive from the world around us (Hume & Millican, 2007). Danemark (2002) does not dismiss causal relationships as purely constructed, but warns against confusing co-variance for a causal relationship. Moreover, she states that:

“(…) a causal statement does not deal with regularities between distinct objects and events (cause and effect), but with what an object is and the things it can do by virtue of its nature. This also entails that objects have the causal powers and liabilities they have, independently of any specific pattern of events. The mechanism is not only existent when A leads to B, but also when A does not lead to B; this is a cardinal point in critical realist

causal analysis, and has far-reaching consequences for social scientific explanations”

(Danermark, 2002, p. 55).

The researcher aims to adopt a similar stance to studying borderline personality features and how they link to treatment outcome in patients with difficult-to-treat depression. Engaging in a therapeutic interaction and addressing one’s difficult emotional and relational experiences is thought of as a multi-layered and multi-determined social phenomenon and the current project is but making a humble attempt to bring into light some of the operating processes in the name of better future treatment provision.

## **2.2. Design and Participants**

In order to meet the aims specified above, the current research project used patient data already available as part of a Randomised Control Trial – The Tavistock Adult Depression Study (TADS). The TADS compared the effectiveness of 18-month psychoanalytic psychotherapy to treatment as usual (TAU) for treatment-resistant depression (TRD) (Fonagy et al., 2015; Taylor et al., 2012). TRD was defined by its duration (a minimum of two years), the number of unsuccessful previous treatments (a minimum of two, at least one of which was with anti-depressant medication), and the severity of depressive symptoms.

The TADS’s study protocol was registered with the International Randomized Control Trial Number Register (ISRCTN40586372) (Fonagy et al., 2015).

A summary of the TADS method and participants is presented next in this chapter, followed by information about the specific data used for the purposes of the current study, the measures used both directly and indirectly by the researcher, and the procedure adopted in assessing personality features and testing their relationship with treatment outcome.

The TADS study protocol (Taylor et al., 2012) and the TADS outcome paper (Fonagy et al., 2015) both use the term “patient” when referring to the study participants. The current project also adopts this term, in order to avoid confusion.

### **2.2.1. Ethical approval and participant consent**

The TADS was granted full ethical approval by the Institutional Review Board of the NHS West Midlands Research Ethics Committee (Appendix C). This approval included secondary data analyses by the TADS research team. The current project did not involve any additional contact with the TADS patients. It was therefore not deemed necessary to apply for another NHS Ethics Approval, as data that have already been collected during the original RCT were used for the purposes of the project. The project was, however, granted ethical approval by the University of Essex (Appendix D).

Participants in the TADS RCT had not given their consent explicitly for their data to be used for the purposes of the current research project. They had, however, given their written consent for the data collected in the course of the trial to be used by the TADS research team for the purposes of secondary analyses past the treatment and follow-up phases (Appendix E). The researcher joined the TADS team on an honorary contract for the duration of her work on the current project (Appendix F). Additional consent was therefore not sought from the TADS participants. Ethical considerations related to this will be discussed in more detail in a later section of this chapter.

### **2.2.2. Recruitment**

The recruitment for the TADS trial took place between 2002 and 2009, through General Practitioners (GPs) working in Primary Care Trusts (PCTs) in Central and North London (Taylor et al., 2012). 425 GPs had been approached, 119 out of whom had subsequently agreed to refer



patients to the trial (ibid). Taylor et al. (2012) do not specify the reasons the rest of the GPs gave for not referring their patients to the TADS.

In total 308 patients were screened for eligibility (Fonagy et al., 2015). The TADS inclusion criteria (Fonagy et al., 2015; Taylor et al., 2012) were:

- Age 18-65
- Current DSM-IV diagnosis of major depressive disorder as assessed by the Structured Clinical Interview for DSM-IV Axis I disorders (SCID-I) (First, Gibbon, Spitzer, & Williams, 1996)
- The current depressive episode having lasted for at least two years
- A minimum score of 14 on the 17-item Hamilton Depression Rating Scale (HDRS-17) (Hamilton, 1960), as well as a minimum score of 21 on the Beck Depression Inventory II (BDI-II) (Beck et al., 1996)
- At least two previous unsuccessful treatment attempts, at least one of which was with anti-depressants. No time-frame was specified for this criterion.

The exclusion criteria, were:

- Having received psychodynamic psychotherapy in the past two years
- Meeting the DSM-IV criteria for psychotic or Bipolar I disorder (currently, or in the last five years)
- Having been in psychiatric treatment for substance dependence in the last two years
- Having moderate or severe learning disability
- Evidence of organic brain disorder

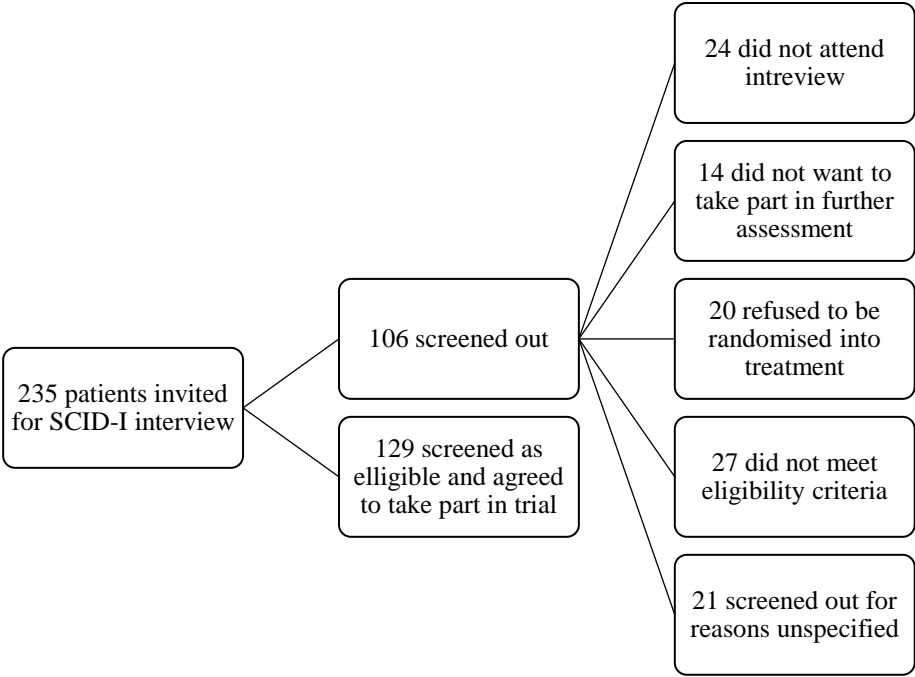
The current study did not include any additional inclusion and exclusion criteria. For a more detailed description of the recruitment process and procedures, refer to Fonagy et al. (2015) and Taylor et al. (2012).

### **2.2.3. TADS eligibility screening and baseline assessment**

Patients referred by their GP and considered to meet the eligibility criteria were invited for the initial SCID-I interview at the Adult Service of the Tavistock & Portman National Health Service (NHS) Foundation Trust, London (Fonagy et al., 2015). Figure 1 summarises this stage of the screening process, based on the information provided by Fonagy and colleagues (ibid).

Figure 1

*TADS Patients' Pre-randomisation Screening*



A statistical analysis compared the demographic characteristics of those who refused to take further part to those who remained in the trial, and no significant differences between the two groups were reported (Fonagy et al., 2015).

A total of 129 patients were randomly allocated either to an 18-month course of weekly psychoanalytic psychotherapy, provided at the Tavistock Centre, London, or to treatment as usual, which was managed by the referring practitioner. The follow-up period for all patients was two years. Fonagy et al. (2015) reported that the randomisation was carried out offsite by an independent statistician. The randomisation procedure balanced for gender, depression severity and medication (on or off) (ibid).

All patients had to complete a range of assessments, at baseline and every three months during treatment. Measures were completed at the end of treatment too, and every six months during follow-up. The author of this thesis was not involved in the collection of any of these data. The list of measures and a very brief description of each are summarised in Table 1 overleaf. More

detailed information about the measures used in the current study is provided later in this chapter. The current study uses available data on all 129 patients who took part in the TADS.

Table 1

*Measures Used in the TADS Trial*

<b>Measure</b>	<b>Authors</b>	<b>Brief summary of measure</b>
Hamilton Depression Rating Scale (HDRS)	Hamilton, 1967	Clinician-rated measure for depression
Beck Depression Inventory (BDI)	Beck et al., 1996	Self-report measure for depression
Clinical Outcomes in Routine Evaluation (CORE)	(Evans et al., 2000)	Self-report measure of general psychological distress
Global Assessment of Functioning (GAF)	APA, 1994	Clinician-rated measure of general functioning
Persons Relating to Others Questionnaire – Version 2 (PROQ2)	Birtchnell, 1999	Self-report measure of object relations
Quality of Life Enjoyment and Satisfaction Questionnaire (Q-les Q)	Endicott, Nee, Harrison, & Blumenthal, 1993	Self-report measure of quality of life specifically developed for people with depression
Client Service Receipt Interview (CSRI)	Beecham & Knapp, 1992	Clinician-rated interview for collecting data on service utilization and calculating comprehensive costs
The Structured Clinical Interview for DSM-IV (SCID-I)	First et al., 1996	Clinician-rated interview of Axis-I diagnoses
Shedler-Westen Assessment Procedure (SWAP-II)	Westen, Waller, Blagov, Shedler, & Bradley, 2007	Clinician-rated measure of personality disorders and “personality health”
Private Theories Interview	Ginner, Werbart, Lavander, & Sahlberg, 2001	Semi-structured interview eliciting information on patient theories about their problems’ formation, pathogenesis and cure, as well as theories of change

#### 2.2.4. TADS baseline characteristics

Table 2 presents a summary of the baseline demographic and clinical characteristics for the 129 patients randomised in the study.

Table 2

*TADS Patients' Baseline Characteristics* (Fonagy et al., 2015)

	LTPP group (N=67)	Control group (N=62)
Age (years, mean $\pm$ SD)	42.7 $\pm$ 10.4	46.1 $\pm$ 9.9
Gender (female, %)	66.7	66.1
Currently married or cohabiting (%)	17.9	17.7
Living alone (%)	82.1	82.3
Tertiary education (%)**	59.7	35.5
Current employment (%)*	52.2	29.0
Receiving state benefits (%)**	41.8	64.5
Duration of depressive illness (years, mean $\pm$ SD)	24.4 $\pm$ 11.6	19.6 $\pm$ 10.8
Duration of current episode (years, mean $\pm$ SD)	3.7 $\pm$ 3.4	3.8 $\pm$ 2.6
Previously failed treatment attempts (N, mean $\pm$ SD)	3.5 $\pm$ 1.4	3.9 $\pm$ 1.8
Previous suicide attempts (N, mean $\pm$ SD)	0.9 $\pm$ 1.3	0.9 $\pm$ 1.3
HDRS-17 score (mean $\pm$ SD)	19.8 $\pm$ 5.1	20.4 $\pm$ 4.9
HDRS-17 severe or very severe depression (%)	53.7	59.6
HDRS-17 moderate depression (%)	34.3	33.9
HDRS-17 mild depression (%)	11.9	6.5
BDI-II score (mean $\pm$ SD)	36.5 $\pm$ 10.1	36.7 $\pm$ 9.5
BDI-II severe depression (score >29) (%)	74.6	77.4
Any comorbid anxiety disorder (%)	73.1	77.4
Any comorbid substance use disorder (%)	19.4	17.7
Any comorbid eating disorder (%)	16.4	9.7
Current Axis I diagnoses (N, mean $\pm$ SD)	3.5 $\pm$ 1.4	3.2 $\pm$ 1.4
GAF score (mean $\pm$ SD)	49.1 $\pm$ 7.0	48.8 $\pm$ 6.1
GAF <50 (%)	53.7	56.5
CORE global distress score (mean $\pm$ SD)	22.8 $\pm$ 6.0	22.5 $\pm$ 6.1
CORE severe distress (score >26) (%)	44.5	40.0

LTPP – long-term psychoanalytic psychotherapy, HDRS-17 – 17-item Hamilton Depression Rating Scale, BDI-II – Beck Depression Inventory - II, GAF – Global Assessment of Functioning, CORE – Clinical Outcomes in Routine Evaluation

\*p<0.02, \*\*p<0.01

### **2.2.5. Treatments**

The following section describes the nature of each of the two TADS treatments .

#### ***2.2.5.1. Long-term psychoanalytic psychotherapy (LTPP) for treatment-resistant depression***

The main characteristics and guiding principles of long-term psychodynamic psychotherapy were discussed in the introduction chapter. This section is concerned specifically with the 18-month psychoanalytic psychotherapy that formed one of the two treatment arms in the Tavistock Adult Depression Study.

Sixty-seven patients were randomised to the LTPP arm of the study, which consisted of 60 sessions (each 50-minutes long) with a clinician accredited by the British Psychoanalytic Council (Fonagy et al., 2015; Taylor, 2015). Once-weekly psychotherapy, time-limited to up to two years, is considered “standard treatment” at the Tavistock and Portman NHS Foundation Trust, where publicly-funded interventions are provided (Taylor, 2015). There were 22 treating clinicians in the LTPP arm of the study, and their average length of clinical experience was 17.45 years (Fonagy et al., 2015).

Taylor (2015; 2003) authored a Treatment Manual specifically written for the purposes of the TADS. The Manual is described by Taylor (2003) as a “description of the psychoanalytic way of working” (p.4), rather than a prescriptive document that lists specific procedures that should be followed rigidly. Furthermore, it highlights the necessity for each therapist’s individual approach to each individual patient, and the importance of the use of clinical judgement by the therapist. The Manual had therefore been developed primarily in order to enable treatment adherence evaluations to take place, rather than to instruct the psychodynamic therapists what to do in their work with the patients (ibid) or to constitute psychoanalytic training in itself (Taylor, 2015). Treatment adherence was indeed assessed as part of the TADS, through the use of the 100-item

Psychotherapy Process Q-sort (Jones, 2000), and a high correlation (82.2%) was reported with the psychodynamic prototype of the measure (Fonagy et al., 2015).

#### **2.2.5.2. *Treatment as usual (TAU)***

The treatment of the patients in the the TAU group was managed in Primary Care by the referring clinician (the GP) (Fonagy et al., 2015). The National Collaborating Centre for Mental Health in the UK has published a specific guidance which recommends evidence-based interventions for individuals diagnosed with depression (National Collaborating Centre for Mental Health (Great Britain) & Royal College of Psychiatrists, 2010). The treatments recommended by this guideline are anti-depressant medication as first line of treatment, followed by Cognitive Behavioural Therapy (CBT) or Interpersonal Therapy (IPT). Long-term psychoanalytic psychotherapy is not included in the guideline.

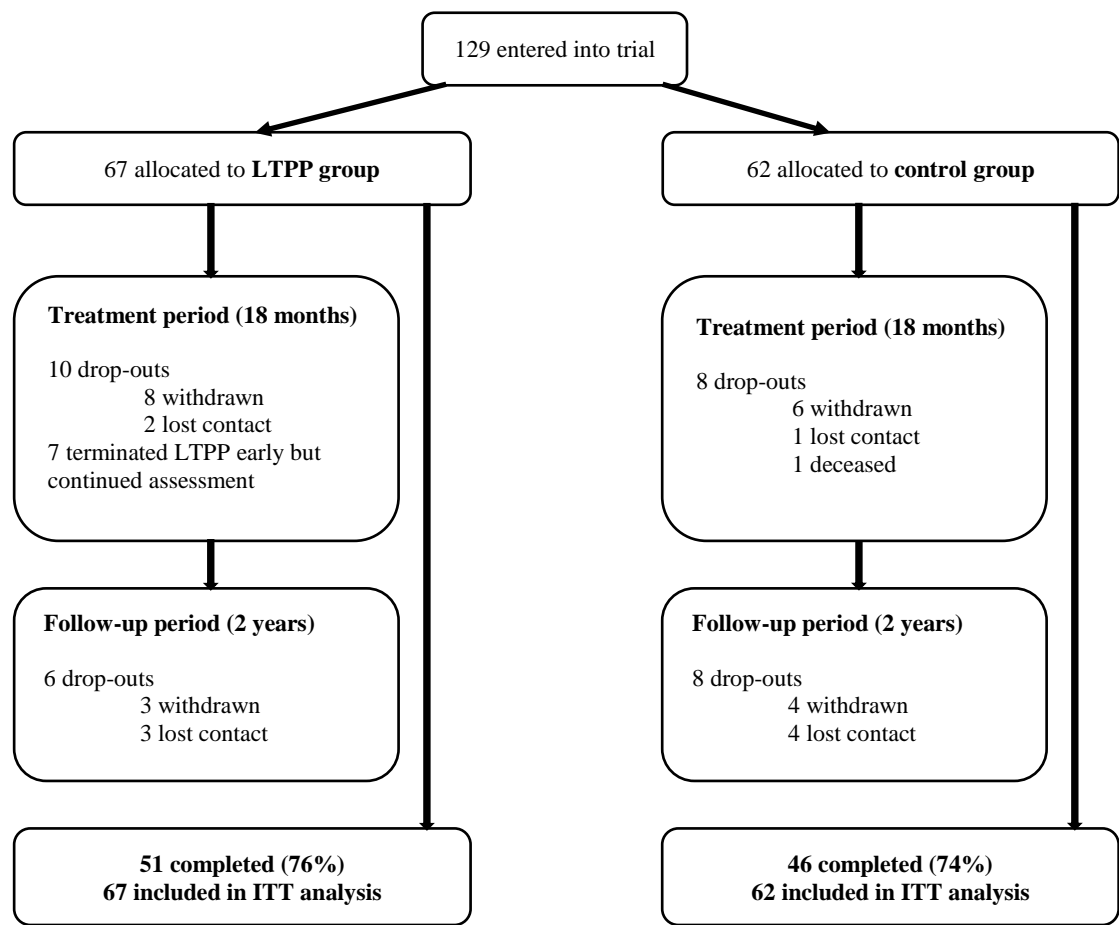
Patients randomised to the TAU group were therefore not in receipt of LTPP. Likewise, those in the LTPP group were not offered any of the psychological interventions recommended by the the National Collaborating Centre for Mental Health (ibid). However, 82% in the LTPP group and 80.7% of patients were in receipt of anti-depressants prior to randomisation, and the average number of medications per patient was 2.1 and 2 for each of the two groups respectively (Fonagy et al., 2015).

#### **2.2.6. Study attrition**

The attrition rates for the LTPP group and the TAU group were similar – 24% and 26% respectively (this included attrition during the two-year follow-up period). Figure 2 (Fonagy et al., 2015) provides details of the drop-out numbers for each group and at each stage of the study.

Figure 2

*TADS Treatment Allocation and Attrition Rates* (adapted from Fonagy et al., 2015)





### **2.3. Current Study Procedure**

#### **2.3.1. Retrospective assessment of personality**

As specified at the beginning of this chapter, the author was appointed as an Honorary Researcher in the TADS team in March 2014. This provided authorised access to the TADS patient data for the initial purpose of carrying out retrospective personality assessments of the 129 patients who took part in the TADS trial. The personality assessments were carried out by a small team of five researchers (two of them were paid and the other three were appointed on honorary contracts). The five researchers were supervised by the TADS project coordinator, Felicitas Rost. All five researchers attended initial training in the use of the SWAP–II, delivered by the TADS project coordinator. The training included practice scoring the of the SWAP–II and team discussions about the scoring process.

The researchers used the Tavistock Psychodynamic Interview (TPI) and the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID–I) in order to conduct the retrospective personality assessments for all 129 participants, using the Shedler-Westen Assessment Profile II (SWAP-II) (Shedler & Westen, 2007). Approximately half of the patients were rated on the SWAP–II by two researchers at baseline and at the end of treatment, in order to establish acceptable inter-rater reliability. Where this was not achieved, a third rating was obtained for the specific patient. The remaining half of the patients were rated by one researcher. The inter-rated reliability statistics will be presented in the results chapter.

The author of this thesis completed 26 SWAP–II ratings at baseline and 20 at the end of treatment (including single, double, and triple ratings). This consisted of listening to approximately 150 hours of clinical material (SCID-I and TPI interviews), in order to rate patients on the SWAP-II. The author also took part in regular team meetings and discussions related to the completion of the SWAP-II profiles.

Table 3 provides a summary of the TADS SWAP–II rating schedule.

Table 3  
*SWAP – II Rating Schedule*

	Single-rated	Double-rated	Triple-rated	Not rated
Baseline	64	57	7	1 – not enough data
End of Treatment	47	52	11	19 – not enough data 1 withdrawn 2 DNA 5 lost contact 1 deceased

A brief description of the SCID-I and the TPI is presented next, followed by a more detailed description of the SWAP–II. This chapter finishes with a description of the other measures used by the researcher for the purposes of the current study.

**2.3.2. Measures used**

***2.3.2.1. Structured Clinical Interview for DSM-IV Axis-I disorders***

The Structured Clinical Interview (SCID) (First et al., 1996) is a semi-structured interview, designed to assess patients on the five axes of the Diagnostic and Statistical Manual for mental disorders, 4<sup>th</sup> Edition (DSM-IV) (APA, 1994). The SCID-I assesses individuals for Axis-I conditions, such as Major Depressive Disorder, Anxiety Disorder and Panic Disorder, to name a few.

The SCID-I’s reliability and validity have been studied by a number of authors (Lobbestael, Leurgans, & Arntz, 2011; Zanarini et al., 2000; Zanarini & Frankenburg, 2001). The Structured Clinical Interview for DSM Disorders Official Website (2015) summarises the SCID-I reliability Kappa values as reported in “the most comprehensive” published studies to date. These range from .35 for Dysthymic Disorder (Zanarini et al., 2000) to 1.0 for Post-Traumatic Stress

Disorder (Zanarini & Frankenburg, 2001). The majority of the Kappa values reported in these studies are above .70, however, which is considered an indicator for good level of agreement.

With regards to its validity, the SCID-I has been considered the “gold-standard” in the field of psychiatric diagnosis (Shear et al., 2000). At the same time, however, it is recognised that “a gold standard for psychiatric diagnoses remains elusive” and the term *best estimate diagnosis* is suggested instead (The Structured Clinical Interview for DSM Disorders Official Website, 2015). Also known as the “LEAD standard”, the best estimate diagnostic approach is longitudinal in nature, and combines structured clinical tools, like the SCID-I, with clinical observations, and information gathered from family members and medical records (ibid).

The SCID-I was initially used during the recruitment and screening stage of the TADS, as specified earlier in this chapter. The measure was repeated at the end of treatment for both arms of the TADS. The interview was slightly amended to fit the purposes of re-assessment at the end of the trial. This included changing some of the language, to acknowledge the follow-up nature of the interview, and incorporating the HDRS-17 questions within the SCID-I, in order to avoid delivering two separate clinical interviews. The HDRS-17 questions were clearly demarcated in the follow-up SCID-I interview schedule.

#### ***2.3.2.2. Tavistock Psychodynamic Interview***

The Tavistock Psychodynamic Interview (TPI) is a semi-structured clinical interview that was specifically designed for the purposes of the TADS. Taylor et al. (2012) describe the TPI as a tool which “draws on well-validated psychodynamic and attachment-based interviews” (p.7). These interviews are the Adult Attachment Interview (Main, Kaplan, & Cassidy, 1985), the Quality of Object Relating Scale (Piper, McCallum, & Joyce, 1993) and the Current Relationships Interview (Crowell & Owens, 1998). By incorporating aspects of these three interviews, the TPI aims to obtain information about the patient’s attachment patterns, as established in early life development, as well as the nature of the patient’s current object

relations. In addition, the TPI focuses closely on key transitional periods during childhood, adolescence and adulthood, and the way in which relationships and events in the patient's life have been cognitively and emotionally processed. The interviewer asks the patient to relay an early life memory and a recent dream, as these are believed to provide further insight into the patient's internal representations of self and others. Overall, however, the interview encourages the patient to speak freely, and the clinician limits prompting as much as possible (Carlyle, 2015). The interview does not rely on interpretations or psychodynamic formulations of the patient's difficulties (ibid).

The TPI was carried out by a trained TADS clinician at baseline, and was repeated two years later (six months after treatment completion for the LTPP group). Whilst the initial interview sought to arrive at an "independent psychodynamic formulation of the participant's illness", the end of treatment interview focused on noticing any changes in interpersonal and psychological functioning. More specifically, the patient was "asked to comment on their relationships, work, physical health, functioning and depression history over the intervening period" (Carlyle, 2015). The patient was also invited to comment on the treatment/support they had received, if any. The request for a recent dream and a childhood memory was also repeated. The clinician was again aiming to provide a free form of narrative, in order to ascertain whether any inter-psychic changes had taken place since the first interview was carried out. Examples of such anticipated changes are the development of "initiative", "curiosity", "sense of self", "capacity for responsibility" and "coherence of self" (ibid).

All SCID-I and TPI interviews at baseline and end of treatment were audio recorded. The current study used the recordings in order to carry out the SWAP-II assessments. The SCID-I and TPI combined have an average length of 2-3 hours for each stage of assessment (pre- and post-treatment). The five researchers who completed the SWAP-II ratings were blind to the type of treatment the patient was randomised at baseline (LTPP vs. TAU). Researcher-blinding was not

fully possible for the end of treatment interviews, however, as some of the interview material contained information about the type of treatment received by the patient. The interview material for each patient was randomly allocated to the researchers. Baseline and end-of-treatment assessments for the same patient were not carried out by the same researcher.

**2.3.2.3. The Shedler-Westen Assessment Procedure, Second Edition (SWAP-II)**

The Shedler-Westen Assessment Procedure (SWAP) was developed by Professor Drew Westen and Dr Jonathan Shedler (Westen & Shedler, 1999b, 1999c). The SWAP-II (Appendix G) is the third edition of the SWAP. It is a 200-item personality measurement tool, in which the clinician rates the patient, without asking them direct questions, as with other existing Axis II diagnostic tools. Rather, “the clinical assessor sorts the [200] statements into eight categories based on the degree to which the statements describe the patient, from 7 (*highly descriptive*) to 0 (*not descriptive*)” (Westen & Shedler, 2007, p. 810). The item distribution is fixed – only a pre-determined number of statements can go into each rating group (Table 4).

Table 4  
*SWAP-II Item Count per Score* (adapted from Blagov et al., 2012)

Score	0	1	2	3	4	5	6	7
Frequency	100	22	18	16	14	12	10	8

This method of assessing personality is known as the Q-sort method (Block, 1961; Westen & Shedler, 1999b). For a comprehensive description of the Q-sort method, the interested reader is referred to Block’s (1961) original text.

**2.3.2.3.1. SWAP – II development and aims**

Shedler and Westen developed the SWAP-200 with three main aims in mind: to refine the existing Axis II personality disorder taxonomy; to suggest an alternative taxonomy for classifying personality disorders; and to identify factors and trait dimensions relevant to

describing personality (Westen & Shedler, 2007). The authors argued that such new taxonomy was necessary for a number of reasons.

First, they highlighted a number of shortcomings of the existing DSM-IV Axis II classification of personality disorders. These included: high comorbidity amongst the 10 personality disorders (and hence poor discriminant validity of the classification); discrepancies between the Axis II categories and empirical data from cluster and factor analyses; a categorical (present/absent) approach to personality disorders, which makes little clinical and empirical sense and neglects individuals who do not meet diagnostic thresholds but nonetheless experience persistent and enduring personality difficulties; lack of acknowledgment of personality strengths, which might counter-indicate personality disorder diagnosis for some individuals; and lack of weighting of specific personality traits, which might bear particular relevance to clinical assessment and treatment (Westen & Shedler, 1999b).

Second, Westen and Shedler (*ibid*) pointed at a number of limitations of the personality disorder assessment tools used at present. For example, they argued that whilst all tools rely on either self-report or direct questioning of the individual, this is not a reliable way of measuring personality processes, as individuals are often unaware of certain personality traits or unwilling to disclose those (in the case of narcissism, for example), or both. When this is combined with the inherent flaws in the DSM-IV classification of personality disorders, on which the assessments are based, a poor validity of these tools results (*ibid*). Furthermore, a large-scale study which asked 1800 experienced psychologists and psychiatrists how they arrive at personality disorder diagnosis in clinical practice, concluded that direct questioning of the patients is rarely adopted in clinical work, and the preferred approach is making clinical inferences based on listening to the patients' narratives and observing the patients' behaviours in relation to the clinician (Westen, 1997).

In order to address these shortcomings, Shedler and Westen developed an alternative assessment and classification tool, the SWAP-200. They stated that the 200 personality descriptions were derived from multiple sources, including DSM-III and DSM-IV diagnostic criteria, items from Axis I which they thought to be relevant to personality functioning, literature from the last 50 years on normal and pathological personality, research on coping and defensive processes, and a number of pilot studies (Westen & Shedler, 2007). Furthermore, the authors claimed that the SWAP was edited multiple times and refined in line with further research since its first version. The SWAP-II was therefore described as the product of ongoing work that had spanned over 12 years of clinical consultations and research (ibid). Shedler (2015) described the clinicians who had taken part in the SWAP development as coming from various theoretical backgrounds (e.g. psychoanalytic, behavioural, etc.), the SWAP thus presenting a shared clinical vocabulary, which does not favour a particular school of thought. Of note, however, only 30% of the clinicians approached (the total number was not specified in the original publication), agreed to take part in one of the studies (Westen, Waller, Shedler, & Blagov, 2014), and no significant demographic differences were reported between those who took part and those who did not, it is unclear whether the remaining 70% of clinicians would have taken a similar approach to describing personality pathology through the SWAP.

In the development stages of the SWAP, clinicians were asked to use the tool in order to rate real-life patients (who were or were not diagnosed with an Axis II personality disorder, but did experience enduring personality difficulties), as well as to rate a “typical” patient for each of the 10 DSM-IV personality disorders: in other words, to describe the personality of a patient diagnosed with, for example antisocial personality disorder, in what they believed to be “the purest form of the condition” (Westen & Shedler, 1999b). In the same study, clinicians were also asked to describe an actual or prototypical healthy, high-functioning patient. In order to avoid an artificial inflation of pathology due to the fixed distribution, Westen and Shedler claim to have

included a wide range of items, including ones describing personality traits not severe enough to warrant Axis II diagnoses, as well as items describing healthy functioning and strengths. A full description of the SWAP-200 and SWAP-II development is beyond the scope of this chapter, but is available in Westen and Shedler's original journal articles (Westen & Shedler, 1999b, 1999c, 2007).

Last, the SWAP-II arguably attempts to address the schism between clinical practice and research by harnessing clinical judgement and inference, and systematising it in an empirical, quantifiable way, which allows for reliable diagnoses and predictions to be made, and for treatment to be planned in a person-centered manner (Shedler, 2015).

#### 2.3.2.3.2. *SWAP personality syndromes and personality traits*

The Q-sort method of assessing personality allows for data collected through the SWAP to be aggregated and analysed in order to derive personality descriptions of a particular type of patient (Westen & Shedler, 1999b). Data collected from the initial SWAP studies were used to derive personality clusters/syndromes based on both hypothetical patients who were deemed to meet criteria for each of the Axis II diagnoses, as well as on real-life patients, as they were described by their treating clinician. Westen & Shedler (1999a; 1999b; 2007) used this data to revise the existing PD constructs, as well as to empirically derive alternative personality configurations. They ran two main types of statistical analyses in order to meet these objectives: Q-sort analysis, which aggregates patients with similar profiles and empirically derives *personality syndromes* (Westen et al., 2012), and trait factor analysis, which derives discrete *trait dimensions* across the set of SWAP items (Westen et al., 2014).

Westen and Shedler define personality syndromes as:

“... diagnostic groupings (...) defined by empirically derived prototypes— descriptions that represent each diagnostic syndrome in its ideal or pure form (based on all 200 SWAP items).



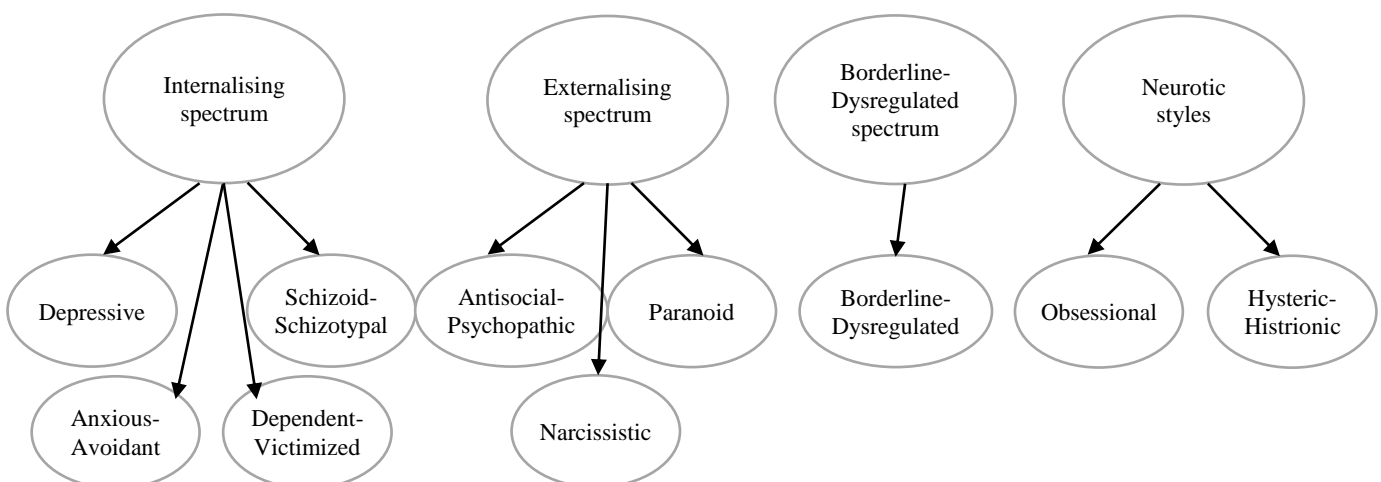
Individual patients are diagnosed dimensionally (on a continuum) based on the degree of resemblance or match with the prototype.” (Westen & Shedler, 2007, p. 815)

This degree of resemblance is statistically calculated as a *T score*, which has a mean of 50 and a standard deviation of 10. The higher the score, the closer the resemblance to the diagnostic syndrome in question. A cut-off score of 60 is used for the purposes of categorical diagnosis (presence/absence of personality disorder), whereas a score between 55 and 60 is considered indicative of significant presence of features of the particular syndrome.

In their latest large-scale study, which collected SWAP data on 1201 patients, Westen et al. (2012) statistically derived 10 diagnostic syndromes: *depressive*, *anxious-avoidant*, *dependent-victimised*, *schizoid-schizotypal*, *antisocial-psychopathic*, *narcissistic*, *paranoid*, *borderline-dysregulated*, *obsessional* and *hysteric-histrionic*. In addition, a *personality health* syndrome (Appendix H) was also derived, denoting the key characteristics of individuals who are highly-functioning. Each syndrome included between 15 and 24 SWAP items, which statistical analysis indicated as correlating highly with the overall profile. Moreover, the analysis also yielded a first order hierarchical factor structure, which is depicted in Figure 3 below.

Figure 3

*Hierarchical Structure of Personality Diagnoses* (adapted from Westen et al., 2012)



In addition to calculating the T-score, the SWAP allows for a narrative account of the patient to be created, which, according to Westen et al. (2012) resembles much more a clinical case formulation as observed in routine clinical practice, in contrast to descriptive psychiatry, which merely provides lists of symptoms.

Furthermore, Shedler & Westen (2010) also gave an example of how a change score can be calculated on an item level, and a new narrative description arrived at, for items where the difference in score was  $>4$ . This approach to examining changes in the personality profiles of the TADS patients was adopted in the analysis of the data in the current study, and the results will be presented in the result chapter to follow.

The current study focuses on the *borderline-dysregulated syndrome*, in particular, which is described by Shedler & Westen (2007) as closest to the DSM-IV Axis II borderline personality disorder. Appendix A provides a list of the 24 SWAP items most descriptive of the borderline-dysregulated syndrome (Shedler, 2015), whilst Appendix B presented the narrative description of the borderline-dysregulated spectrum (Westen et al., 2012).

#### 2.3.2.3.3. *Reliability and validity of the SWAP-II*

Westen & Muderrisoglu (2003, 2006) reported good reliability of the SWAP-200, as measured through the inter-rater coefficients between two clinician raters in a sample of 24 outpatients. More specifically, in the first publication of their results, the authors reported an inter-rater correlations of  $r > .80$  for the personality syndrome scales. The second publication, based on the same sample, reported inter-rater correlation coefficient of  $r > .88$  for the trait dimension scales. Marin-Avellan, McGauley, Campbell, & Fonagy (2005a) reported similarly high inter-rater reliability ( $r > .91$ ). In the same study, as well as in a subsequent one, based on a larger sample (Marin-Avellan, McGauley, Campbell, & Fonagy, 2005b) a strong predictive validity was reported, as measured by independently-assessed factors, such as the nature of the the index

offence committed by the patient and their future violent behaviour. More specifically, individuals who scored higher on the SWAP-200 antisocial PD scale, were more likely to have committed a violent index offence, as well as more likely to engage in violent behaviour on an inpatient ward in the 12 months following the assessment.

Westen, Waller, Blagov, Shedler, & Bradley (2007) reported good validity of the SWAP-II as measured by correlations between cross-informants and correlations with independent measures, such as self-reported arrest history. In addition, Westen & Shedler (2007) summarised the results of a few studies where results on the SWAP were shown to predict a range of external independent criteria, such as suicide attempts, hospitalisations and adaptive functioning as measured by the Global Assessment Functioning index (GAF).

Blagov, Bi, Shedler, & Westen (2012) addressed some of the main criticisms to the validity and reliability of the SWAP-II. For example, they presented counter-arguments to the critique that the SWAP-II's fixed score distribution inflates the T-scores artificially (Block, 2008). For a full discussion of the SWAP-II's reliability and validity, the reader is directed to Blagov et al. (2012).

#### 2.3.2.3.4. *SWAP – II ratings using the SCID-I and Tavistock Psychodynamic Interview*

The SWAP-II is typically scored on the basis of information gathered through the *Clinical Diagnostic Interview* (CDI), which was designed specifically for this purpose (Westen & Muderrisoglu, 2006). The TADS team did not carry out the CDI, however.

Lewis (2008) compared the use of the TPI and the SCID-I to the use of the CDI when rating participants on the SWAP-II. She concluded that the interviews are similar in their content, structure and flow, whilst also highlighting areas of difference. For example, the CDI is designed to be delivered by the same clinician, who then rates the individual, using the SWAP-II. This allows the assessor to observe the patient and their non-verbal/visual presentation and

interactional style – information that is then used to score some of the SWAP items. Lewis (2008) used the SWAP-II to retrospectively score a sub-sample of 25 TADS participants and concluded that despite the inherent limitations, the use of the TPI and the SCID-I is reliable. She based her conclusions on high correlation scores between her SWAP-II and other measures of personality for her sample. Carlyle (2015), who developed the TPI, indeed stated that one of the interview's aims was to be "sufficiently free" in order for other existing or future measures to be applied to the qualitative data collected by it. Furthermore, she suggested that one type of clinical question that might be addressed by the interview subsequently, is whether any "key personality or object relation's indicators" might be linked to specific "outcomes" (ibid).

An anticipated difficulty in the current research, which is not addressed by either Lewis (2008) or the SWAP developers in any of their publications, is the rating of items where there is insufficient information. A score of "0" is generally assigned in order to denote an item as "not descriptive" for the patient. However, it is not unlikely that in the current study some items might well be descriptive of a patient, but for the relevant information to not have been elicited by the SCID-I or the TPI. In such case scores of "0" might be better interpreted as "missing data". Furthermore, it is generally recommended that the SWAP-II is scored either following the administration of the CDI, or, when this is not practical, after a minimum of six clinical hours with the patient (Shedler, 2015; Shedler & Westen, 2007). The deviation from this guideline is clearly a limitation in the current study and this will be addressed further in the discussion chapter of the thesis.

#### *2.3.2.3.5. The use of the SWAP II by newly-trained researchers*

The SWAP-II measure was originally developed and tested through the involvement of hundreds of experienced clinicians (psychiatrists and psychologists). Its effective use relies significantly on "clinical judgement" and expertise. In the current study the measure was used by five junior researchers with some clinical experience. One of the researchers (not the author of this thesis)

had over 10 years of clinically-relevant experience. Whilst the limited clinical experience of some of the researchers might be seen as a potential limitation, both in terms of evaluating the SWAP-II ratings' reliability, as well as ethically, a study looking at the convergence between SWAP-II and self-report (PAI) data for antisocial and borderline PD by Bradley, Hilsenroth, Guarnaccia, & Westen (2007) used "advanced clinical psychology graduate students" to do the SWAP-II assessments and reported good reliability of the measure. In addition, the TADS researchers were supervised by another senior researcher, who has been trained in using the SWAP-II procedure and who is also in possession of vast experience in administering personality assessments for research purposes. This supervisor is also in receipt of ongoing support and guidance by one of the SWAP-II primary developers – Dr Shedler.

### **2.3.3. Other measures**

#### ***2.3.3.1. Structured Clinical Interview for DSM-IV Axis-II disorders – Patient***

##### ***Questionnaire (SCID-II-PQ)***

The SCID-II-PQ is a self-rated personality disorders screening tool (First, Gibbon, Spitzer, Williams, & Benjamin, 1997). It consists of 119 yes/no items, and is not intended to be used as a stand-alone personality disorders measure. Rather, the questionnaire is designed to alert the clinician about a possible PD diagnosis, so that the more thorough semi-structured SCID-II interview is carried out. The SCID-II interview was not conducted as part of the TADS, but all patients were asked to complete the SCID-II-PQ pre- and post-treatment. The SCID-II-PQ alone is not a reliable measure of personality pathology, and it is therefore not used as such in the current study. The author nonetheless analysed some of the SCID-II-PQ in order to cross-reference it with results from the SWAP-II.

### 2.3.3.2. *The Hamilton Depression Rating Scale (HDRS-17)*

The primary outcome measure in the TADS was the the Hamilton Depression Rating Scale (HDRS-17) (Hamilton, 1960). Depression was also measured by the Beck Depression Inventory, 2<sup>nd</sup> Edition (Beck et al., 1996), which was treated as a secondary measure (Fonagy et al., 2015).

The HDRS is a 17-item clinician-rated measure, which covers symptoms such as depression, feelings of guilt, suicide and anxiety (Hamilton, 1960) (Appendix I). Some of the items are rated on a five-point scale, where a score of “0” denotes an absence of the specific symptom, and a score of “4” denotes a high severity of the symptom. Other items are rated on a three-point scale (0-2), the rationale for this being that “quantification of the variable is either difficult or impossible” (Hamilton, 1960, p. 57). The scale also contains four additional items, which are not included in the final scoring. These are *diurnal variation*, *depersonalisation and derealisation*, *paranoid symptoms*, and *obsessional and compulsive symptoms*. Hamilton (1960) justified the exclusion of these items from the final score, by arguing that the first one refers to the type of depression (not its presence and intensity), and the latter three do not occur frequently enough to make a clinical difference.

Unlike other widely-used measures of depression (e.g. the BDI-II), the HDRS-17 requires the rater to be a health-care professional with adequate clinical experience. Hamilton (1960) reiterated the importance of the rater’s ability to make accurate clinical judgements, particularly for items he considered more difficult to define, such as general somatic symptoms and hypochondriasis (ibid). In addition, he recommended that the scale is completed independently by two raters, but that the rating is based on the same clinical interview with the patient, where the final score of the HDRS is the average of the two raters’ scores.

The TADS used the measure in its original form, adding items to track increases in sleep, appetite and weight (Fonagy et al., 2015) (Appendix J). Each patient was rated by a trained interviewer, who was blind to the patient’s treatment allocation, and the interview was

subsequently double-rated by another coder, with an inter-rated coefficient of .89 having been reported by Fonagy et al. (2015). The following severity categories were used in the TADS: 0-7 “not depressed”; 8-13 “mild depression”; 14-18 “moderate depression”; 19-22 “severe depression”; and  $\geq 23$  “very severe depression”. Full remission was denoted by HDRS score of 8 or lower, where as a score of  $\leq 12$  was considered indicative of partial remission (ibid).

Trajković et al. (2011) carried out a meta-analysis of studies which had reported HDRS’s reliability calculations, and had been published between 1960 and 2008. These included both studies designed to test the HDRS’s psychometric properties, and studies of clinical samples where the HDRS was one of the employed measures. Trajković et al. (2011) evaluated the scale’s reliability by estimating internal consistency, inter-rater reliability and test-retest reliability via the meta-analytic method on 409 journal articles. They concluded that overall the HDRS is a reliable assessment tool for depression in clinical settings, as it has good internal consistency, high inter-rater reliability, and yields similar results when repeated on multiple occasions. The authors also highlighted some exceptions to the findings. For example, their results suggested that the “loss of insight” scale item has a very low internal consistency, as measured through total-item correlations. In addition, inter-rater reliability was found to increase significantly the more recent the studies were, suggesting that accumulated experience of using the HDRS and better training have improved the scale’s use. Finally, test-retest reliability was found to be highest when there was a short period between the two assessment episodes and when each assessment was carried out by the same rater. The longer the time period between the assessment episodes, the poorer the test-retest reliability. However, Trajković et al. (2011) highlighted that a longer time lapse might also be related to changes in symptoms, rather than be indicative of a poor test-retest reliability, as such. A more general limitation of this meta-analysis, as identified by its authors, is that of all publications screened, only 7.4% reported reliability coefficients and many of the studies lacked important information (e.g. participants’

characteristics). Thus, the conclusions about the HDRS reliability need to be treated with caution, as they refer to a small subsample of the studies that used the scale.

Bagby, Ryder, Schuller, & Marshall (2004) carried out a review of the psychometric properties of the HDRS, including articles published between 1980 and 2003. Seventy articles were included in the review and the results were presented in relation to main psychometric criteria: reliability, item response and validity. First, the authors reported an overall good internal, inter-rater and test-retest reliability, when the HDRS total score is considered. When examined on an item-level, however, the scale was reported as less reliable. For example, the item “loss of insight” was again found to be problem-ridden. There was also a great inter-rater and test-retest variation for many of the 17 scale items.

Second, further criticism was directed towards the weighting of the individual items (some of them rated on a 3-point scale, whilst others on a 5-point scale). This, in combination with the varying likelihood of ascribing a high/low score to certain items regardless of the severity of depression experienced by the patient, was argued to ultimately result in the scale’s weakened capacity to detect change (*ibid*).

Third, the authors examined five types of validity: content, convergent, discriminant, factorial and predictive. Whilst content validity was reported to be good, the HDRS was criticised for not being adequately constant with the DSM-IV definition of depression. For example, some symptoms, such as “psychotic anxiety” are captured by the HDRS but are not included in the DSM diagnostic criteria, whilst “other important features of DSM-IV depression are often buried within more complex items and sometimes are not captured [by the HDRS] at all” (Bagby et al., 2004, p. 2170). This critique was linked to the reported convergent validity, which had been found in different studies to be good with all other measures of depression, except for two, one of which is the SCID-I. The HDRS discriminant validity was also reported as good overall, but only on the total-score level, and less so on an item level. Nonetheless, a number of studies had



reported the HDRS to have better discriminant validity than the BDI-II. The authors concluded that overall reliability and validity standards appear to be generally met for the HDRS. However, they argued that the scale needs to be revised, firstly as it does not match adequately the contemporary DSM-IV conceptualisation of depression, and secondly, as it does not have a clearly defined factor structure which has proven to be consistent across populations.

The current study uses the HDRS as a main outcome measure. This decision was made in part in order to maintain consistency with the method of the TADS (where the HDRS was used at the primary measure of depression), and partially due to the clinician-rated nature of the HDRS. The use of clinical judgement and inference was shown to be a central feature of the SWAP-II, as described earlier in this chapter. A clinician-rated measure of depression is thus considered to be theoretically consistent with the other main measure used in the current project (the SWAP-II).

#### ***2.3.3.3. Clinical Outcomes in Routine Evaluation – Outcome Measure (CORE-OM)***

The CORE-OM (Appendix K) is a 34-item self-rated generic measure of psychological distress (CORE IMS Ltd, 2016). It covers four domains: well-being, social functioning, problems/symptoms, and risk (to self and others). The total clinical score is the mean score of all rated items, and denotes the individual's current level of overall psychological difficulties. Items are scored on a scale from 0 to 4 (from “not at all” to “most or all of the time”) and refer to the week preceding the completion of the questionnaire. The CORE-OM manual specifies cut-off scores that differentiate clinical from non-clinical populations. The CORE-OM total cut off score for men is 1.19 and for women – 1.29 (CORE System Team, 2016).

The CORE-OM was introduced as a measure of clinical outcome, in response to criticisms of the lack of routine evaluation of psychotherapy interventions (Evans et al., 2000). Its developers aimed at designing a measure, which was short, easy to understand, and compatible with the different schools of therapy (ibid).

With regards to its psychometric properties, the CORE-OM was reported to have high internal consistency and test-retest reliability, as well as excellent convergent validity against other self-report measures used in routine practice. This information was based on an initial study of 2000 participants (counselling/psychotherapy patients and lay-participants), as well as on subsequent data from 23 clinical sites in the UK, and a non-clinical sample (Evans et al., 2000, 2002).

Furthermore, the CORE-OM has been argued to be sensitive to clinical change, and to reliably discriminate between clinical and non-clinical populations (ibid).

A more recent study with over 3000 patients in the UK, also reported good test-retest reliability for the CORE-OM and concluded that the tool measures psychological disturbance reliably (Barkham, Mullin, Leach, Stiles, & Lucock, 2007).

The current study used the CORE-OM total score, as a measure of general psychological distress. The author decided against using the separate CORE-OM domains (e.g. well-being, or social functioning) as recent research has questioned the face validity of the measure's domains and subdomains, and has claimed that the tool is "a large psychological distress scale with considerable item redundancy" (Bedford et al., 2010, p. 51).

#### ***2.3.3.4. General Assessment of Functioning (GAF)***

The Global Assessment of Functioning Scale (GAF) (Appendix L) comprises Axis V of the DSM-IV (APA, 1994). The measure is clinician-rated and aims at evaluating the patient's overall level of functioning in the psychological, social and occupational domains. The APA (1994) states that "the GAF scale may be particularly useful in tracking the clinical progress of individuals in global terms, using a single measure" (p.32). "Intended to be a generic, rather than a diagnosis-specific" measure (Aas, 2010, p.1), the GAF has been widely adopted internationally (ibid).

The GAF is divided into 10 categories of functioning, each of which is rated on a 10-point scale. The clinician is asked to choose a single value that best describes the patient's current level of functioning. When making this decision, the clinician has to consider both symptom severity, as well as functioning. However, to be ascribed to one of the 10 categories, the patient needs to match either the symptom or functioning description of the category, and not both, meaning that "the final GAF rating always reflects the worse of the two" (p.33). For a more detailed description of the GAF rating procedure, please refer to the DSM-IV Manual (APA, 1994).

The reliability of the GAF has been assessed by a number of authors. Jones, Thornicroft, Coffey, & Dunn (1995), for example, evaluated the measure as applied to a sample of 103 outpatients, diagnosed with chronic mental health conditions, such as schizophrenia. The authors reported satisfactory reliability for all cases. They also assessed the GAF in relation to two other measures of clinical need – the level of support a patient requires, and the changes to anti-psychotic medication – and concluded that the GAF reflects adequately symptom and functioning severity. Another study (Startup, Jackson, & Bendix, 2002), on inpatients with diagnosis of schizophrenia, reported excellent GAF inter-rater reliability, at all three measurement time-points (admission, 6- and 12-month follow-up). These authors also correlated the GAF scores with scores on two symptom measures and one measure of social behaviours, and reported significant results for the two follow-up time-points, but not for the point of admission.

Aas (2010) reviewed the GAF literature and concluded that overall reliability is good, although less so in routine clinical settings. Another recent study by Grootenboer et al. (2012) investigated the measure's validity and reliability for over 432 outpatients diagnosed with major depression and treated in "normal clinical practice" in the Netherlands. The authors reported weak inter-rater reliability, and poor discriminant validity. Furthermore, GAF scores were significantly correlated with disease severity, as measured by other clinical assessment tools, as well as with physical functioning, despite the DSM-IV instructing clinicians to not base their ratings on

physical symptomatology. There was also an association between the GAF ratings and a measure of social functioning.

Finally, some authors highlighted the minimal training required for clinicians to be able to complete the GAF, which arguably makes it an easy-to-deliver measure in routine clinical practice (Jones et al., 1995; Startup et al., 2002). Other authors, however, warned against an over-reliance on the GAF as an outcome measure. For example, reviewing the existing GAF literature, Moos, Nichol, & Moos (2002), concluded that “GAF ratings tend to be more closely associated with diagnoses and psychiatric symptoms than with social and occupational functioning” (p.731). Grootenboer et al. (2012) questioned the use of the GAF to determine treatment needs and evaluate the quality of services, given the measure’s poor reliability and validity (as established by their study).

It can thus be concluded that the existing GAF literature points at mixed results, with regards to the measure’s reliability, validity and applicability in treatment outcome research. This will be considered in further detail in the discussion chapter of the thesis.

## **2.4. Ethical Considerations**

### **2.4.1. Confidentiality**

All data has been dealt with, listened to (in the case of audio recordings) and saved in line with the National Health Service Information Governance guidelines (Department of Health, 2003).

### **2.4.2. Personality disorder diagnosis**

It is not unlikely that at least some of the participants will reach the cut-off scores for a personality disorder diagnosis, as measured by the SWAP-II. In such cases it might transpire that the particular individual has not been diagnosed before (they might have completed a DSM-based measurement tool, but not met those sets of criteria), particularly as participants in the TADS were referred for the treatment of depression, and not personality disorder. Whereas other

outcome and diagnostic tools used in the TADS were employed during the treatment and follow-up phases, the SWAP-II was carried out retrospectively, after the participants had left the trial. Yet, no patients were contacted following the end of the trial, in order to be informed of potentially meeting PD diagnostic criteria. This poses the ethical dilemma whether participants are entitled to know the outcome of any retrospective assessments, particularly if that might lead to a re-formulation of their difficulties or additional or alternative forms of interventions/treatments (treatment for personality disorder, in particular).

### 3. Results

This chapter begins with a summary of the SWAP-II scoring, inter-rater reliability correlations and account of the missing values. The chapter then explores more closely the prevalence of the different personality disorders, including borderline-dysregulated personality disorder and features in the TADS patient group. The results of the statistical analysis performed using SPSS version 21, and looking at the relationship between the borderline-dysregulated syndrome and treatment outcome is presented next. The chapter finishes with exploring the relationship between specific borderline personality characteristics and depression, psychological distress and general functioning.

#### 3.1. SWAP-II Scoring and Calculation of Inter-rater Reliability

As specified in the method chapter of this thesis, the SWAP adopts a prototype matching approach to constructing personality profiles.

“A *diagnostic prototype* is a SWAP-200 description of a recognized personality disorder or syndrome. It is not a description of an individual person, but rather a richly detailed description of a disorder or syndrome in its “ideal” or pure form.” (Shedler, 2009, pp. 5–6)

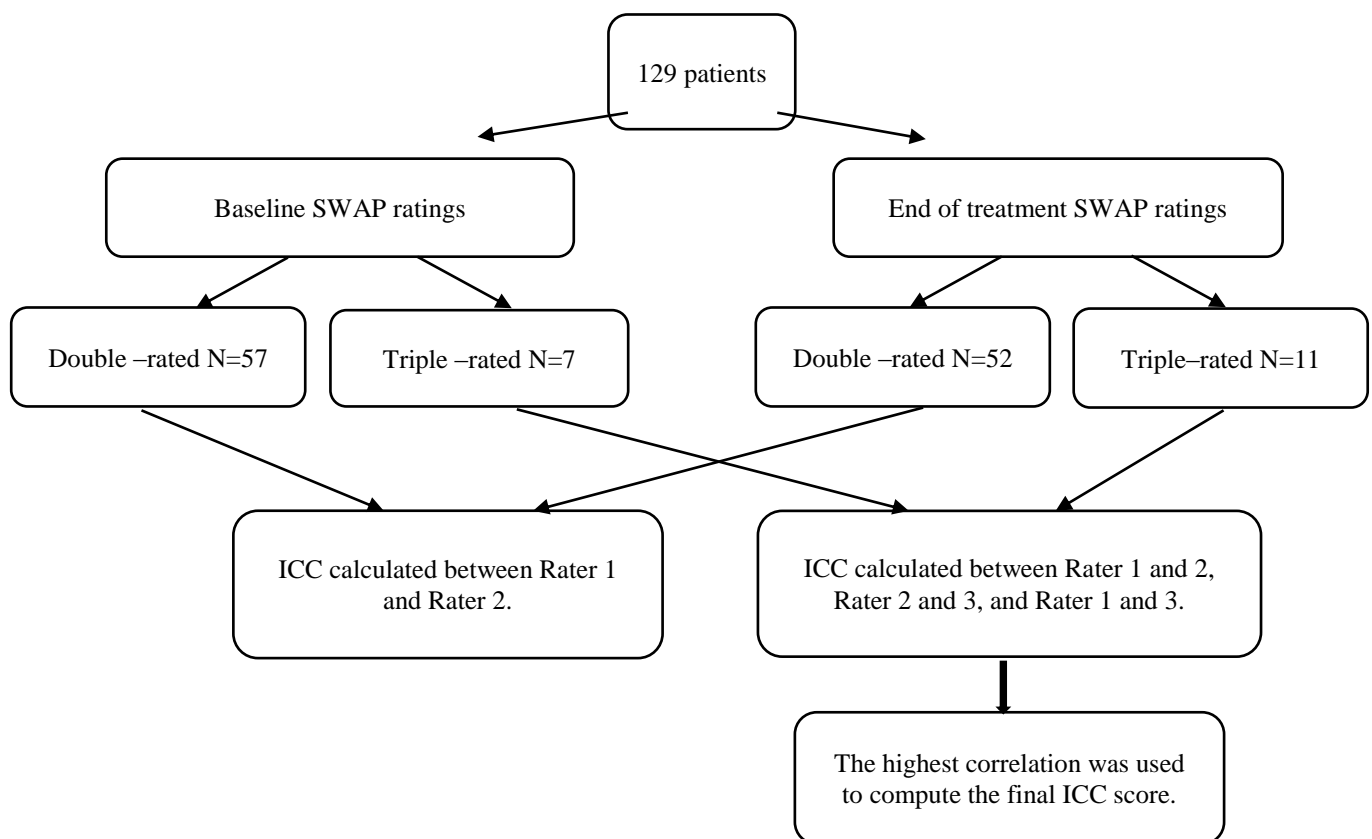
The T-scores, calculated through the SWAP-II algorithm, thus reflect the degree of similarity or “match” between the patients’ SWAP-II score and each of the diagnostic prototypes (“the better the match, the more applicable the diagnosis”, p.6). The SWAP-II calculates T-scores for the 10 DSM-IV personality disorders; for the 11 personality syndromes, derived empirically through the SWAP-II developers’ research; and for the 16 personality traits. The T-score ranges from 0 to 100, where 10 points denote one standard deviation.

### 3.1.1. Overall SWAP-II inter-rater reliability and scoring

The SWAP-II scoring procedure was described in detail in the previous chapter. Figure 4 reminds the reader of the number of patients whose SWAP-II scores were double- and triple-rated and summarises the process of inter-rater calculations. All double-ratings were correlated to establish inter-rater reliability. In addition to calculating a correlation coefficient, an Intraclass Correlation Coefficient (ICC) was calculated by the project co-ordinator (not the author of this thesis) using the SPSS data analysis software package. ICC values  $<.40$  are considered to indicate “poor” inter-rater agreement, ICC values between  $.41$  and  $.75$  - “fair” inter-rater reliability, and values above  $.75$  - “excellent” agreement (Fleiss, 1973; Fleiss, Levin, & Paik, 2013; Shrout & Fleiss, 1979). These criteria were used when interpreting the SWAP-II ICC scores and any cases with poor reliability were rated by a third researcher.

Figure 4

*ICC Calculation for SWAP-II Double- and Triple Ratings*



The average ICC coefficient at baseline, based on 64 double- and triple ratings, was calculated to be .80, and therefore considered as indicating excellent agreement between the raters. The ICC coefficient at 18-months, based on 63 double- and triple ratings was .70, which indicated fair agreement. The Spearman-Brown prophecy formula was also used to compare two raters, which yielded a corrected ICC and correlation co-efficients for both time-points. The corrected ICC coefficient at baseline was .89, and at 18-months it was .83. Appendices Ma and Mb present the full list of ICC and Correlation coefficients for all double and triple SWAP-II ratings at baseline and end of treatment.

The SWAP-II scores were calculated via an SPSS syntax sent by Dr Jonathan Shedler to the TADS project co-ordinator. In short, this entailed entering the raw SWAP-II scores into the algorithm, which calculated a T-score for each of the SWAP-II prototypes and traits. Where participants were double-rated, their data was entered in the syntax for each of the ratings, and the final scores were averaged, producing a single T-score for each of the prototypes and traits. In the case of triple ratings, only the two that had the higher correlation were used, with their resulting T-scores averaged in order to produce a final SWAP-II set of scores.

### **3.1.2. SWAP-II Borderline-dysregulated items inter-rater reliability**

Inter-rater reliability was calculated not only for the SWAP-II measure as whole, but for each of the 200 items at baseline, too. The same statistical procedures were followed, as described above, in calculating correlation and ICC coefficients.

Appendix N presents the ICC and correlation statistics for the borderline-dysregulated personality prototype items. The introduction chapter of this thesis presented the full list of the items, 24 in total. When the Fleiss' (1973) criteria were applied, only half of the items (12) qualified as having fair or excellent ICC coefficients. The author of this thesis used the initial ICC values, as opposed to the corrected ones, as this was considered to be a more conservative estimate of inter-rater reliability.



Table 5 below presents a list of the items with poor inter-rater agreement, and a list of those with fair or excellent one. This information will be used later in the chapter, when borderline-dysregulated prototype items are linked to treatment outcome.

Table 5

*SWAP-II Borderline-dysregulated Items' Inter-rater Reliability*

<b>Poor Inter-rater reliability (<math>r &lt; .40</math>)</b>	<b>Fair – Excellent Inter-rater reliability (<math>r &gt; .40</math>)</b>
Is unable to soothe or comfort him/herself without the help of another person (i.e., has difficulty regulating own emotions).	Emotions tend to change rapidly and unpredictably
Tends to “catastrophize”; is prone to see problems as disastrous, unsolvable, etc.	Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.
When upset, has trouble perceiving both positive and negative qualities in the same person at the same time (e.g., may see others in black or white terms, shift suddenly from seeing someone as caring to seeing him/her as malevolent and intentionally hurtful, etc.).	Tends to become irrational when strong emotions are stirred up; may show a significant decline from customary level of functioning.
Manages to elicit in others feelings similar to those s/he is experiencing (e.g., when angry, acts in such a way as to provoke anger in others; when anxious, acts in such a way as to induce anxiety in others).	Is prone to intense anger, out of proportion to the situation at hand (e.g., has rage episodes).
Tends to draw others into scenarios, or “pull” them into roles, that feel alien or unfamiliar (e.g., being uncharacteristically insensitive or cruel, feeling like the only person in the world who can help, etc.).	Tends to stir up conflict or animosity between other people (e.g., may portray a situation differently to different people, leading them to form contradictory views or work at cross purposes).
Lacks a stable sense of who s/he is (e.g., attitudes, values, goals, and feelings about self seem unstable or ever-changing).	Tends to be needy or dependent.
Is prone to painful feelings of emptiness (e.g., may feel lost, bereft, abjectly alone even in the presence of others, etc.).	Tends to fear s/he will be rejected or abandoned.
Appears to fear being alone; may go to great lengths to avoid being alone.	Tends to become attached quickly or intensely; develops feelings, expectations, etc. that are not warranted by the history or context of the relationship.
Tends to engage in self-mutilating behaviour (e.g., self-cutting, self-burning, etc.).	Tends to feel misunderstood, mistreated, or victimized.

Tends to make repeated suicidal threats or gestures, either as a “cry for help” or as an effort to manipulate others.	Struggles with genuine wishes to kill him/herself.
Tends to act impulsively (e.g., acts without forethought or concern for consequences).	Relationships tend to be unstable, chaotic, and rapidly changing.
Tend to feel unhappy, depressed or despondent.	Work-life and/or living arrangements tend to be chaotic or unstable (e.g., job or housing situation seems always temporary, transitional, or ill-defined).

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Only SWAP-II borderline-dysregulated items which had a “fair” or “excellent” inter-rater agreement were used in the individual item analysis, which will be reported later in this chapter.

**3.2. Missing Values**

**3.2.1. SWAP-II**

A SWAP-II profile at baseline was created for all but one TADS participant. As already presented in the previous section, 7 patients (11.3%) in the TAU group and 12 patients (17.9%) in the LTPP group did not have SWAP-II profiles at the end of treatment. The reasons for this were summarised in the methods chapter of this thesis.

It is unclear what the SWAP-II PD distribution and prevalence would have been, if there was no missing data at the end of treatment. The patients who did not have an end-of-treatment SWAP-II profile completed, might have been very different from those who did have a personality profile for that time point.

To address this issue, the author created two groups in the SPSS data set: of those who had an 18-months SWAP-II profile completed, and those who did not have a profile completed, and compared the mean values for each of the personality syndromes’ T-scores. The means were calculated both for the entire TADS sample, as well as for the TAU and LTPP groups separately. A difference in the means and standard deviations of 3 points or less was considered not clinically significant for further statistical analysis to be carried out. A difference of 4 points or

more was considered significant, as the SWAP-II developers consider a  $>4$  change in score to denote a clinically meaningful difference (Shedler & Westen, 2010). Only the *depressive syndrome* mean-values in the LTPP group met this criteria, with those who did not have a SWAP-II profile completed at 18 months ( $N=12$ ) having a *depressive personality* mean value of 58.42, and those who did have a SWAP-II profile completed at the end of treatment ( $N=54$ ) – a mean value of 63.37.

An independent t-test confirmed that this difference was statistically significant, too ( $t(64) = -2.67, p < .05$ ): participants, for whom a SWAP-II profile was not completed at the end of treatment, scored lower at baseline on the *depressive syndrome scale* ( $58.42 \pm 1.87$ ), than those who had a SWAP-II profile completed at the end of treatment ( $63.37 \pm .77$ ). The difference represented a small-sized effect  $r = .10$ . The implications of this difference for interpreting the rest of the findings will be addressed in the discussion chapter.

### **3.2.2. Hamilton Depression Rating Scale (HDRS-17)**

A number of participants in both the TAU and LTPP groups had missing HDRS-17 values at 18- and 42-months. For the TAU group the missing values were 16 (25.8%) at 18 months and 17 (27.4%) at 42 months. For the LTPP group, 15 people (22.3%) did not have a HDRS-17 score reported at 18 months, and 20 people (29.9%) – at 42 months.

The baseline HDRS-17 mean values of those who completed the measure at 18- and 42- months, and those who did not, were compared, through the same statistical procedure employed with the SWAP-II missing data. No significant differences were identified.

### **3.2.3. Global Assessment of Functioning (GAF) and CORE-OM**

Similar to the HDRS-17 scores, there was a high number of missing values for the other two outcome measures used in the current study – the GAF and the CORE-OM. These are summarised in Table 6 overleaf.

Table 6

*GAF and CORE-OM Missing Values*

Allocation group			<b>GAF rating at Baseline</b>	<b>GAF rating at 18 months</b>	<b>GAF rating at 42 months</b>	<b>CORE-OM at Baseline</b>	<b>CORE-OM at 18 months</b>	<b>CORE-OM at 42 months</b>
<b>TAU</b>	N	Valid	62	44	44	59	38	39
		Missing	0	18(29%)	18(29%)	3(4.8%)	24(38.7%)	23(37.1%)
<b>LTPP</b>	N	Valid	67	52	47	63	45	42
		Missing	0	15(22.3%)	20(29.9%)	4(6%)	22(32.8%)	25(37.3%)

The GAF and CORE-OM baseline mean values of those participants who did not complete the measures at 18- and 42-months were compared to those who completed them. No significant differences were found, indicating that the participants whose values were missing at the latter two time-points, were not better or worse in terms of their global functioning and psychological distress than those for whom data was available throughout.

Fonagy et al. (2015) reported that “missing values were not a major problem” (p.315) in the TADS data analysis, with outcome measures data being available across all time-points for 82% of the participants for the primary measures (HRDS-17) and 75% - for the secondary measures (GAF and CORE-OM). No significant difference in completion rates was reported between the TAU and LTPP groups.

The author decided to treat missing values by excluding them from any subsequent data analyses, as opposed to computing missing values, by, for example, using data available at the nearest other time-point when an outcome measure was completed (e.g. at 12- or 15-months). This decision was made as it was considered that “borrowing” scores from earlier/later time-points will complicate and confuse the subsequent interpretation of the results.

The rest of this chapter presents the results of the different statistical analyses performed in order to investigate the relationship between borderline personality, and treatment outcome. The

SWAP-II provides both categorical, as well as dimensional data. The analysis of each is reported in turn.

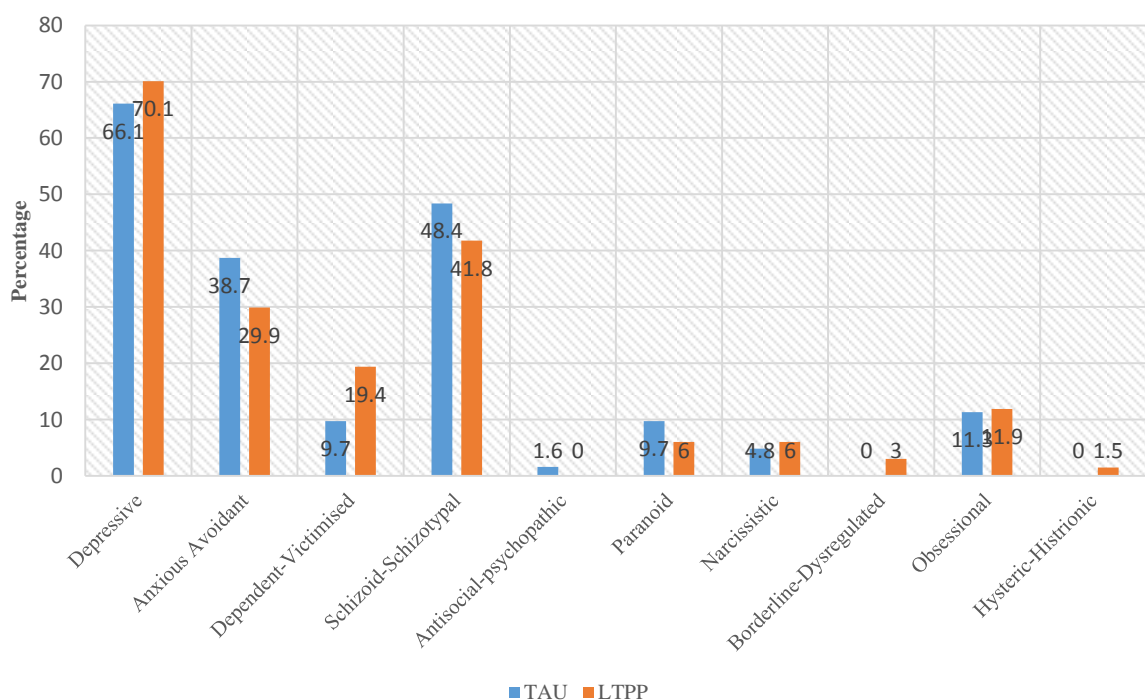
### 3.3. Personality Disorder Features and Diagnoses

The introduction and method chapters described some of the current limitations of the DSM-IV personality disorders taxonomy. The developers of the SWAP-II claim that the empirically derived personality syndromes are an improved alternative to the current Axis-II classification (Shedler, 2015; Westen et al., 2012). The author of this thesis decided to therefore use the ten SWAP personality syndromes, as a primary measure of personality disorder and the SWAP-II “personality health” syndrome, as specified in the methods chapter.

Graph 1 below presents the categorical prevalence for all 10 SWAP-II personality disorders at baseline (TAU N=62, LTPP N=66). The reader is reminded that the SWAP-II cut-off T-score for personality disorder is 60. All participants were rated on the SWAP-II at baseline, except for one person in the LTPP group, where there was insufficient information for the scoring to be completed.

Graph 1

*SWAP-II Personality Disorders Prevalence at Baseline*

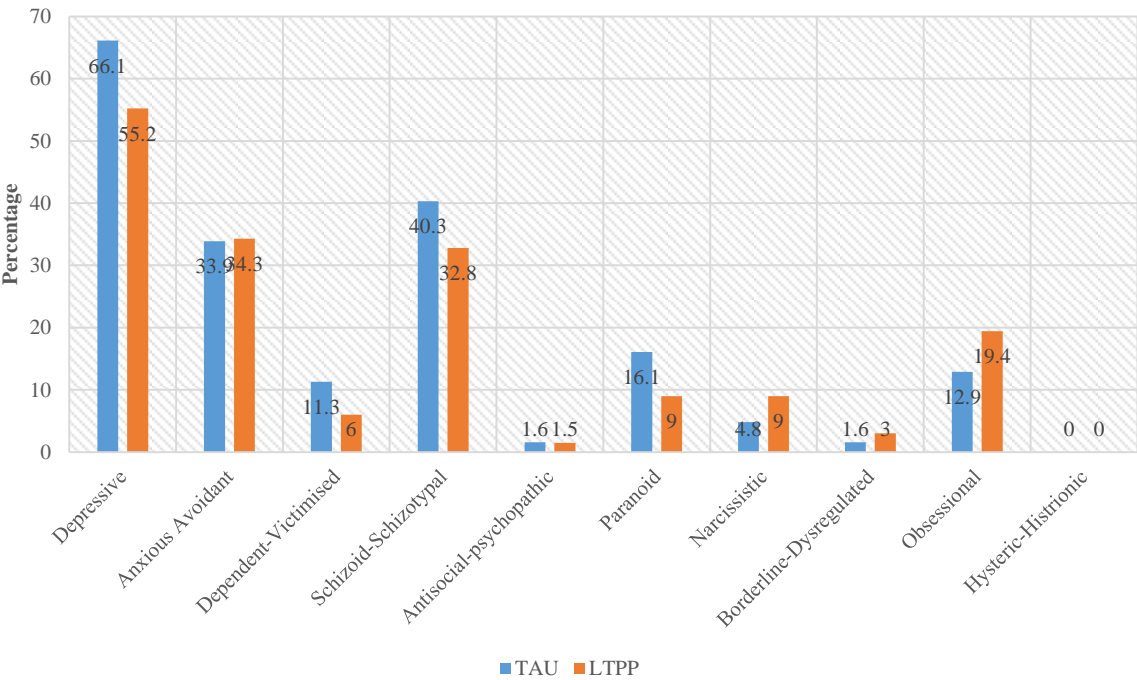


An independent t-test was carried out, in order to check for significant differences between the two groups for each of the SWAP-II personality disorder scales, at baseline, as well as for the “personality health” scale. No such differences were identified through the analysis (Appendix P).

Graph 2 displays the personality disorders prevalence for the TAU and LTPP groups at 18 months (N=55 for both groups). No significant between-group differences were identified at this time point, either (Appendix Q).

Graph 2

*SWAP-II Personality Disorders Prevalence at the End of Treatment (18 months)*

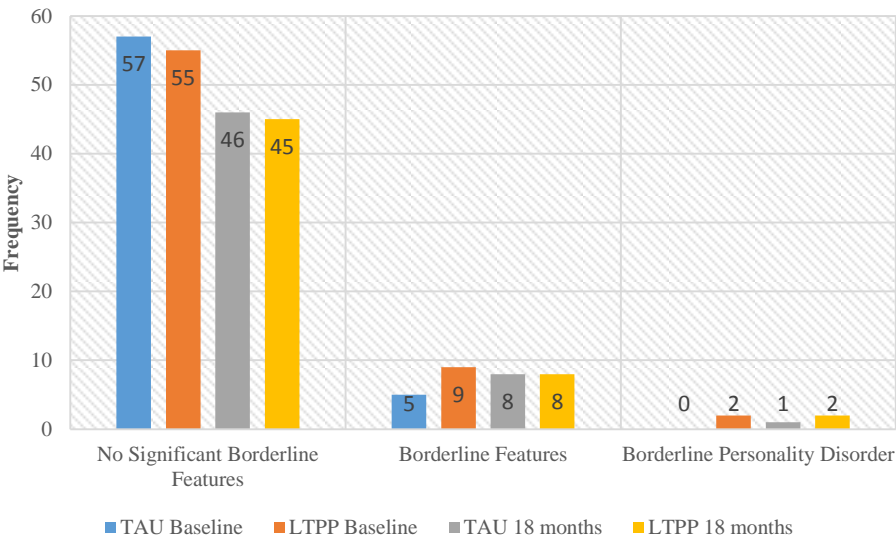


3.3.1. Borderline-dysregulated prototype

Graph 3 below presents the categorical distribution for the *borderline-dysregulated* personality prototype for each time-point and allocation group.

Graph 3

SWAP-II Borderline-dysregulated Prototype Frequency at Baseline and End of Treatment



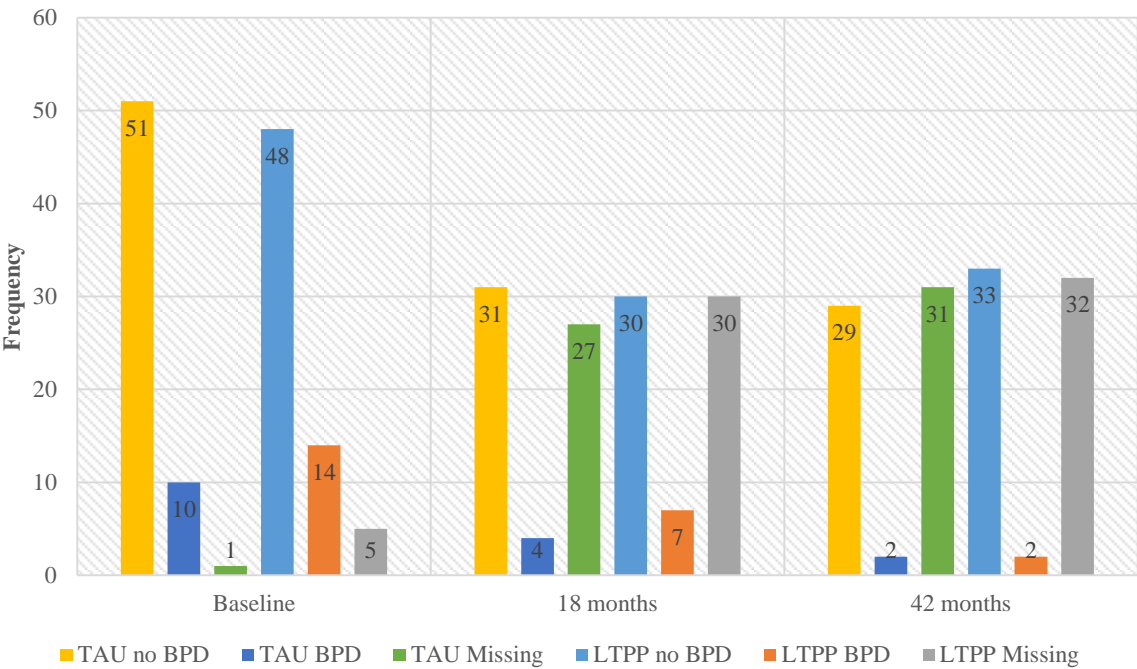
Of note, although there is no significant difference between the TAU and LTPP group in their *borderline-dysregulated* mean values, the graph above shows that only five people in the TAU group met criteria for *significant features*, and none for *borderline-dysregulated personality disorder*, compared to nine people meeting criteria for features and two for personality disorder in the LTPP group at baseline. The categorical distribution was more even between the two groups at the end of the treatment period, as shown in the graph.

Graph 4 presents the borderline personality disorder prevalence, as measured by the SCID-II-PQ. A cut-off score of 5 on the BPD sub-scale was used, in order to establish which patients met SCID-II-PQ criteria for the disorder (van Alebeek, van der Heijden, Hessels, Thong, & van Aken, 2015). This measure was considered to have more limitations than the SWAP-II, as discussed previously. The graph below suggests a much higher BPD prevalence in both groups

at baseline and end of treatment, than measured by the SWAP-II (10 patients in the TAU group and 14 – in LTPP). The SCID-II-PQ also had much higher missing values than the SWAP at the end of treatment (27 in TAU and 30 in LTPP, compared to 7 in TAU and 12 in LTPP for the SWAP-II).

Graph 4

*SCID-II-PQ Borderline Personality Disorder Frequency at Baseline, End of Treatment, and End of Follow-up*

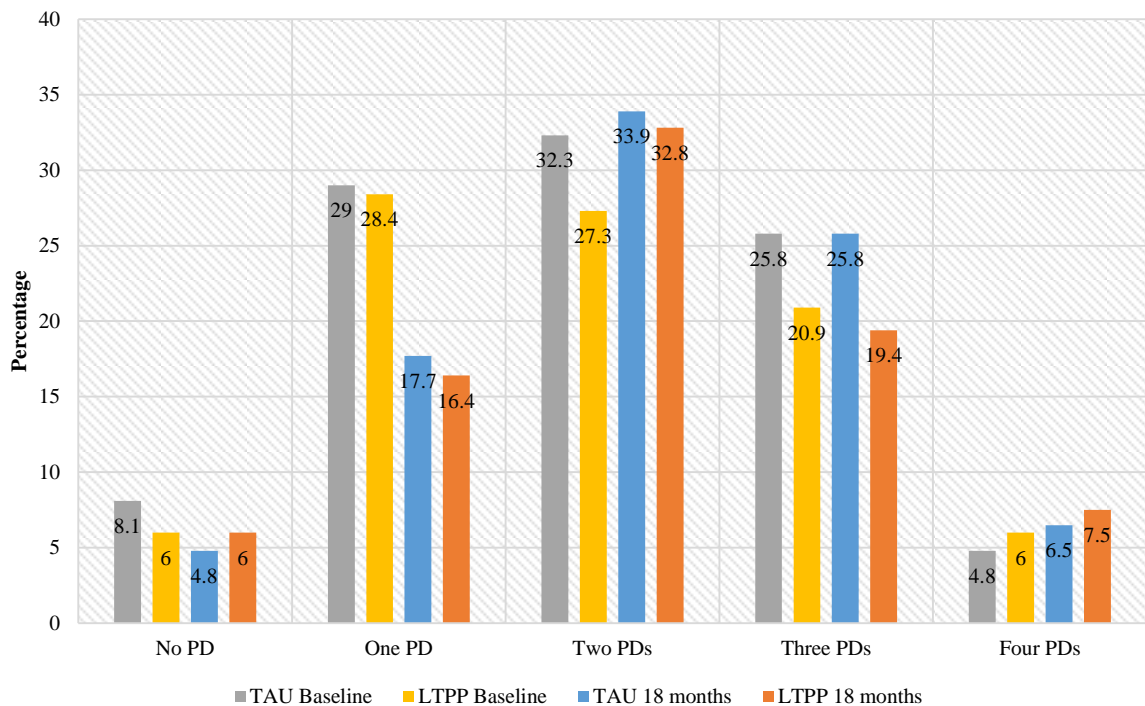


**3.3.2. SWAP-II personality disorders comorbidity**

The TADS patient group was characterised not only by high prevalence of personality disorder, as measured by the SWAP-II, but also by significant PD comorbidity. Graph 5 displays the prevalence of multiple PDs, when the SWAP-II was used.



Graph 5

*SWAP-II Multiple Personality Disorders Prevalence*

The average number of personality disorders, as measured by the SWAP-II and SCID-II-PQ, was also calculated and is presented in Table 7, together with the total number of cases, and the number of missing values. The SCID-II-PQ cut-off scores for each disorder varied, ranging from 3 to 6 (First et al., 1997).

Table 7

*Average Number of Personality Disorders, as Measured by the SWAP-II and SCID-II-PQ*

Allocation group			SWAP-II Baseline	SWAP-II End of treatment	SCID-II-PQ Baseline	SCID-II-PQ End of treatment	SCID-II-PQ End of follow-up
TAU	Mean		1.9	2.13	4.21	3.38	3.52
	N	Valid	62	55	61	34	31
		Missing	0	7	1	28	31
LTPP	Mean		1.92	2.07	4.56	3.61	2.89
	N	Valid	66	55	62	36	35
		Missing	1	12	5	31	32

An independent t-test did not identify any significant differences between the two groups (at any of the times points, and for neither of the two personality measures). A paired sample t-test did not find any significant differences in the number of SWAP-II PDs at the end of treatment, compared to baseline, in either group.

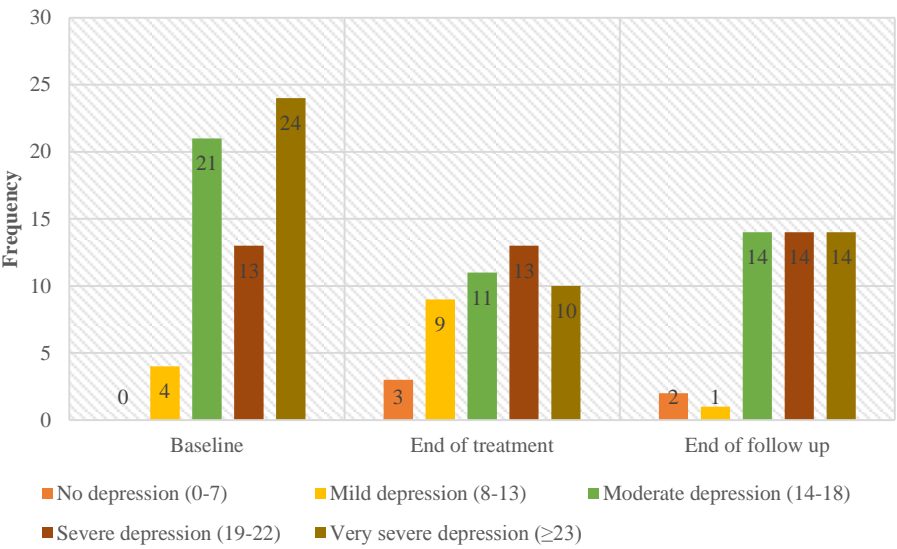
3.4. Borderline-dysregulated Personality as a Categorical Variable

3.4.1. Cross-tabulation analysis

The categorical distribution for the borderline-dysregulated syndrome was presented at the beginning of this chapter. Graphs 6 and 7 display the HDRS-17’s categorical distribution at baseline, end of treatment and follow-up, for each of the treatment groups.

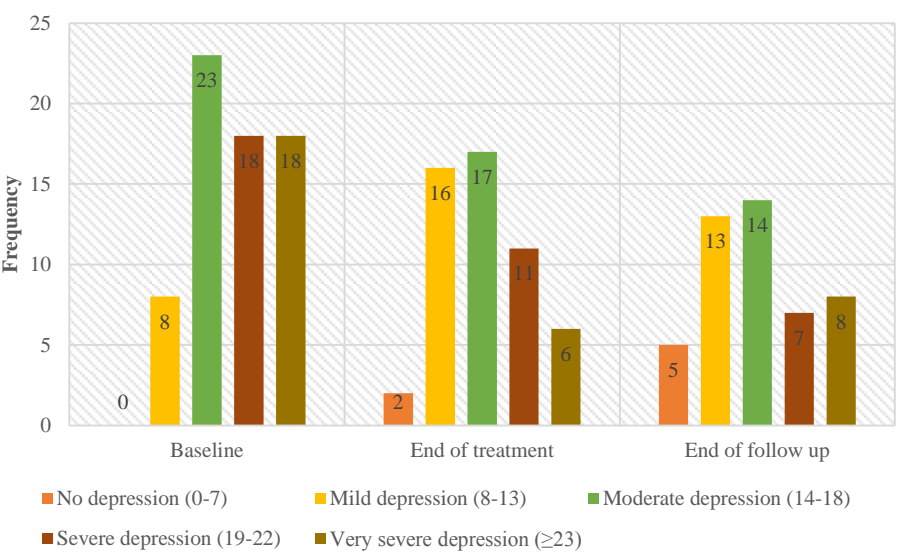
Graph 6

*HDRS Categorical Distribution in the TAU Group*



Graph 7

*HDRS Categorical Distribution in the LTPP Group*



The author considered running cross-tabulation analysis in SPSS in order to examine the relationship between borderline-dysregulated personality, as a categorical variable, and treatment outcome, as measured by the HDRS-17. One of the assumptions that needs to be met, in order to carry out a cross-tabulation analysis, is that a minimum of 5 cases are present in each cross-tabulation cell. Categorical distribution of the borderline-dysregulated score at baseline and 18 months, and that of the HDRS-17 at baseline, 18- and 42-monhts, shows a presence of only a few cases in some of the categories. The author therefore considered collapsing some of the categories – for example, “no significant features” ( $T < 55$ ) and “significant features or disorder” ( $T \geq 55$ ). For the HDRS-17 variable, a consideration was given to collapsing the categories of “no depression”, “mild” and “moderate” depression, and “severe or very severe depression”. There is, however, a significant difference between the experiences of a person who reports no symptoms of depression and those of an individual reporting moderate depression. Collapsing these categories and entering them in cross-tabulational analysis was therefore considered inappropriate.

### 3.4.2. Patients with borderline-dysregulated features or disorder

As identified at the beginning of this chapter (Graph 3), few participants scored high enough on the SWAP-II borderline-dysregulated prototype scale, to be considered as having significant borderline *features* or borderline personality *disorder* at baseline (TAU N = 5, LTPP N = 11).

A one-way repeated measures ANOVA with Bonferoni post-hoc tests was conducted to determine whether there was a statistically significant difference in HDRS-17, CORE-OM and GAF scores in the course of treatment and follow-up for each group. There was an increase of the GAF scores in the LTPP group from  $43.33 \pm 7.737$  at baseline to  $55.00 \pm 8.672$  at the end of treatment. This increase of  $11.667 \pm 3.201$  [mean  $\pm$  standard error] points was statistically significant ( $p < .05$ ). No other statistically significant changes in the three outcome measures' scores were identified.

This suggests that despite their lack of significant improvement of depression and psychological distress, patients who scored as having borderline features or disorder at baseline in the LTPP group did present with better general functioning after 18 months of therapy. The reader is reminded, however, that the GAF is a clinician-rated measure. The implication of this, together with some further limitations of the GAF, will be discussed in the final chapter of this thesis.

The same set of analyses was carried out for those who met borderline features or disorder criteria at 18 months (TAU N = 9, LTPP N = 10). No significant changes in the HDRS-17, CORE-OM and GAF scores were identified in either group, which suggests that these patients did not benefit from the treatment, nor did they deteriorate, based on their scores on the three outcome measures. A paired-sample T-test identified, however, a significantly higher SWAP-II borderline-dysregulated score at the end of treatment ( $56.67 \pm 1.414$ ), compared to baseline ( $51 \pm 5.326$ ),  $t(8) = -3.604$ ,  $p < .05$ , in the TAU group.

These results should be considered with caution due to the low number of patients who met borderline-dysregulated features/disorder criteria, making the comparison of mean values less reliable in detecting trends and changes.

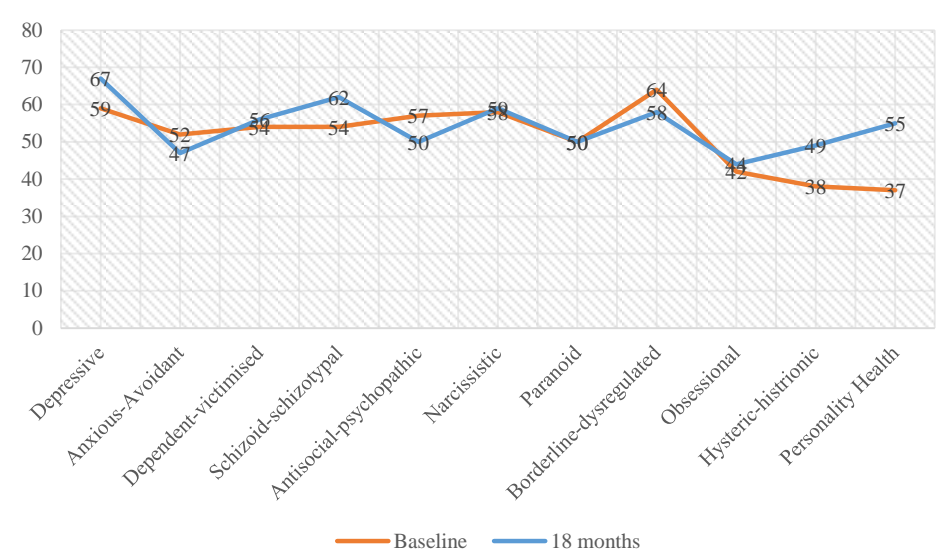
Looking at individual scores and change patterns might therefore be more meaningful, despite having very limited value in drawing generalisable conclusions. Appendices Qa and Qb present a summary of this group of patients' borderline-dysregulated scores at baseline and at the end of treatment, including the calculated borderline and depression change scores. One trend that can be observed when the individual cases are studied, is that in both groups some patients change categorically from having borderline "features" at baseline to "no features" at the end of treatment, whilst others moved from "no features" to "features". In many of the cases the change score was  $> 5$  points (half a Standard Deviation). Likewise, some patients in both groups experienced improvement in their depression, whilst others remained as depressed or deteriorated. As reported above, however, the mean pre- and post-treatment/post-follow up values were not significantly different, which suggests that any further conclusions about trends, based on individual case analyses, should be made with caution.

#### ***3.4.2.1. Borderline patients' full personality profile***

A previous section in this chapter presented the personality disorders prevalence and rates of comorbidity in the TADS sample. Like the rest of the TADS patients, those who scored as having features or disorder on the borderline-dysregulated scale, also scored above the SWAP-II 55-point cut-off on at least one more of the prototype scales. Graph 8 presents the full pre-and post-treatment personality profile of one of the three LTPP patients, who met criteria for borderline-dysregulated disorder. The full personality profiles of the other two LTPP patients, as well as of the TAU patient who met criteria for BPD at 18 months, can be found in Appendix R.

Graph 8

Patient 114 SWAP-II Personality Profile



Whilst no generalisable conclusions can be drawn from this data, it illustrates the complexity of these patients' presentation and alerts against thinking about each person only in terms of their borderline-dysregulated presentation. It is also interesting that all four patients scored significantly higher on the SWAP-II personality health scale at the end of treatment, despite their personality difficulties persisting, as captured by the other subscales, at that time point.

It is beyond the scope of this thesis to include and comment on the full personality profiles of the patients who presented with borderline-dysregulated features. A similar complexity of personality presentation, as captured by the SWAP-II scores, was observed there, too.

### 3.5. Borderline-dysregulated Personality as a Continuous Variable

#### 3.5.1. Correlational analysis

In order to further investigate the relationship between the borderline-dysregulated personality syndrome and treatment outcome for depression in the TADS sample, a change score was calculated for the three main outcome variables: HDRS-17, GAF and CORE-OM. The HDRS-17 and the CORE-OM change scores were computed by subtracting each measure's score at 18- and 42 months, from its baseline score. Thus, the higher the change score, the lower the severity of

symptoms at the end of treatment and follow-up. Conversely, negative change score values indicate deterioration in the patient's presentation at the end of treatment and 2-year follow-up. For the GAF change score, the baseline score was subtracted from the 18- and 42-month scores, as an increase in score indicates improvement in general functioning. Like with the HDRS-17 and CORE-OM change scores, a positive GAF change score thus denotes improvement, and a negative one – a deterioration in functioning.

A number of variables were included in the correlational analyses, as follows:

- Outcome measures change scores (HDRS-17, GAF and CORE-OM)
- Borderline-dysregulated disorder scores, as measured by the SWAP-II
- Participants' age at baseline
- Number of attended therapy sessions (for the LTPP group)
- Number of Axis I disorders at baseline, end of treatment, and follow-up

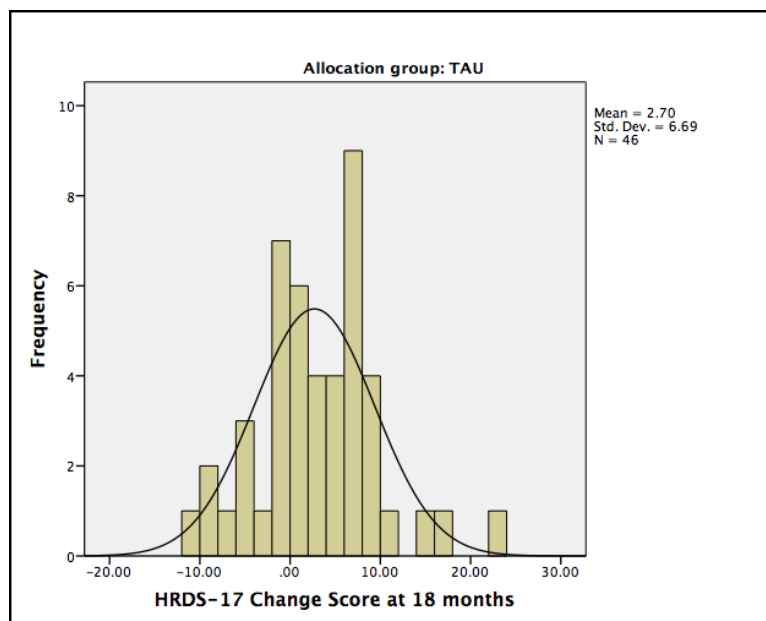
Whilst this thesis is primarily concerned with the relationship between the borderline-dysregulated personality syndrome, as measured by the SWAP-II, and depression, as measured by the HDRS-17, the author decided to test whether any of the other above-listed variables have a significant relationship with the change scores of the three outcome measures. This decision was made because, as discussed in the introduction chapter of this thesis, many authors have reported personality disorders to predict poor treatment outcome (e.g. Petersen et al., 2002). The same has been argued to be true for multiple Axis-I diagnoses, reflecting clinical complexity and consequently – poorer prognosis when only one condition, such as depression, is targeted during treatment (e.g. Zimmerman et al., 2014). Finally, the number of attended sessions has also been found by some authors to moderate treatment outcome (e.g. Hundt et al., 2014; Roseborough, 2005).

The statistical exploration of the above was undertaken, in order to establish whether any other variables, besides the borderline-dysregulated personality syndrome, might interact with treatment outcome, and thus might need to be controlled for in the next stages of the data analysis.

All tests of correlation were run using the *Pearson* correlation statistic, after assumptions of normal distribution were tested and established for all variables included in the analyses. Graph 9 below gives an example of the HDRS-17 histogram (testing normal distribution) at 18 months. The same analysis of normality was run for the remaining variables too.

Graph 9

HDRS-17 Histogram at 18 months



Two - tailed tests were deemed appropriate, as the author could not make an advance prediction about the direction of correlations.

### ***3.5.1.1. Borderline-dysregulated Q-sort correlation with the number of Axis I disorder, and the outcome measures***

The SWAP-II borderline-dysregulated Q-factor baseline score, the number of Axis I disorders at baseline, 18- and 42-months, and the three outcome measures' change scores at 18- and 42-



months were all correlated. The results are presented in Table 8 overleaf. As can be seen, the borderline-dysregulated prototype baseline score did not correlate significantly with any of the other variables in the TAU group. There was, however, a significant relationship between this personality prototype score and the number of Axis I disorders at 18- and 42-months in the LTPP group ( $r = .504, p < .01$ , and  $r = .347, p < .05$ , respectively), which suggests that the more patients resembled a borderline personality disorder presentation at baseline, the more Axis I disorders they had at the end of treatment and at a 2-year follow-up.

The number of Axis I diagnoses at baseline was not significantly related to any of the outcome variables' change scores. Higher number of Axis I disorders at the end of treatment was only linked to less improvement in general functioning at 42 months in the LTPP group ( $r = -.312, p < .05$ ). Changes in psychological distress (as measured by the CORE-OM) were not significantly linked to the number of Axis I disorders or to the SWAP-II borderline-dysregulated prototype score.

In both the TAU and LTPP groups, a higher number of Axis I diagnoses at 42-months were related to less improvement in depression at follow-up (but not the end-of-treatment) ( $r = -.426, p < .01$ , and  $r = -.491, p < .01$ , respectively). More Axis I diagnoses at 42 months was also linked to less improvement in general functioning (as measured by the GAF) at the end of treatment and follow-up in the LTPP group ( $r = -.368, p < .01$ , and  $r = -.494, p < .01$ , respectively).

Table 8 *Correlation Coefficients between the SWAP-II Borderline-dysregulated Q-factor, Number of Axis-I Disorders, HDRS-17, GAF and CORE-OM*

Allocation group		Borderline-Dysregulated	Number of Axis I diagnoses at Baseline	Number of Axis I diagnoses 18m	Number of Axis I diagnoses 42m	HDRS Change Score 18m	HDRS Change Score 42m	GAF Change Score 18m	GAF Change Score 42m	CORE Change Score 18m	CORE Change Score 42m
TAU Group	Borderline-Dysregulated N	1 62	-0.036 62	0 46	0 46	-0.012 46	0.007 45	0.189 44	0.21 44	0.005 35	-0.048 36
	Number of Axis I diagnoses Baseline N	-0.036 62	1 62	.675** 46	.630** 46	-0.081 46	-0.022 45	0.147 44	0.121 44	0.018 35	-0.037 36
	Number of Axis I diagnoses 18m N	0 46	.675** 46	1 46	.679** 39	-.309* 44	-0.232 38	-0.074 43	0.111 37	-0.035 33	-0.146 30
	Number of Axis I diagnoses 42m N	0 46	.630** 46	.679** 39	1 46	-0.241 38	-.426** 43	0.171 37	-0.228 42	-0.188 28	-0.322 33
LTPP Group	Borderline-Dysregulated N	1 66	0.175 66	.504** 50	.347* 46	-0.056 51	0.054 46	-0.079 51	0.135 46	0.086 42	0.084 42
	Number of Axis I diagnoses at Baseline N	0.175 66	1 67	.680** 51	.539** 46	-0.047 52	-0.192 47	-0.254 52	-0.11 47	0.221 43	0.141 42
	Number of Axis I diagnoses 18m N	.504** 50	.680** 51	1 51	.742** 44	-0.265 51	-0.249 44	-.312* 51	-0.119 45	-0.035 42	-0.091 40
	Number of Axis I diagnoses 42m N	.347* 46	.539** 46	.742** 44	1 46	-0.177 44	-.491** 45	-.368* 44	-.494** 46	-0.033 37	-0.284 42

\*\* Correlation is significant at the 0.01 level (2-tailed); \* Correlation is significant at the 0.05 level (2-tailed).

### 3.5.2. The Borderline-dysregulated change score

Finally, a borderline-dysregulated change score was calculated, too, by subtracting the T-score at 18-months from the baseline score. A positive change score value thus denotes a decrease on the borderline-dysregulated prototype sub-scale, whilst a negative score denotes a score increase (i.e. the person resembling *more* the borderline-dysregulated pure prototype).

The rationale for calculating the change score was that, as presented in the introduction chapter of this thesis, most authors agree that it is difficult to establish a causal link between personality disorders and depression, and some have argued that prolonged experiences of depression may lead to personality changes, in turn. The author therefore decided to investigate whether changes in the borderline-dysregulated prototype score were related to changes in depression, general functioning and psychological distress, as well as to the number of Axis I disorders, at the three different time-points. These results are presented in Table 9 overleaf.

With regards to the three outcome measures, in the TAU group a decrease of the borderline-dysregulated score was related to a decrease in depression at 18-months ( $r = .324, p < .05$ ), to increase in general functioning at 18- and 42-months ( $r = .438, p < .01$ ; and  $r = .372, p < .05$ ), and to decrease in psychological distress at 18 months ( $r = .548, p < .01$ ).

In the LTPP group a lower borderline-dysregulated score was significantly related only to decrease in depression at 42-months ( $r = .319, p < .05$ ).

In neither of the groups were changes in the borderline-dysregulated score significantly associated with the number of Axis I disorders at baseline, end of treatment or end of follow-up. A decrease in the borderline-dysregulated score was, however, associated with an increase in the personality health Q-factor score, in both groups.

Table 9

*Correlation Coefficients between the SWAP-II Borderline-dysregulated and Personality Health Change Scores, and Number of Axis I Disorders, HDRS-17, GAF and CORE-OM*

Allocation group		Borderline Dysregulated Change Score	Personality Health Change Score 18m	HDRS-17 Change Score at 18 months	HDRS-17 Change Score at 42 months	GAF Change Score at 18 months	GAF Change Score at 42 months	CORE-OM Change Score at 18 months	CORE-OM Change Score at 42 months	Number of Axis I diagnoses at Baseline	Number of Axis I diagnoses at 18 months	Number of Axis I diagnoses at 42 months
<b>TAU</b>	Borderline Dysregulated Change Score	1	.500**	.324*	.275	.438**	.372*	.548**	.193	-.053	.011	-.065
	N	55	55	46	45	44	44	34	35	55	45	45
	Personality Health Change Score	.500**	1	.108	.163	.153	.149	.260	.010	-.128	.166	-.050
	N	55	55	46	45	44	44	34	35	55	45	45
<b>LTPP</b>	Borderline Dysregulated Change Score	1	.454**	.224	.319*	.131	.239	.306	.287	-.113	-.121	-.145
	N	54	54	50	46	50	46	41	42	54	50	46
	Personality Health Change Score	.454**	1	.200	.398**	.319*	.415**	.306	.461**	.002	-.069	-.303*
	N	54	54	50	46	50	46	41	42	54	50	46

\*\*, Correlation is significant at the 0.01 level (2-tailed), \* Correlation is significant at the 0.05 level (2-tailed).

### 3.5.3. The personality health change score

As noted above, increased SWAP-II personality health scores were linked to decreased borderline-dysregulated score in both groups ( $r = .500, p < .01$ , and  $r = .454, p < .01$ , for the TAU and LTPP groups respectively).

In addition, in the LTPP group a higher personality health score at the end of treatment was also related to a decrease in depressive symptoms at 42-months (HDRS-17) ( $r = .398, p < .01$ ), increase in general functioning (GAF) and 18- and 42-months ( $r = .319, p < .05$ , and  $r = .415, p < .01$ , respectively), and decrease in psychological distress (CORE-OM) at 42 months ( $r = .461, p < .01$ ). The personality health change score was negatively related to the number of Axis I disorders at 42 months, too ( $r = -.303, p < .05$ ), indicating that the bigger the increase in personality health, the fewer the Axis I diagnoses at follow-up.

In the TAU group the personality health change score did not correlate significantly with any of the outcome measures, or with the number of Axis I disorders at the three time points.

### 3.5.4. The rest of the SWAP-II Q-factors change scores

The SWAP-II was developed as an overall measure of personality, and individuals receive a score on all prototype subscales. These scores are subsequently interpreted in relation to one another, in order to make clinical sense of the person's overall presentation. It would therefore be artificial and clinically meaningless to explore only the borderline-dysregulated prototype in isolation, without considering how it relates to the other personality prototypes.

Furthermore, it is in the nature of correlational analysis that it tells us little about causation or the impact that other variables might have on the outcome measures of interest. More specifically, depression might be linked to other personality constellations, which might or might not in turn also be related to the borderline-dysregulated prototype.

In order to investigate this hypothesis, the author carried out a correlational analysis of the rest of the Q-factor prototypes' change scores and the three outcome measures. The correlation statistics are presented in Appendix S.

There were a number of significant correlations between different SWAP-II prototype change scores. For the purposes of the current project, it would suffice to focus on those personality prototype change scores that had a statistically significant relationship to the borderline-dysregulated score and to the three outcome measures' change scores. This is summarised in Table 10.

Table 10

*Correlation Coefficients Between the SWAP-II Personality Prototypes' Change Scores, and the HDRS-17, GAF and CORE-OM*

Allocation group		Borderline Dysregulated Change Score	Personality Health Change Score	HDRS-17 Change Score 18m	HDRS-17 Change Score 42m	GAF Change Score 18m	GAF Change Score 42m	CORE-OM Change Score 18m	CORE-OM Change Score 42m
<b>TAU</b>	Dependent-Victimised Change Score	-0.218	-0.055	0.036	-0.11	-0.162	-.372*	-0.037	-0.08
	N	55	55	46	45	44	44	34	35
	Paranoid Change Score	.455**	.349**	0.142	0.141	0.083	0.083	0.214	0.112
	N	55	55	46	45	44	44	34	35
	Obsessional Change Score	-.378**	-0.077	-0.24	-0.258	-0.19	-.369*	-0.078	0.128
	N	55	55	46	45	44	44	34	35
<b>LTPP</b>	Depressive Change Score	0.058	0.245	0.119	.357*	0.164	.499**	.311*	0.227
	N	54	54	50	46	50	46	41	42
	Anxious Avoidant Change Score	0.041	.471**	0.228	.436**	.404**	.568**	0.261	.351*
	N	54	54	50	46	50	46	41	42
	Dependent-Victimised Change Score	.303*	0.141	0.07	0.255	-0.229	0.063	0.101	0.039
	N	54	54	50	46	50	46	41	42
	Paranoid Change Score	.439**	.458**	0.23	0.115	0.115	-0.019	0.21	0.198
	N	54	54	50	46	50	46	41	42
	Hysteric-Histrionic Change Score	0.247	-0.02	-0.013	-0.099	-0.275	-.344*	0.02	-0.085
	N	54	54	50	46	50	46	41	42

\*\* . Correlation is significant at the 0.01 level (2-tailed), \* Correlation is significant at the 0.05 level (2-tailed).

As displayed in table 10, in the TAU group none of the SWAP-II personality prototype change scores were significantly related to changes in depression, except for the borderline-dysregulated change score (Table 8). Interestingly, patients who scored higher on the dependent-victimised and obsessional scales at the end of treatment, compared to their baseline scores, also tended to have better general functioning at 42 months (as measured by the GAF). An increase in the obsessional personality score was also linked to a decrease in the borderline-dysregulated scores at the end of treatment, whereas a decrease in patients' paranoid score was associated with less resemblance with the borderline prototype.

In the LTPP group lower depressive and anxious-avoidant personality scores were linked to a decrease in depression severity and increased general functioning at 42 months. The reader is reminded that the borderline-dysregulated change score was significantly related to the HDRS change score at 42 months, as well. Decreases in anxious-avoidant personality scores were significantly linked to better general functioning at the end of treatment and to less psychological distress at the end of follow-up, too. The dependent-victimised and paranoid change scores were not related to any of the outcome measures, but a decrease on both scales was associated with a decrease on the borderline-dysregulated scale. Finally, patients who presented as more hysteric-histrionic at the end of treatment also seemed to function better at the end of the follow-up period. Both the hysteric-histrionic and the dependent-victimised mean scores at 18 months were significantly lower than those at baseline, as reported earlier in this chapter.

An increase in personality health at the end of treatment, as measured by the SWAP-II, was associated with a decrease in paranoid features in both groups, and with lower anxious-avoidant personality scores in the LTPP group.

### **3.5.5. Borderline-dysregulated prototype item analysis**

The results presented in the previous section indicate a significant association between the borderline-dysregulated change score and a number of the outcome variables in both groups. In



addition, the changes in patients' degree of resemblance to the borderline-dysregulated personality disorder appeared to be linked to other personality changes, including to the patients' "personality health".

These results are interesting, given the small number of patients who met the criteria for borderline features or disorder. The author therefore decided to analyse the correlations between the 12 borderline-dysregulated SWAP-II items which had fair or excellent inter-rater agreement (as reported at the beginning of this chapter), and the three outcome measures' change scores. This was calculated both for the borderline items' scores at baseline, as well as for the borderline items' change scores.

Tables 11a and 11b present the statistically significant correlations (the rest of the statistics can be found in Appendices Ta and Tb).

The results presented in Table 11a show that only a few of the SWAP-II borderline-dysregulated items correlated with the three outcome measures. In the TAU group those were three items representing insecure attachment, chaotic life-style and self-harm. More specifically, higher scores on the item indicating "unstable, chaotic, and rapidly changing" relationships at baseline were associated with less improvement in depression and general functioning at the end of treatment. Patients who presented as having urges to kill themselves were also less likely to experience a decrease in psychological distress by the end of follow-up. Interestingly, feeling more fearful of rejection or abandonment at baseline was associated with a decrease in psychological distress at 42 months, as measured by the CORE-OM.

In the LTPP group higher scores on the item reflecting tendency to stir up conflict or animosity in others were linked to patients reporting less psychological distress at the end of follow-up.

A number of the borderline-dysregulated items' change scores were associated with treatment outcome at 18- and 42-months in both groups (Table 11b). In the TAU group, a decrease in

affect dysregulation and insecure attachment and an increase in chaotic relationships were all associated with improvement in depression at 42 months. Decreased scores on two further items indicating affect dysregulation were associated with better general functioning at 42 months.

In the LTPP group, a decrease in depression at the end of follow-up was only linked to a decrease in affect dysregulation (the person's tendency to become less rational when experiencing strong emotions). A decrease in insecure attachment (feeling misunderstood and victimized) was associated with improved general functioning at the end of treatment, and decrease in splitting (stirring up conflict and animosity between others) was related to less psychological distress at the end of follow-up.

Table 11a

*Borderline-dysregulated Baseline Items Correlations with the HDRS-17, GAF and CORE-OM*

Allocation group	SWAP-II Item	Item type	HDRS-17 Change Score at 18 months	HDRS-17 Change Score at 42 months	GAF Change Score at 18 months	GAF Change Score at 42 months	CORE Change Score at 18 months	CORE Change Score at 42 months
TAU	Tends to fear she/he will be rejected or abandoned	Insecure attachment	0.219	0.005	0.098	-0.09	.385*	.382*
	N		46	45	44	44	35	36
	Relationships tend to be unstable, chaotic, and rapidly changing	Chaotic lifestyle	-.313*	-0.202	-.344*	-0.02	0.068	0.065
	N		46	45	44	44	35	36
	Struggles with genuine wishes to kill him/herself	Self-harm	-0.075	-0.099	-0.158	-0.091	-0.219	-.345*
	N		46	45	44	44	35	36
LTPP	Tends to stir up conflict or animosity between other people	Splitting	0.153	0.222	0.042	0.214	.363*	.418**
	N		51	46	51	46	42	42

\*\*. Correlation is significant at the 0.01 level (2-tailed), \* Correlation is significant at the 0.05 level (2-tailed).

Table 11b

*Borderline-dysregulated Items' Change Scores Correlations with the HDRS-17, GAF and CORE-OM*

Allocation group	SWAP-II Item	Item type	HDRS-17 Change Score at 18 months	HDRS-17 Change Score at 42 months	GAF Change Score at 18 months	GAF Change Score at 42 months	CORE Change Score at 18 months	CORE Change Score at 42 months
TAU	Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.	Affect dysregulation	0.091	.386*	0.186	0.302	0.182	0.157
	N		39	39	38	39	31	35
	Tends to feel misunderstood, mistreated or victimized	Insecure attachment	0.253	.385*	0.142	0.26	0.208	0.202
	N		39	39	38	39	31	35
	Relationships tend to be unstable, chaotic, and rapidly changing	Chaotic lifestyle	-0.307	-.480**	-0.176	-0.232	-0.091	-0.19
	N		39	39	38	39	31	35
	Is prone to intense anger, out of proportion with the situation at hand	Affect dysregulation	-0.02	0.147	0.093	.317*	0.063	0.181
	N		39	39	38	39	31	35
LTPP	Emotions tend to change rapidly and unpredictably	Affect dysregulation	-0.088	0.137	0.102	.445**	0.142	0.059
	N		36	36	35	36	29	32
	Tends to stir up conflict or animosity between other people	Splitting	0.096	0.276	0.018	0.158	0.163	.350*
	N		44	41	44	41	37	38
	Tends to feel misunderstood, mistreated or victimized	Insecure attachment	0.241	0.077	.358*	0.068	0.114	0.163
	N		44	41	44	41	37	38
	Tend to become irrational when strong emotions are stirred up	Affect dysregulation	0.11	.327*	-0.013	0.253	-0.028	0.144
	N		44	41	44	41	37	38

\*\*. Correlation is significant at the 0.01 level (2-tailed), \* Correlation is significant at the 0.05 level (2-tailed).

### **3.5.6. Regression Analysis**

A regression analysis was finally carried out, in order to test whether participants' SWAP-II borderline-dysregulated score at baseline predicts their HDRS-17, GAF and CORE-OM change scores at 18 and 42 months.

The full regression statistics are presented in Appendix U. None of the regression models reached a significance level of  $p < .05$ .

The regression analysis results also indicated that very little of the variation in any of the three outcome measures' change scores could be accounted for by the borderline-dysregulated SWAP-II score at baseline. Given that most of the participants had a borderline-dysregulated baseline score below 55, these regression results give little information about the impact that borderline-dysregulated features or disorder have on treatment outcome. This question was partially addressed earlier in this chapter, where the analysis results for those who met the SWAP-II criteria for borderline features or disorder were presented. The small number of those participants did not allow for further statistical analyses to be carried out, however.

The results presented in this chapter will be further interpreted in the final, discussion chapter of this thesis next.

#### 4. Discussion and Conclusion

The introduction chapter presented the concepts of treatment resistant depression and borderline personality disorder, as understood through the medical and psychoanalytic models.

In reviewing the literature to date, it became apparent that theoretical differences aside, most researchers and clinicians agree that personality plays at least some role in the course and treatment of depression. This is evidenced by the high rates of psychiatric comorbidity between depression and personality disorders, as well as by the number of research studies investigating the link between personality disorder and/or personality traits and depression. Borderline personality disorder, in particular, has been the subject of many such investigations. Yet, no study to date has addressed the link between borderline personality disorder, treatment resistant depression and treatment outcome, particularly in the course of psychodynamic psychotherapy.

Moreover, most studies of personality disorder and depression have used a categorical approach to personality, where the presence/absence of a disorder is considered. The role of personality disorder features, in the absence of a formal diagnosis has been so far generally neglected in the literature. Of note, what is meant by “features” in the current study is the degree of resemblance to borderline personality disorder in individuals, who do not present with the full features of the disorder and are therefore not diagnosed with BPD.

The current study has attempted to address this gap in the literature, which is of both theoretical and clinical relevance. The *borderline-dysregulated* personality prototype, as measured by the SWAP-II, was used in studying borderline personality both as a dimensional, as well as a categorical variable. Its relationship to the three TADS outcome measures – the HDRS-17, the CORE-OM and the GAF was explored in both the treatment as usual (TAU) and in the long-term psychotherapy (LTPP) group. A summary of these findings, together with their interpretation, is

presented in this final chapter. The chapter finishes with a discussion of the limitations of this study, as well as some suggestions for future research and clinical practice directions.

## 4.1. Interpretation of Results

### 4.1.1. Borderline-dysregulated personality

#### 4.1.1.1. *Borderline-dysregulated features and disorder*

The *borderline-dysregulated* score at baseline did not correlate significantly with any of the outcome measures in either group. This result should not be confused with borderline personality disorder not being related to or predictive of treatment outcome, however, as none of the TAU patients, and only two (3%) of the LTPP patients met criteria for BPD at baseline, as measured by the SWAP-II. Thus, the results only indicate that whether patients scored higher or lower on the borderline-dysregulated scale at baseline, did not have a significant link to their depression, general functioning and psychological distress at the end of treatment and follow-up.

A small number of patients in each group - 5 (8%) in TAU and 11 (16%) in LTPP - met SWAP-II criteria for having significant borderline-dysregulated *features* or *disorder* (although there were only two people who scored above cut-off for disorder and they were both in the LTPP group). These figures are somewhat in line with epidemiological statistics of 6% BPD prevalence in primary care settings, and 10% in outpatient clinical settings (APA, 2013).

A correlational analysis explored the link between the *borderline-dysregulated* prototype, as a continuous variable, and the number of Axis I disorders at baseline, end of treatment, and end of follow-up. Interestingly, the borderline score did not correlate significantly with Axis I disorders at baseline in either group, but higher scores on the prototype at baseline were significantly linked to a higher number of Axis I disorders at 18- and 42-months in the LTPP group. In this group the higher number of Axis I disorders at the end of treatment and follow-up was also

linked to poorer general functioning, and to less improvement in depression (at the end of follow-up only). The number of Axis I disorders at 18- and 42-months in the TAU group was related only to smaller changes in depression scores at these two time points, but not to the borderline prototype, or to general functioning.

The interpretation of these results is difficult, when based only on correlation statistics. One possible way of understanding the data is that in the LTPP group the more patients resembled the borderline-dysregulated prototype, the more likely they were to meet criteria for multiple Axis I disorders at the end of treatment and follow-up, which in turn is associated with poorer treatment outcome, as measured by the GAF (at 18 months) and the HDRS-17 and GAF (at 42 months). It is not possible to establish a causal relationship between any of the variables. It seems plausible to argue, however, that in the LTPP group higher borderline-dysregulated scores were linked to greater clinical complexity, expressed in the multiple Axis I diagnoses at the end of treatment. A patient who presents with multiple difficulties, is therefore less likely to benefit significantly from a single intervention (Levy, 2016). The same results were not observed in the TAU group, where a correlation statistic of  $r = 0$  indicated a non-linear relationship between the borderline score at baseline and the number of Axis I disorders at the end of treatment and follow-up.

Finally, the results chapter offered a closer look at the full personality profiles of those patients who scored above 55 on the borderline-dysregulated SWAP-II scale, and therefore presented with significant features or with borderline-dysregulated disorder. Shedler (2016) warns against interpreting T-scores too rigidly, however, as the SWAP-II cut-off scores are provided as a guideline, as well as to ensure some “backward compatibility” with previous classification tools, such as the DSM, which presents personality disorders in categorical terms. Thus, the selection of those who had a borderline-dysregulated score  $\geq 55$  might not be the most clinically meaningful way to look at a sub-sample of patients. Yet, it is one way of choosing some cases



where borderline personality appears to be partially expressed, in order to investigate the implications it has for the treatment of depression.

All patients who met criteria for borderline features or disorder either at baseline or at the end of treatment (or both) were presented, in terms of their borderline-dysregulated scores and outcome measures scores at the three time-points, together with the corresponding change scores. The borderline-dysregulated change score for most patients with borderline features/disorder was  $\geq 5$  points, and for four patients it was  $\geq 10$  points. The reader is reminded that the SWAP-II prototype scales are standardised, with 10 points representing one standard deviation. A change in score equal to or larger than 10 points is considered both statistically, as well as clinically significant. Shedler (2016) further argues that a change of .5 standard deviations has clinical significance too, comparing this to recent NICE guidelines on clinically significant change in the use of anti-depressants. It can therefore be concluded that, for the majority of patients with borderline features or disorder in both TAU and LTPP groups, the treatment appeared to exacerbate their personality difficulties, rather than to alleviate them. This conclusion was statistically supported for the TAU group, where the post-treatment borderline-dysregulated mean score was significantly higher than the baseline one.

Furthermore, these patients' improvement in depression, general functioning and psychological distress was also questionable. In the TAU group only three people had a positive HDRS change score (indicating decrease in depression) at 42 months, and none of the patients reached full or partial remission of depression ( $\text{HDRS} \leq 8$ , and  $\leq 12$ , respectively). In the LTPP group there was a significant improvement on the GAF measure at 18 months, but no significant improvement of depression or psychological distress. A further look into the individual data showed that in the LTPP group 7 people had a positive HDRS change score at 42 months, but only one of them reached full remission ( $\text{HDRS}=4$ ) and one – partial remission ( $\text{HDRS}=12$ ).

A categorical distribution of the patients in terms of their depression score revealed no shifts at the end of treatment and follow-up, in comparison to baseline, for those who presented with borderline features/disorder at the end of treatment. On the contrary, having borderline features or disorder in the LTPP group at 18 months appeared to make it more likely that the patient also scored as severely or very severely depressed on the HDRS.

These results are based on a very small sample, and should therefore be interpreted with caution. Yet, they pose some very important clinical and research questions. The first one is – can patients who present with borderline personality features or disorder, and depression, improve in terms of their depression? Previous RCTs, which looked at the effectiveness of treatments for BPD, suggest that they can. For example, five RCTs comparing the effectiveness of Mentalization Based Treatment (MBT) for BPD to other treatments, all reported a significant decrease in depressive symptoms, based on scores on the Beck Depression Inventory (Bateman & Fonagy, 2001; Bateman & Fonagy, 1999, 2009; Jørgensen et al., 2013) and the Mood and Feelings Questionnaire (Rossouw & Fonagy, 2012). In these studies a decrease in depression was also reported to be significantly greater for those in the MBT groups, compared to the other treatments. Another study, comparing Manualised Cognitive Behavioural Therapy to Treatment as Usual for BPD, also reported a significant decrease in depression, as measured by the HDRS, although the between-group differences were not statistically significant (Tyrer et al., 2004). It is thus important to consider why, in the TADS patient group, neither borderline features, nor depression appear to improve in the course of either of the treatments offered, for those who presented with borderline features/disorder.

A recent, yet unpublished meta-analysis on the treatment of BPD, presented in the 22<sup>nd</sup> Annual BACP Conference by Prof Kenneth Levy, might give some possible answers to this question (Levy, 2016). More specifically, Levy argued that borderline personality disorder is often not

recognised in outpatient clinical settings, and treatments are offered for other presenting difficulties, such as major depression. He considered this to have detrimental consequences for treatment outcome, as borderline personality disorder affects both the course and outcome of depression treatments, but not the other way around (an argument substantiated by the BPD RCT results reported above). He therefore warned against “privileging” the interventions for depression and linked this to research indicating that having even one BPD symptom increases the likelihood of suicide in patients being treated for mood disorders (e.g. Zimmerman et al., 2014). Levy therefore stressed the importance of assessing patients who present with difficulties such as depression and anxiety, for underlying borderline personality symptoms, and stated that for many patients, borderline personality features remain undiagnosed for an average of 6-10 years, which leads to under-treatment or mistreatment. He then presented the results of a meta-analysis (in press), which included 73 studies on the treatment of BPD, and identified a number of common treatment factors, all believed to contribute to treatment effect sizes. These included the structured nature of BPD treatments, and the more active, engaged stance of the therapist, where particular attention is being paid to the therapeutic frame and relationship. In addition, therapists’ supervision was also reported to be an important therapeutic factor.

This gives rise to the second question, namely: if depression can be treated in the context of borderline personality disorder (or features) what adjustments need to be made to treatment and how relevant is this to the outcomes of the TADS study?

The first chapter of this thesis introduced some of the existing theories on borderline personality disorder. Relevant to this part of this discussion appears to be what mentalization theories suggest to be a main difficulty for patients diagnosed with BPD – namely to conceive, process and think about their own affective and mental states, as well as those of other people (Bateman & Fonagy, 2016). Target (2016) states:

“In infancy and psychotherapy, before affects can be symbolized and regulated, they need to be located in the inner world. Gradually the action they demand becomes metaphorical – lived out only in the embodied mental activity of unconscious phantasy.” (p.208)

Fonagy & Luyten (2016) provide further rationale for adapting interventions for patients who present with borderline personality features. More specifically the authors point at the anxious-preoccupied attachment strategies, characteristic of these patients. They put forward the argument that, in order to survive in context of early life adversity and trauma, individuals with BPD have lower threshold for attachment system activation – they are more sensitive to interpersonal stress, which in turn activates the amygdala system in the brain and leads to proximity seeking; this is, at the same time, associated with a deactivation of the “neural systems associated with controlled social cognition, including systems involved in judging the trustworthiness of others” (p.747), and leads to a “vicious interpersonal cycle” marked by high levels of both anxiety and avoidance. Fonagy & Luyten (ibid) further emphasise the adaptive function of anxious-preoccupied strategies in early life for individuals who were deprived of a stable and supportive environment, where emotional states have been recognised and thought about by the care-giver, fostering the development of mentalizing capacities and the ability to self-soothe and self-regulate. These strategies become, however, maladaptive, in different social context and further feed into chronic feelings of hopelessness and helplessness, which are expressed through negative affect, such as depression.

The process of the therapist recognising affective states (including feelings of depression) and reflecting them back to the patient, was compared by Target (2016) to the process of “marked mirroring”, which leads to the individual beginning to first own, and then make sense of their emotional experiences. Only when this capacity is developed enough, she argued, can the patient

hear, understand and make use of the therapist's other interventions, such as interpretations in a psychoanalytic treatment, for example.

Bateman & Fonagy (2016) present further ideas about treatment modifications required for patients who presenting with both depression and features of BPD. They argue that treatments for depression are bound to be unsuccessful, unless BPD is taken into account. This is due to limitations in these patients' capacity for reflective functioning – namely, when strong emotions are experienced, the individual manages them through action (e.g. self-harming behaviours) rather than through *thinking* about their mental representations of self- and others. High levels of arousal and anxiety are further thought to be triggered by long silences in therapy, or focus on interpretations of the patient-therapist relationship; this is considered to be unhelpful, and on occasions even harmful to the patient. Bateman & Fonagy (ibid) therefore suggest that treatments for BPD and depression adopt an active, validating and supportive stance, where the initial focus is on recovering the capacity to mentalize, as suggested by Taget (2016), too.

Implicit marked mirroring in both the TAU and LTPP might therefore be considered insufficient for those patients who present with borderline features or disorder. The lack of improvement in these patients' personality presentation and depression suggests that more active adaptation of their treatment, and a more explicit consideration of their personality profiles might need to take place, if treatment is to be of significant and lasting benefit.

Some important limitations are worth noting here, too. The first one is that the above-presented arguments are only partially based on statistical analysis of the data, which was carried out for the small number of TADS patients who met criteria for borderline features or disorder. This limits the findings' reliability and generalisability.

Second, most of these patients met criteria for at least one more personality disorder, as well as in many of the cases – for multiple personality features. Taylor (2003) pointed to the constant interaction between different aspects of the personality, as well as between personality and the environment. He stated that object relations, personality structure and the experience of symptoms are all “dimensional rather than categorical, and dynamic rather than static in nature” (Taylor, 2003, p. 28). Focusing on borderline personality features and depression alone, without examining the contribution of other personality features and wider environmental factors is bound to be a limited way of approaching patients’ presentation and treatment outcome. Yet, it is one step further in the direction of understanding the complex nature of these patients’ difficulties and the support needs they might have.

Third, it is hard to ascertain the degree of clinical significance these personality changes hold. As noted already, Shedler (2016) suggested that a change score of more than 5 points on a *scale* level denotes a clinically significant change, whilst also stressing the importance of evaluating clinical significance on an individual, case-by-case basis, too. Furthermore, the DSM-5 advises against giving a BPD diagnosis to individuals diagnosed with depression, when only cross-sectional information is available (APA, 2013). The Manual stresses the importance of ascertaining the longitudinal and enduring nature of personality pathology, as well as its onset in adolescence/early adulthood, which is one of the main criteria for PD diagnosis in the DSM. The same longitudinal approach would need to be taken when assessing personality changes. This was unfortunately not within the remit of the current project.

Given the small number of patients who actually met criteria for *borderline features or disorder*, the author decided not to carry out any higher order analyses, such as analysis of variance, but rather to explore how changes in the borderline dysregulated score in the course of treatment related to treatment outcome.

#### ***4.1.1.2. Borderline-dysregulated personality change score***

A borderline-dysregulated personality change score was calculated in order to test whether changes in patients' degree of resemblance of the borderline personality disorder prototype were significantly linked to changes in any of the outcome variables at the end of treatment and follow-up. Furthermore, as the borderline-dysregulated prototype is one of eleven Q-factor prototypes, change scores were also calculated for the remaining ten prototypes and correlations were calculated for all of them with each other.

Presented below is the summary and interpretation of this set of analyses for each of the treatment groups.

##### ***4.1.1.2.1. The LTPP group***

Four of the eleven SWAP-II personality prototypes' change scores were significantly related to changes in one or more of the outcome measures. These were the *depressive*, *anxious-avoidant*, *borderline-dysregulated* and *hysteric-histrionic* scales.

A decrease on the *depressive* and *anxious-avoidant* prototype score at the end of treatment was significantly linked to a decrease in the symptoms of depression both at 18- and 42-months, as well as to a decrease in psychological distress and increase in general functioning. Moreover, changes in the two personality prototypes were also significantly related to one another.

A decrease in the *hysteric-histrionic* score, on the other hand, was only associated with an increase of the patient's general functioning at the end of the follow-up period. Interestingly, this prototype was negatively correlated with the *anxious-avoidant* one.

Lower scores on the *borderline-dysregulated* prototype were related to a decrease in depressive symptomatology at the end of the follow-up period, as well as with increase on the *personality*

*health* prototype. Increased *personality health*, in turn, was significantly related to a decrease in depressive symptoms, psychological distress, fewer Axis I disorders, and to an increase in general functioning, all at the end of the follow-up period. Furthermore, better personality functioning was also significantly related to lower scores on the *anxious-avoidant* and *paranoid* prototypes. Finally, the *borderline-dysregulated* prototype was significantly associated with the *paranoid* and the *dependent-victimised* prototypes (decrease in one leading to decrease in the other).

Whilst it is hard to offer the reader a comprehensive interpretation of these results, which will account for all changes in the personality prototype scores in relation to the three outcome measures, the finding suggest that in the LTPP group changes in the different personality disorder features in the course of treatment and follow-up, was related to improvement in depression and general functioning. This appears in line with previous literature, where personality difficulties, in general, were linked to poor outcome in treatment-resistant depression (e.g. Petersen et al., 2002).

What is more difficult to establish is whether there is a causal relationship – namely, whether a decrease in personality difficulties in general, and borderline personality disorder/features in particular, accounts for better treatment outcome for depression. Moreover, it could be argued that personality health is the product of, rather than the cause for, a decrease in depression, and the reduction in personality difficulties is also a consequence of individuals being less depressed, and therefore abler to function in the world. If this were indeed the case, however, one might expect that an increase in personality health and decrease in personality difficulties, as measured by higher change scores on the SWAP-II prototypes listed above, would be significantly related to lower depression scores, less psychological distress and better general functioning at the end



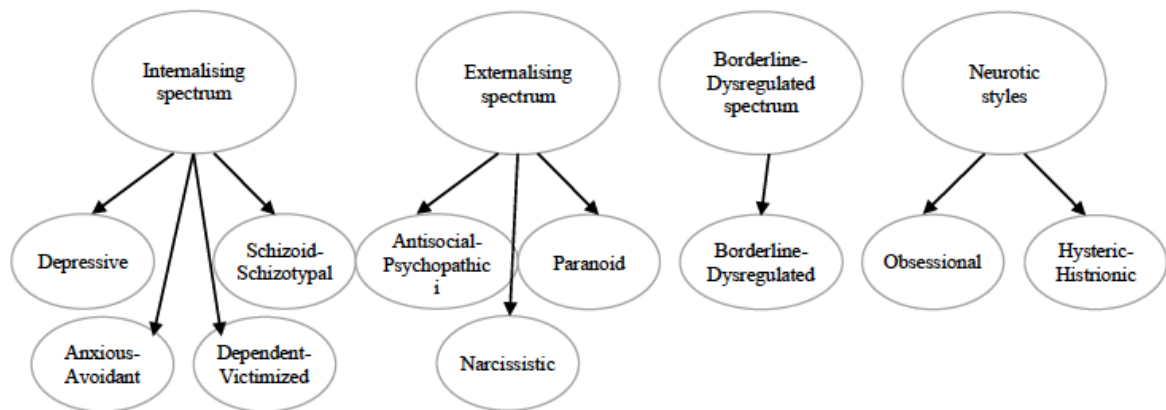
of treatment. This was indeed the case for the *depressive* and *anxious-avoidant* prototypes, but not for the *borderline-dysregulated* one, which is the main subject of this thesis.

The statistically significant relationship between the *borderline-dysregulated* and *personality health* prototypes, on the one hand, and the outcome measures on the other hand at the end of the follow-up period, but not at the end of treatment, supports the hypothesis that personality difficulties and depression interact, rather than one leading to the other in a straight-forward manner (e.g. Howland & Thase, 2005; Kay, Garside, Beamish, & Roy, 1969; Scott, 1988). Furthermore, the link between changes in the borderline score and improvement in depression could also be accounted for by the so-called “sleeper effect” of psychodynamic psychotherapy (Fonagy et al., 2015). This refers to the observed increase in patients’ improvement in the LTPP group in the course of the two-year follow-up, which was argued to explain the bigger effect sizes of the difference between the LTPP and TAU groups, in relation to the main outcome data (ibid). In other words, patients’ functioning (personality as well as their experiences of depression) continued to improve in the follow-up period.

Returning to the SWAP-II literature, the reader is also reminded that apart from the eleven personality prototypes, the SWAP developers also derived (through factor analysis) three overarching personality spectra: *internalising*, *externalising* and *borderline*, as well as a separate cluster of *neurotic styles* (Westen et al., 2012). These were presented in more detail in the methods chapter of this thesis, but Figure 5 overleaf reminds the reader of the spectra, and the specific PDs that fall under each of them.

Figure 5

*Hierarchical Structure of Personality Diagnoses* (adapted from Westen et al., 2012)



It appears that the TADS patient group fell mostly in the *internalising* spectrum (particularly as the *anxious-avoidant*, *depressive* and *schizoid-schizotypal* disorders were also highly prevalent in the LTPP and TAU groups). Given the relatively low prevalence of *borderline* disorder and features, it seems unusual that this prototype, which also forms a separate personality spectrum (ibid) correlates with changes in the treatment outcome measures' scores. The following definition by Shedler (2015) might be relevant to the interpretation of these results:

“Individuals in the borderline-dysregulated spectrum are qualitatively distinct from stable internalizers or stable externalizers. Their perceptions of self and others are unstable and changeable, and they show impaired ability to regulate emotion. As a result, they tend to oscillate between emotions characteristic of both internalizing and externalizing spectrum pathology (e.g., depression, anxiety, rage). They may best be described as “stably unstable” (Schmideberg, 1959).” (Shedler, 2015, p. 241)

This conceptualisation of the borderline presentation might make more meaningful the correlations between the *borderline* change score on the one hand, and both the *dependent-victimised* and the *paranoid* prototype change scores. In other words, a decrease in borderline presentation is linked to a decrease in feelings of depression, as well as to other personality dysfunctions which fall under both the internalizing and externalizing spectra, but are not best

captured by either.

Interestingly, higher scores on the *hysteric-histrionic* prototype were linked to better general functioning at the end of the follow-up period. Whilst this might be a counter-intuitive result at first (as it suggests more pathology being linked to better functioning), Westen et al. (2012) actually argue that the *neurotic* styles (one of which the hysteric-histrionic prototype is) are characterized by less impairment than the other personality syndromes and “may therefore be considered character *styles* rather than disorders” (p.282).

Similarly to Westen et al. (2012) , Kernberg & Caligor (2005) conceptualised personality disorders across three levels: borderline, neurotic and psychotic. The neurotic level of organisation, they argued, is characterised by more integrated self- and others- representations, than the borderline and psychotic one, as well as by the use of more “mature” defences and a better reality-testing. Obsessive-compulsive, hysterical and depressive-masochistic personality disorders were considered to fall under this category.

#### 4.1.1.2.2. The TAU group

In the TAU group only the *borderline-dysregulated* prototype change score was significantly related to the HDRS-17 at the end of treatment. Furthermore, a decrease in the prototype score was associated with an increase in global functioning (GAF) at the end of treatment and at follow-up, and a decrease in psychological distress (CORE-OM) at the end of treatment.

Interestingly, an increase in the *dependent-victimised* and *obsessional* scores was significantly linked to an increase in global functioning at 42-months.

Like in the LTPP group, a decrease in the *borderline-dysregulated* score was associated with a decrease in the *paranoid* score. A negative correlation was observed with the obsessional prototype score, however – the lower the borderline-dysregulated score, the higher the obsessional one.

One way of interpreting the results in the TAU group is to again refer to the factor model by Westen et al. (2012). It can then be argued that in the TAU group improvements in general functioning were linked to a decrease in a more borderline-type functioning and a shift towards internalizing (dependent-victimised) and neurotic presentation (obsessional personality). At the same time such an interpretation does not account for the categorical shifts in the borderline prototype, observed in this group: whilst only five people scored above cut-off for borderline-dysregulated features and none for disorder at baseline, at the end of treatment eight people met criteria for features and one for disorder. It is also interesting to notice that in the TAU group anxious-avoidant and depressive PD were also highly prevalent at baseline and end of treatment, and yet these prototypes' change scores did not correlate significantly to treatment outcome.

#### *4.1.1.2.3. Differences between the two groups*

The first difference between LTPP and TAU is that changes in score on the borderline-dysregulated prototype in the former were significantly related to a decrease in depression at the end of follow up only, whereas in the TAU group this link was statistically significant only at the end of treatment. This might be accounted for by the “sleeping effect” in the LTPP group, as described earlier. It could also be argued that in the LTPP group treatment outcome, as measured by the HDRS, CORE-OM and GAF, appeared to be related to changes in a number of the personality prototypes, which was less so in the TAU group.

Another interesting difference is that in the LTPP group the borderline-dysregulated prototype was related to personality health, which in turn was also significantly linked to treatment outcome. This was not the case in the TAU group. This, as argued earlier, might be linked to the different aims of the LTPP and TAU treatments – the former focusing more intently on leading to changes at the level of personality organisation, and the latter, generally speaking, targeting symptoms of depression alone.

It remains unclear, however, why the borderline-dysregulated personality prototype was significantly related to treatment outcome in both groups, despite the low number of people who met criteria for *features* or *disorder*. In the following section the author presents a discussion of the analysis of specific borderline-dysregulated characteristics and their link to treatment outcome in the two TADS treatment groups.

#### ***4.1.1.3. Borderline-dysregulated items and their link to treatment outcome***

The introduction chapter summarised different conceptualisations of borderline personality disorder, as well as the diagnostic descriptions provided by the DSM-5 and by psychoanalytic authors. Regardless of the theoretical model used, borderline personality has been described in the literature as a constellation of different characteristics, encompassing different domains of functioning (e.g. affect regulation; interpersonal relationships etc.). Some authors have further attempted to refine the borderline construct by identifying aspects of it that are most characteristic and distinctive of borderline personality disorder.

Westen & Shedler (2007), for example, referred to an unpublished examination (Bradley, Shedler, & Westen, 2006) of the highest-ranking SWAP borderline-dysregulated items, which led to a narrowing down to 15 “core” items, considered to be both characteristic and distinctive of BPD (e.g., “Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.”), as well as to a “stable core of items” which were characteristic but not distinctive – mainly thought to indicate negative affectivity, observable in other personality disorders, too. In addition, the same item analysis identified features that are “highly distinctive” of BPD, but only observable under extreme stress (e.g., “Tends to engage in self-mutilating behavior”). The distinction of those features of the disorder which are stable over time, from those which “wax and wane” is considered important (Westen & Shedler, 2007), as a focus on the latter more than the former, can lead to the wrong impression that borderline personality disorder is unstable over time (Shedler, 2016). Yet, some of the more stable features are at the same time argued to be less

distinctive of BPD (e.g. negative affectivity). Shedler (2016) indeed proposed that the borderline-dysregulated prototype item “Tends to feel unhappy, depressed or despondent” is not a BPD-specific item, as it indicates depression, but argued that all other 23 prototype items are specific, albeit still a combination of “stable” and “unstable” ones (e.g. the items relating to self-harming behavior).

Clarkin, Fonagy, Levy, & Bateman (2015), argued that *impulsivity* is most characteristic of the disorder, but warned that the concept of impulsivity overlaps with other concepts such as “sensation-seeking, risk-taking, lack of planning, inability to delay gratification, insensitivity to consequences of action, and alteration in the perception of time” (p.355). In addition, the authors noted that negative affect is also a characteristic feature of BPD, but is found in other disorders. According to Clarkin et al. (ibid), however, negative affect in borderline patients is distinct by its transiency and fluctuations, as well as by being triggered by environmental stimuli.

In a nutshell, it appears that some of the characteristics of BPD are more distinctive of the disorder than others, and that certain expressions of it, like self-harming behaviours, might be only observable under stress, but not at all times. The analysis of each borderline-dysregulated item aimed, therefore, to examine which of the borderline-dysregulated items had specific relevance for treatment outcome, as measured by the HDRS, CORE-OM and GAF scales.

#### *4.1.1.3.1. Borderline-dysregulated items at baseline*

Only two items at baseline correlated negatively with treatment outcome in the TAU group. These represented chaotic lifestyle, which predicted poorer outcome on the measure of depression at the end of treatment, and self-harm which predicted less improvement in psychological distress at the end of the follow-up period. Interestingly, one of the items indicating insecure attachment, correlated significantly with the psychological distress change score, suggesting that the more insecurely attached patients were at the beginning of the trial, the

higher they scored on their 18- and 42-months CORE-OM compared to their baseline score on the measure.

The reader is reminded that out of the three outcome measures, the CORE-OM is the only self-rated one. One hypothesis about the above-reported results could be that the more insecurely-attached patients sought the therapist's/researcher's approval more and their higher CORE-OM scores at the end of treatment and end of follow-up in the TAU group were an expression of a tendency to please the other.

It is important to note here that attachment styles classifications go beyond a "secure-insecure" distinction, however, but attachment nuances were not captured by the current study. This is a limitation of this study, for, as presented earlier, research has found *anxious-preoccupied* attachment strategies to be of a particular relevance to BPD, and to the way in which individuals make use of therapeutic interventions (Bateman & Fonagy, 2016; Fonagy & Luyten, 2016).

In the LTPP group a similar positive correlation was observed between one of the items measuring splitting and the CORE-OM change score at 18 and 42 months. This result is even more bewildering and hence difficult to interpret. One way of making sense of it could be that the CORE-OM scores for patients more prone to splitting, were not reflecting as accurately their state of mind, but rather only the more positive (split-off) aspects of their experiences. It is unclear why this trend was observed in the LTPP but not in the TAU group.

#### *4.1.1.3.2. Borderline-dysregulated item change scores*

Patients who experienced less improvement in affect instability and insecure attachment (feeling victimised and mistreated, in particular) in the course of treatment in the TAU group, also experienced less improvement in depression at 42 months. Smaller change scores on two further affect dysregulation items were also associated with less improvement in general functioning at

the end of follow-up in this group. Interestingly, an increase in relationship instability was linked to improvement in depression at 42 months.

This latter finding is also bewildering. A possible explanation could take into account the prevalence of dependent and avoidant PDs in the TADS sample, which might suggest that patients overall were more likely to avoid interpersonal conflict and multiple break-ups and separations. An increased relationship instability score might thus reflect a decrease in interpersonal dependency and subjugation, which for some patients might have been a contributing factor to their depression. This hypothesis remains speculative and would need to be tested further, however.

In the LTPP group lower change score on one of the affect dysregulation items alone (becoming irrational when strong feelings were stirred up) was related to less improvement in depression. Better general functioning at 18 months was linked to feeling less victimised and misunderstood, whereas a decrease in splitting was associated with less psychological distress at the end of the follow-up period.

A big limitation of this part of the analysis needs to be noted here, however. More specifically, only 12 out of the 24 items that describe the borderline-dysregulated prototype had a fair to excellent inter-rater reliability correlations, as reported earlier in the results chapter. This led to the other half of the items being omitted from the analysis due to poor reliability. These included the four items describing processes of *projective identification* and *identity diffusion*, as well as one of the two items describing *splitting*, and two of the items describing *self-harm*. Instability and lack of cohesiveness in one's internal representations of self and others, together with the use of more primitive defences such as splitting and projective identification, have been considered by a number of authors to be central characteristics of BPD (e.g. Green, 1977; Kernberg, 1978; Winnicott, 1969). Repeated suicidal and self-harm threats and gestures are also highly characteristics of the disorder (APA, 2013; Clarkin et al., 2015), despite the more



fluctuating nature of this particular trait (Westen & Shedler, 2007). Not being able to test how these items relate to treatment outcome is a drawback of the current study. The limitation of the poor reliability of certain items will be returned to later in this chapter.

The borderline-dysregulated items which were included in the analysis thus encompassed mainly *affect dysregulation*, *insecure attachment* and *chaotic lifestyle*. It is therefore not surprising that predominantly improvements in the patient's ability to regulate emotions (affect), as well as a change in their attachment patterns were linked to improvements in depression, general functioning and psychological distress.

As noted previously, a number of publications have explored the link between attachment and the capacity to mentalise (e.g. Fonagy & Luyten, 2016). One's capacity to regulate affect, develops in the context of one's relational capacities (secure attachment to others). When one's way of relating to others is marked by insecurity and instability, deficits in affect regulation ensue. Mentalization theory suggests that when one's emotions cannot be represented and regulated internally (be given meaning to, in the context of the individual thinking about their own and others' minds), three modes of less adaptive functioning take over: *psychic equivalence* (inner states being experienced as equivalent to external facts), *teleological mode* (inner experiences being regulated only by action), and *pretend mode* (thoughts and feelings can be thought about and described but get dissociated from the person's actual lived experience).

Splitting, projective-identification and self-harm can be seen as expressions of these three modes of responding to and regulating intense affect, characteristic of borderline personality disorder. Insecure attachment and affect dysregulation alone, however, are not exclusively characteristic to BPD, but can encompass a wider spectrum of personality difficulties. More specifically, it appears that affect dysregulation and insecure attachment in the TADS patient group, both at the beginning, as well as at the end of treatment, were linked to experiences of psychological distress in general, and depression in particular. Improvement in the course of treatment on the

three outcome measures is associated with improvements in affect regulation and attachment style. This is despite the fact that only two people in the whole patient group met criteria for borderline-dysregulated disorder, and a small proportion (8% in TAU, and 13% in LTPP) scored above the SWAP-II cut-off for borderline features.

The Psychodynamic Diagnostic Manual (PDM Task Force, 2006) considers borderline personality as a level of organisation subsuming other personality disorders and hence an expression of a personality difficulties more generally. Whether borderline personality disorder is a separate, albeit heterogeneous construct, akin to other personality constructs in terms of its distinct structure and manifestation, or a higher level personality organisation, which includes different personality disorders, including BPD, appears to remain an on-going debate in the literature (Meehan & Levy, 2015). The results from both the TAU and the LTPP groups provide some evidence in support of the more encompassing nature of certain borderline features, such as insecure attachment and affect dysregulation, and their relationship with depression and general distress and functioning. Due to poor item reliability, however, other aspects of the borderline prototype, which might be particularly distinctive of it (e.g. self-destructive behaviours and identity diffusion) could not be entered into the analysis.

#### **4.1.2. Personality disorders frequency and comorbidity**

Another very striking finding presented in the Results chapter of this thesis, was that the majority of the TADS participants (over 90%) met SWAP-II criteria for at least one personality disorder. Moreover, the average number of personality disorders per patient were approximately 2 at baseline and 2 at the end of treatment for both groups, when the SWAP-II measure was used. The three most prevalent personality disorders both in the LTPP and TAU group were *depressive, anxious-avoidant* and *schizoid-schizotypal*.

The author also calculated the average number of personality disorders as measured by the SCID-II-PQ. Based on this measure, patients in the TAU group met criteria for an average of 4.2, 3.4 and 3.5 personality disorders at baseline, end of treatment and end of follow-up respectively, and patients in the LTPP group met criteria for an average of 4.6, 3.6, and 2.9 personality disorders at the three time points, respectively. The SCID-II-PQ thus points to a larger personality disorder comorbidity than the SWAP-II.

These results need to be interpreted with a lot of caution. As noted in the introduction chapter, the SCID-II-PQ is a screening tool, which is to be used in combination with the SCID-II semi-structured interview, where the interviewing clinician asks further and more detailed questions, before arriving at a personality disorder diagnosis. It is therefore likely that the SCID-II-PQ, when used on its own, over-estimates the prevalence of personality disorder. Furthermore, it is a self-rating tool, which makes it less reliable (when compared to a clinician- or multiple-informant based diagnosis). The SCID-II-PQ will therefore not be considered in any further detail in the rest of this chapter.

The differences in the average number of PD diagnoses as measured by the SWAP-II and SCID-II-PQ are generally in line with what was reported by Lewis (2008), who used an earlier version of the SWAP – the SWAP-200 – to look at a sub-sample (N=25) of TADS participants and found an average PD prevalence of 1.2 SWAP-200 Q-factor diagnoses, and 4.6 SCID-II-PQ diagnoses. Another recent study by Marin-Avellan, McGauley, Campbell, & Fonagy (2014) also demonstrated that when the SWAP is used as a measure of personality disorders, there is 50% less PD comorbidity, compared to using the SCID-II. At the same time, the current project found an average of roughly 2 SWAP-II personality disorders per participant, both at the beginning and end of treatment, which is twice as high as the one PD diagnosis in a “typical patient”, reported by Westen & Shedler (1999).

One possible interpretation of the high number of personality disorder prevalence and comorbidity in the TADS patient group could be that it is a reflection of the high complexity of the TADS patients' presentation and difficulties. As reported in the method chapter, the majority of participants met criteria for "severe" to "very severe" depression, as measured by the HDRS at baseline, and had experienced re-current depression for many years with a number of treatments (approximately four on average) offered to them, which had led to little or no improvement.

Furthermore, a high proportion of the patients in both groups (over 70% in both groups) met criteria for anxiety disorder, and had an average of over 3 Axis I disorders at baseline. Although the data collected on abusive and traumatic experiences was incomplete and therefore not officially reported in the main outcome journal article (Fonagy et al., 2015), a large number of patients also appear to have experienced one or more traumatic events at different stages of their life. It could therefore be hypothesised that the high personality disorder prevalence is an expression of the enduring difficulties of these patients, which go far beyond problems with mood alone and spread across different areas of their lives in a complex and incapacitating manner.

A more detailed interpretation of the personality disorders prevalence and comorbidity also goes beyond the aims and scope of the current thesis, and will hopefully be addressed in a separate future publication by the TADS research team. The issues of complexity, in general, and trauma, in particular, are addressed next.

#### **4.2. The Role of Trauma and Victimisation**

The Introduction chapter of this thesis presented some theoretical and clinical ideas about the link between attachment difficulties and the experience of trauma and victimisation. Insecure attachment was in turn linked to personality development, as expressed through stable

representations of self- and others, the ability to regulate emotions, and the capacity to mentalize. Despite the different views on what constitutes “trauma” and which types of trauma are particularly predictive of borderline pathology, there seems to be a unanimous agreement amongst authors that most borderline patients have experienced some form of early life adversity. Bateman & Fonagy (2016) draw attention to the overlap between BPD and “complex traumatic stress disorder”. The British Psychological Society has further suggested that the term “personality disorder” is replaced by “complex trauma” or “complex trauma reaction” (Division of Clinical Psychology Beyond Functional Psychiatric Diagnosis Committee, 2015).

The SWAP-II borderline prototype item “Tends to feel misunderstood, mistreated or victimised” was linked to a few of the outcome measures. For example, decrease of score on this item was associated with lower depression scores in the TAU group at 42 months, and improved general functioning in the LTPP at the end of treatment.

A significant proportion of the TADS patients had reported experiences of loss, physical and sexual violence, and bullying. The exact statistics of this have not been yet published, as the analysis of this part of the data set is not complete, but a summary was presented by the TADS research team during the 45<sup>th</sup> SPR International Annual Meeting (Booker, 2014). Some patients had experienced a combination of adverse events, on a number of occasions throughout their life, prior to taking part in the TADS. In addition, 18% of participants had reported being in an abusive relationship at the beginning of treatment. This calls for a discussion of patients’ experiences of feeling mistreated and victimized, as measured by the SWAP-II, and the link with actual experiences of trauma and abuse, as well as the implications this has for the treatment of depression.

André Green argues that identity fragmentation, deficits in thinking, and the use of splitting and projection are all unconsciously resorted to by patients with BPD, in order to defend the ego against getting in touch with complex underlying traumatic “constellations”, which threaten to

cause a “psychic catastrophe” (Green, 2000). Attachment theorists take a different view, in arguing that fragmentation and difficulties in metalizing are caused by disrupted early attachments (as summarised above), rather than by repression or the overreliance on certain unconscious defences. Clearly more patients in the TADS group had reported traumatic experiences than those who could be considered to present with borderline features or disorder. The link between insecure attachment, affect dysregulation and treatment outcome might, however, also be further understood in relation to such experiences. It is therefore interesting that this has not been addressed in the main TADS outcome paper (Fonagy et al., 2015), and that the data on adverse experiences is incomplete, and thus not suitable for being entered into the analysis of the current thesis, either.

Herman (1997) draws attention to trauma victims often not being believed in society and states that silencing and denial are common social processes. She argues that both social and political contexts need to allow for the holding of traumatic realities into consciousness. “Repression, dissociation and denial are phenomena of social as well as individual consciousness” (p.9).

Alessandra Lemma presents a similar argument, although with more general reference: “What we see and hear in the clinical situation tells the story of an individual, couple, or family and their relationships, but it is also a current, running commentary on the society we live in” (Lemma, 2012, p. 67). She insists that it is the mental health professionals’ moral obligation to use their ideas and understanding of the mind in impacting what happens on a social level, outside of the consulting room.

Both Herman and Lemma’s arguments call for researchers and clinicians to be more daring in asking questions about social processes, including injustice and adversity, and in inviting their contemporaries to know and think about these wider issues. The TADS patients’ experiences of often multiple loss and violence should not, therefore, be left out of sight and out of mind. This thesis did not succeed in factoring such experiences in an organised and systematic way into the

data analysis, but the author certainly invites the reader to think about the implications this might have on the results in particular, and on the approach to understanding and treating “treatment-resistant depression”, more generally.

### **4.3. Further Limitations of the Current Study**

#### **4.3.1. The SWAP-II**

##### ***4.3.1.1. The use of the SWAP-II Borderline-Dysregulated Q-sort as a measure of borderline personality disorder***

One of the main limitations of the current study is linked to the use of the borderline-dysregulated T-score as a single measure of BPD. To the best of the author’s knowledge, no previous project has used the SWAP-II in this way. Moreover, the personality profiles of those who met criteria for borderline features or disorder demonstrated that personality is a constellation of different features. The description of a patient only in terms of their borderline characteristics, as well as the manipulation in statistical analysis of the borderline prototype as a separate variable, is bound to carry some artificiality. At the same time, this study demonstrated how the SWAP-II produces personality profiles marked by less personality disorder comorbidity, as compared to the SCID-II-PQ. The alleged advantages of the SWAP over the more traditional, DSM-informed categorical approach to personality disorder, were presented in the previous chapters of this thesis. It thus appears that, despite the limitations, the SWAP-II might be a better approach to capturing personality and describing it, both categorically, as well as dimensionally, through the use of the tool’s narrative descriptions.

##### ***4.3.1.2. Differences between SWAP-II completers and non-completers***

One of the findings presented in the previous chapters was that those who were rated on SWAP-II at the end of treatment had a significantly higher average *depressive PD* score than those who were not rated (for reasons listed in the methods chapter). This means that the SWAP data

available at 18 months was representing the patients with a higher degree of difficulties, at least based on their *depressive PD score* at baseline. It is difficult to know whether the rest of the results would have been different, if all data were available.

#### ***4.3.1.3. On scoring the SWAP-II using the TADS clinical material***

A further limitation is related to the type of information used for the scoring of the SWAP-II. Shedler & Westen (2007) advised that the SWAP is scored after the completion of the Clinical Diagnostic Interview (CDI) (Westen & Muderrisoglu, 2003), developed particularly for this purpose. When the CDI cannot be completed, it is recommended that the SWAP-II is scored after a minimum of 6 psychotherapy sessions, in order to allow a good-enough knowledge of the patient and their presentation.

As already discussed in the method chapter, Lewis (2008) compared the CDI to the Tavistock Psychodynamic Interview (TPI) and concluded that the latter can be used as a replacement of the former, despite some of the differences between the two interviews. It is worth noting some of these differences again and the limitations they carry. The four main areas, which are attended to in the CDI interview, but not explicitly asked about in the TPI, concern the patient's sexual life and experiences, their experiences of and feelings about themselves as an individual, the way they manage difficult emotions, and any recent difficulties they might have encountered.

The TPI does not contain a question about sexuality or the patient's current sexual relationships (although it does ask about past and current intimate relationships). This led to the lack of sufficient information required to score any of the SWAP-II items linked to sexual identity, sexual perversions or the patient's attitude towards sexual experiences more generally (e.g. "Appears to associate sex with danger"). This omission is a significant one, as it has led to these items being scored as "0" (i.e. not at all descriptive of the patient), when this information, if available, might have been central to both the clinical formulation, as well as to the patient's personality profile.



A similar argument can be made about the CDI asking in a more detailed manner about the patient's self-image (e.g. "Do your feelings about yourself change a lot? Do you ever feel like you don't know who you are, or like the different sides of you don't fit together?" (Lewis, 2008, p. 110)). As already pointed out throughout this thesis, a lack of stable sense of self is one of the central borderline personality disorder features. The same is true about affect dysregulation, which is more thoroughly investigated during the CDI interview. It is thus unclear whether the TADS patients' score on the borderline-dysregulated prototype would have been different, had this information been available.

At the same time the TPI asks some questions, which are not included in the CDI, but nonetheless provide valuable information about the patient's functioning and personality. These include questions about the patient's early life-, as well as more recent experiences of separation and loss, and questions about current social support and about the patient's understanding of their depression. These questions provided the SWAP-II raters with some insight about the patient's relationships with others, including how others are perceived and described by the patient, as well as with information about the patient's capacity to think about their own minds and the minds of others (or in other words "mentalize"). The importance of early experiences of loss and adversity has been referred to in this thesis already. The TPI asked the patients questions which allowed for the exploration of such experiences.

#### ***4.3.1.4. On the importance of well-informed clinical formulation in making accurate and valid personality assessments***

Shedler (2009) stated the the SWAP "relies on clinicians to do what they do best: provide psychologically rich descriptions of the individual patients they know and treat" (p.3). He further recommended that additional sources of information, such as patient records and other psychological tests, should also be used when the SWAP is scored. A wide range of information

was available in the TADS, but it was not possible to fully follow this recommendation for pragmatic reasons (i.e. limitations in time and resources).

Furthermore, the TADS was a pragmatic randomised trial, which means that it tested the effectiveness of interventions already delivered as part of the National Health Service (NHS) in the UK. Clinicians in current NHS settings rarely have enough time to get to know the patient very well, especially in the context of time-limited therapies, which might last 6 sessions in total (e.g. in the case of CBT interventions in primary care). Assessments are usually carried out in no more than one session and if an intervention is offered, it is not unusual that this is delivered by a different clinician. Some patients might have prolonged and multiple contacts with health-care services, but brief treatments provided by different clinicians are more the rule than the exception. This way of working places significant limitations on the clinicians' ability to arrive at a detailed clinical formulation of the patient's difficulties, which includes not only the presenting problem (e.g. symptoms of depression) but also the individual's overall way of functioning and personality organisation. The access to 2-4 hours of recorded assessment material for the purposes of completing the SWAP-II can therefore be regarded as unusual, compared to the limited information and contact time clinicians have before drawing an initial formulation and making treatment recommendations.

Finally, Westen & Shedler (2007) pointed out that the SWAP-II has mainly been used for research purposes. The tool's use in clinical settings and clinical trials has not been thoroughly evaluated (*ibid*), particularly when the SWAP-II was used by a team of (five) researchers, who completed the assessments. It can be argued that the personality profiles might have been more clinically accurate if they were completed by the assessing clinician, who had direct contact with the patient, instead. This is another limitation of the study, and one which was the product of pragmatic restrictions.

### 4.3.2. Outcome measures' limitations

The TADS used the HDRS-17 as a main treatment outcome measure. Secondary measures included the CORE-OM and GAF. Each of these scales and its psychometric properties was presented in the methods chapter. It is important to also note some further limitations, too.

#### 4.3.2.1. *The HDRS-17*

A main critique of the HDRS-17, presented earlier in this thesis, has been the lack of an updated version, which reflects developments in the conceptualisation of depression (Bagby et al., 2004; Gibbons, Clark, & Kupfer, 1993). Yet, no convincing successor of the HDRS seems to have been identified and adopted in the study of depression and its treatments to date.

Furthermore, Bagby et al. (2004) highlighted that the HDRS was developed with the primary aim of measuring the treatment effects of anti-depressants in the late 1950s/early 1960s. This point is important to consider, as depression can be conceptualised in different ways, as demonstrated in the introduction chapter, with different treatments having very distinctive therapeutic aims. For example, anti-depressants' main objective is to reduce the observable, behavioural symptoms of depression, such as sleep disturbance and anxiety. Psychodynamic psychotherapy, on the other hand, aims primarily to achieve greater personal integration, by changing the way the person functions as a whole (Taylor, 2015). Symptom reduction, in that sense, remains a secondary aim, or even a by-product of the main treatment aim. It can thus be argued that the HDRS-17 does not capture optimally changes in the TADS patients' experiences of depression. This hypothesis is further supported by the lack of significant change in the *depressive* PD scores pre- and post-treatment in either of the groups. Depressive PD, as measured by the SWAP-II, was highly prevalent amongst the TADS patients (66.1% and 70.1% in TAU and LTPP, respectively). Furthermore, high scores on the *depressive* PD scale in the TAU group were also linked to poorer general functioning at the end of follow-up. The lack of change in the patients' *depressive* PD presentation might suggest that any therapeutic gains

remained on the level of symptom reduction, rather than impacting the patients' personality functioning.

#### ***4.3.2.2. The GAF***

The General Assessment of Functioning (GAF) measure also has important limitations, which are relevant to the current study. More specifically, different authors have questioned the measure's reliability in capturing clinical improvement.

Moos, Nichol, & Moos (2002), for example, assessed the GAF's value in guiding clinical decision making and predicting treatment outcome, among nearly 10 000 patients with psychiatric and substance misuse diagnoses. The authors used a range of clinician- and self-rated measures and analysed the data they yielded in relation to the GAF scores. They concluded that there was little or no relationship between GAF ratings and symptoms, social and occupational treatment outcome, and hence stated that "these findings cast doubt on the value of including GAF ratings as predictors of treatment outcome". The prognostic accuracy of the GAF has been questioned by subsequent authors too, who have argued that other measures predict prognosis and treatment outcome better (e.g. Aas, 2010). The DSM (APA, 2013) has indeed removed the GAF in its latest 5<sup>th</sup> edition, stating that the measure has been criticised for its "conceptual lack of clarity" and "questionable psychometrics in routine practice" (p.16). The APA (2013) has replaced the GAF with the WHO Disability Assessment Schedule (*ibid*).

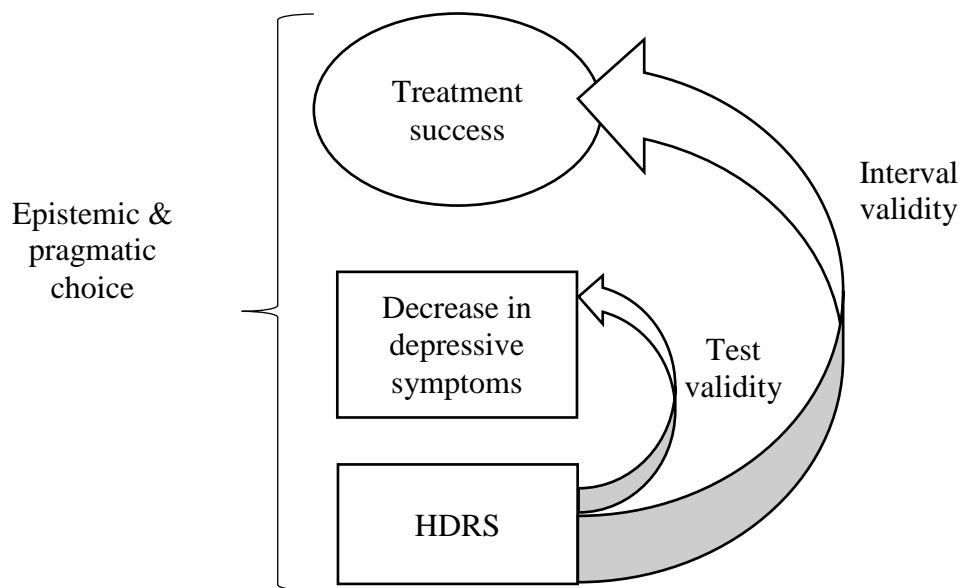
These limitations need to be born in mind when thinking about the changes of the TADS participants' general functioning, as reported in this study. It can be argued that these changes reflect more shifts in symptomatology, than an actual change in the patients' day-to-day experiences across different domains of their lives.

#### 4.3.2.3. The validity of the “outcome measures” in measuring treatment outcome

Another important consideration concerns the extent to which the HDRS, GAF and CORE-OM measure actual treatment outcome. In a recent conference presentation Truijens (2016) addressed the issue of *epistemic validity* in psychotherapy research, or, in other words: how valid are different measures in assessing the *treatment outcome* of psychotherapy interventions. She gave an example with the Beck Depression Inventory, which has good test validity – the scale captures adequately the patient’s experiences of depression. Figure 6 summarises problems arising when using the BDI or similar measures, like the HDRS, to test not depression, but treatment success.

Figure 6

*Test vs. Epistemic Validity* (Adapted from Truijens, 2016)



Truijens argued that the test validity of a measure could not and does not cover the whole epistemic process, which is lodged into the context within which it takes place – social research, and individual. This issue is very relevant to the TADS study, in general, and the current project in particular. More specifically, the main research question concerned the impact of borderline personality features/disorder on treatment outcome for patients presenting with treatment-

resistant depression. Treatment outcome was measured only in terms of the HDRS, CORE-OM and GAF change scores. The TADS also collected qualitative information, however, in the form of interviews with patients and therapists. This information has not been yet fully analysed and published, but it poses the question of what is considered an “outcome” – a change on a depression rating scale or the individual’s experience of having received some support and how this support has impacted on their subjective experiences of depression, as well as on their way of relating to themselves and the world around them, more generally. Truijens (*ibid*) referred to a recent study by Cornelis et al. (2016) which indeed demonstrated how different treatment outcome results might change, based on the type of measurement tool used (in that example - the BDI score, as compared to the patient’s cortisol levels and their use of anti-depressant medication).

Out of the three “outcome measures” used in the current study, the CORE-OM was the only one which was developed with the particular aim of measuring the effectiveness of different types of psychotherapy. Even still, its developers also advised against using the CORE-OM as a stand-alone measure of clinical change and effectiveness, and noted that each service needs to choose other supplementary measures, based on the type of intervention it is delivering (Evans et al., 2000).

#### **4.4. Summary of Clinical Implications and Suggestions for Further Research**

The results of this thesis pointed at the complex personality presentation of the TADS patients, who were being treated in the trial for “treatment-resistant depression”, either through long-term (18-month) psychodynamic psychotherapy, or through treatment as usual. The length and severity of these patients’ depression were much higher than those of other depressed patient groups, on which recommendations such as the NICE guidelines have been based (Fonagy et al., 2015). Axis I comorbidity, and the presence of significant and often multiple personality difficulties were the rule, more than the exception for the TADS participants. A retrospective

measure of personality pathology – the SWAP-II – further pointed at the high prevalence of PDs in the TADS population.

The current study did not find a significant link between borderline personality disorder or features at the beginning of treatment, and treatment outcome, as measured by changes in patients' HDRS, CORE-OM and GAF scores. Very few patients met criteria for borderline-dysregulated disorder or features, although this number increased in the TAU group by the end of treatment, compared to baseline. Those who did meet these criteria, appeared to experience little benefit from the the treatment offered in both the TAU and LTPP groups, with the exception of significant improvement in general functioning, as measured by the GAF, in the LTPP group. The TAU patients in this subgroup generally deteriorated in their borderline presentation in the course of treatment.

These findings have important clinical implications. First, they demonstrate that not all patients benefit from the interventions offered, even when they present (at least at face value) with the same symptoms – for example, of major depression. The study's results appear in support of the psychoanalytic view of mental health difficulties being lodged in the development of the personality, particularly for individuals whose depression is “resisting” first-line forms of treatment (e.g. anti-depressants or brief psychological therapies). Fast-tracking patients to and through treatments, only on the basis of meeting a set of psychiatric diagnostic criteria is, therefore, not sufficient in accurately predict the effectiveness of interventions. A thorough assessment of the person's presentation, including any possible personality difficulties, seems very much needed for patients presenting with more complex and enduring difficulties. Only then can a better-informed decision be made about treatment suitability.

Whist this study did not look into other personality disorders in detail, a significant finding appeared to be the overall lack of improvement of most of the PD scores, as measured by the SWAP-II, and the increased scores on some of the PD scales by the end of the treatment. The

possible harm inflicted by inappropriate interventions has been highlighted by some authors (e.g. Fonagy & Bateman, 2006), although this seems to remain an issue, which is generally not openly discussed in RCTs. The thesis discussed different treatment adaptations suggested in the literature, such as considering the patient's capacity to "mentalize", where a more borderline presentation is apparent, and adapting the therapeutic stance in order to meet the individual idiosyncratic treatment needs. Such an approach to treatment calls for on-going reformulation of the patient's difficulties, in addition to a thorough assessment.

This is, however, made difficult by the limited access to resources in contemporary public health care services. Diagnoses and treatment guidelines are one way of organising the immense mass of human complexity and experiences and responding to it in treatment. Diagnostic labels arrived at through assessment might serve as a shortcut to deciding what interventions to use, and how to adapt them, but they are always bound to simplify what a whole living person brings into the consulting room - namely their histories, their past and current relationships, the myriad of their conscious and unconscious experiences, and of course – the reflection of the society they live in.

The TADS patients with borderline features or disorder, all seemed to fit many diagnostic boxes, with no one intervention or theory being able to fully capture their complexity. Further research therefore needs to continue examining the links between chronic and difficult-to-treat depression, and personality disorder (including borderline personality).

Shedler (2009) gives some further guidance on how these issues need to be addressed clinically:

“Although acute Axis I symptoms may prompt the patient to seek treatment, underlying personality patterns must be addressed to achieve lasting treatment gains. The therapist should identify and explore self-defeating patterns in the patient's thinking, behavior, and relationships, including the relationship with the therapist, and help the patient gain insight into the ways in which he discounts or repudiates his own emotional needs or



inhibits his capacity to fulfil them.” (p.11)

Future research needs to also consider using a wider range of measures, which are not predominantly focused on symptoms and illness (like the HDRS and GAF), but more accurately reflect the individuals functioning and lived experiences across different areas of their lives. The same is true for personality disorder measures. The SWAP-II assessments in this study had a number of limitations, and future research should seek to improve the methodology, by, for example, recruiting the treating clinicians into rating the SWAP-II.

Finally, further refinement of both the theoretical and clinical understanding of what works for whom in treating TRD is needed. Blatt (2015) highlights the “Dodo bird effect” in treatment outcome for bona fide therapies for depression, where roughly half of the patients in RCT’s benefit from the intervention offered, whilst the other half do not. The current study identified that improvements in affect regulation and insecure attachment are associated to improvements in depression, general functioning and psychological distress. More research is needed to identify the specific processes that account for such changes and whether and how they depend on the patient’s personality, their history and the type of treatment offered.

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## Appendices

### Appendix A – SWAP-II Description of Borderline-dysregulated Personality

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#### **Affect dysregulation**

Emotions tend to change rapidly and unpredictably.

Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.

Tends to become irrational when strong emotions are stirred up; may show a significant decline from customary level of functioning.

Is prone to intense anger, out of proportion to the situation at hand (e.g., has episodes of rage).

Is unable to soothe or comfort him/herself without the help of another person (i.e., has difficulty regulating own emotions).

Tends to “catastrophize”; is prone to see problems as disastrous, unsolvable, etc.

Tends to feel unhappy, depressed, or despondent.

#### **Splitting**

When upset, has trouble perceiving both positive and negative qualities in the same person at the same time; sees others in black or white terms (e.g., may swing from seeing someone as caring to seeing him/her as malevolent and intentionally hurtful).

Tends to stir up conflict or animosity between other people (e.g., may portray a situation differently to different people, leading them to form contradictory views or work at cross purposes).

#### **Projective identification**

Manages to elicit in others feelings similar to those she/he is experiencing (e.g., when angry, acts in such a way as to provoke anger in others; when anxious, acts in such a way as to induce anxiety in others).

Tends to draw others into scenarios, or pull them into roles, that feel alien or unfamiliar (e.g., being uncharacteristically insensitive or cruel, feeling like the only person in the world who can help).

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**Identity diffusion**

Lacks a stable sense of who she/he is (e.g., attitudes, values, goals, and feelings about self, seem unstable or ever-changing).

Is prone to painful feelings of emptiness (e.g., may feel lost, bereft, abjectly alone even in the presence of others).

**Insecure attachment**

Tends to be needy or dependent.

Appears to fear being alone; may go to great lengths to avoid being alone.

Tends to fear she/he will be rejected or abandoned.

Tends to become attached quickly or intensely; develops feelings, expectations, etc. that are not warranted by the history or context of the relationship.

Tends to feel misunderstood, mistreated, or victimized.

**Self-harm (desperate efforts to self-regulate)**

Tends to engage in self-mutilating behavior (e.g., self-cutting, self-burning).

Tends to make repeated suicidal threats or gestures, either as a “cry for help” or as an effort to manipulate others.

Struggles with genuine wishes to kill him/herself.

**Chaotic lifestyle**

Relationships tend to be unstable, chaotic, and rapidly changing.

Work life and/or living arrangements tend to be chaotic or unstable (e.g., job or housing situation seems always temporary, transitional, or ill-defined).

Tends to be impulsive.

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**Appendix B - Narrative Description of the Borderline-dysregulated Personality**

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*Summary Statement: Individuals with Borderline-Dysregulated Personality have impaired ability to regulate their emotions, have unstable perceptions of self and others that lead to intense and chaotic relationships, and are prone to act on impulses, including self-destructive impulses.*

Individuals who match this prototype have emotions that can change rapidly and spiral out of control, leading to extremes of sadness, anxiety, and rage. They tend to “catastrophize,” seeing problems as disastrous or unsolvable, and are often unable to soothe or comfort themselves without the help of another person. They tend to become irrational when strong emotions are stirred up, showing a significant decline from their usual level of functioning. Individuals who match this prototype lack a stable sense of self: Their attitudes, values, goals, and feelings about themselves may seem unstable or ever-changing, and they are prone to painful feelings of emptiness. They similarly have difficulty maintaining stable, balanced views of others: When upset, they have trouble perceiving positive and negative qualities in the same person at the same time, seeing others in extreme, black-or-white terms. Consequently, their relationships tend to be unstable, chaotic, and rapidly changing. They fear rejection and abandonment, fear being alone, and tend to become attached quickly and intensely. They are prone to feeling misunderstood, mistreated, or victimized. They often elicit intense emotions in other people and may draw them into roles or “scripts” that feel alien and unfamiliar (e.g., being uncharacteristically cruel, or making “heroic” efforts to rescue them). They may likewise stir up conflict or animosity between other people. Individuals who match this prototype tend to act impulsively. Their work life or living arrangements may be chaotic and unstable. They may act on self-destructive impulses, including self-mutilating behavior, suicidal threats or gestures, and genuine suicidality, especially when an attachment relationship is disrupted or threatened.

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**Appendix C – Tavistock Adult Depression Study Ethics Approval**

**NHS**

**West Midlands Multi-centre Research Ethics Committee**

Our Ref: MT/MREC/02/7/35/approval  
(Please quote in all correspondence)

Professor Phil Richardson  
Tavistock & Portman NHS Trust  
120 Belsize Lane  
London  
NW3 5BA

Directorate of Public Health and Policy Development  
Birmingham Health Authority  
St Chad's Court  
213 Hagley Road  
Birmingham  
B16 9RG

Tel: 0121 695 2382  
Fax: 0121 695 2233  
email: maureen.thrupp@hq.birminghamha.wmids.nhs.uk

28 May 2002

Dear Professor Richardson

**Research Protocol Title: RCT of psychoanalytic psychotherapy for refractory depression**

The Chairman of the Multi-centre Research Ethics Committee has considered the amendments submitted in response to the Committee's earlier review of your application on 25<sup>th</sup> April 2002, as set out in our letter dated 2<sup>nd</sup> May 2002.

The documents that have now been approved are as follows:

*Patient Information Sheet, dated May 2002*  
*Information for Research Participants regarding the use of Videotapes, dated May 2002*  
*Application Form, dated 19<sup>th</sup> March 2002*  
*Consent Form, dated March 2002*  
*GP Information, undated*  
*Protocol, undated*  
*BDI Questionnaire*  
*CORE Questionnaire*  
*PROQ-2 Questionnaire*  
*Q-les Questionnaire*  
*PRP Questionnaire*  
*CSRI Questionnaire*  
*CV, undated*

The Chairman, acting under delegated authority, is satisfied that these accord with the decision of the Committee and has agreed that there is no objection on ethical grounds to the proposed study. I am, therefore, happy to give you our approval on the understanding that you will follow the conditions of approval set out below. A full record of the review undertaken by the MREC is contained in the attached MREC Response Form. The project must be started within three years of the date on which MREC approval is given.

The Central Office for Research Ethics Committees is responsible for the operational management of Multi-centre Research Ethics Committees

### Conditions of Approval

- No research subject is to be admitted into the trial until agreement has been obtained from the appropriate local research ethics committees.
- You must follow the protocol agreed and any changes to the protocol will require prior MREC approval.
- If projects are approved before funding is received, the MREC must see, and approve, any major changes made by the funding body. The MREC would expect to see a copy of the final questionnaire before it is used.
- You must promptly inform the MREC and appropriate LRECs of:
  - (i) deviations from or changes to the protocol which are made to eliminate immediate hazards to the research subjects;
  - (ii) any changes that increase the risk to subjects and/or affect significantly the conduct of the research;
  - (iii) all adverse drug reactions that are both serious and unexpected;
  - (iv) new information that may affect adversely the safety of the subjects or the conduct of the trial.
- You must complete and return the standard progress report form to the MREC one year from the date on this letter and thereafter on an annual basis. This form should also be used to notify the MREC when your research is completed.

**While the MREC has given approval for the study on ethical grounds, it is still necessary for you to obtain management approval from the relevant Clinical Directors and/or Chief Executive of the Trusts (or Health Boards/HAs) in which the work will be done.**

### Local Submissions

It is your responsibility to ensure that any local researcher seeks the approval of the relevant LREC before starting their research. To do this you should submit the appropriate number of copies of the following to the relevant LRECs:

- this letter
- the MREC Application Form (including copies of any questionnaires)
- the attached MREC response form
- Annex D of the Application Form
- one copy of the protocol
- the final approved version of the Patient Information Sheet and Consent Form

It is important to check with the respective LRECs the precise numbers of copies required as this will vary and failure to supply sufficient copies could lead to a delay. In addition, you should submit to LRECs only the revised paperwork reflecting the requirements of the MREC as referenced in the response form.

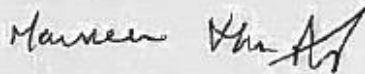
**Local Sites**

Whilst the MREC would like as much information as possible about local sites at the time you apply for ethical approval it is understood that this is not always possible. You are asked, however, to send details of local sites as soon as a researcher has been recruited. This is essential to enable the MREC to monitor the research it approves.

**ICH GCP Compliance**

The MRECs are fully compliant with the International Conference on Harmonisation/Good Clinical Practice (ICH GCP) Guidelines for the Conduct of Trials Involving the Participation of Human Subjects as they relate to the responsibilities, composition, function, operations and records of an Independent Ethics Committee/Independent Review Board. To this end it undertakes to adhere as far as is consistent with its Constitution, to the relevant clauses of the ICH Harmonised Tripartite Guideline for Good Clinical Practice, adopted by the Commission of the European Union on 17 January 1997. The Standing Orders and a Statement of Compliance were included on the computer disk containing the guidelines and application form and are available on request or on the Internet at <http://www.corec.org.uk>

Yours sincerely



**Maureen Thrupp**  
**Administrator, MREC West Midlands**

*Encl: MREC Response Form*

**Please note change of contact details:**

**MREC West Midlands**  
**Birmingham and The Black Country Health Authority**  
**27 Highfield Road**  
**Edgbaston**  
**Birmingham**  
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## Appendix D – University of Essex Ethics Approval



University of Essex

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Human Sciences**

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02 April 2015

MRS ANELIYA MEROLLA  
104 CALABRIA ROAD  
LONDON  
N5 1HT

Dear Aneliya,

**Re: Ethical Approval Application (Ref 14021)**

Further to your application for ethical approval, please find enclosed a copy of your application which has now been approved by Dr Wayne Wilson on behalf of the Faculty Ethics Committee.

Yours sincerely,

Lisa McKee  
Ethics Administrator  
School of Health and Human Sciences

cc. Sarah Manning-Press, REO  
Frances Blumenfeld, supervisor

**Appendix E – Tavistock Adult Depression Study Participant Consent Form**

The Tavistock & Portman  
NHS Trust

**PATIENT CONSENT FORM**

**Title of Project:** Tavistock Adult Depression Study

**Name of Researcher:**

Please initial box

1. I confirm that I have read and understand the Information sheet dated October 2009 (version 3) for the above study and have had the opportunity to ask questions.	
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected.	
3. I have read and understand the information sheet about the recording of my sessions. I understand that these recordings will be used for research and may be used for training purposes. I agree to the recordings of the sessions as part of the Tavistock Adult Depression Study, on condition that they will be treated confidentially as set out in the Patient Information Sheet and in accordance with the Data Protection Act 1998.	
4. I understand that sections of any of my medical notes may be looked at by members of the research team where it is relevant to my taking part in research. I give permission for these individuals to have access to my records and understand that any information extracted will be treated according to the confidentiality guidelines stated above.	
5. I agree to take part in the above study.	

\_\_\_\_\_  
Name of Patient

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of Person taking consent  
(if different from researcher)

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

\_\_\_\_\_  
Name of Researcher

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Date

Psychotherapy Evaluation Research Unit  
The Tavistock & Portman NHS Trust, Tavistock Centre, 120 Belsize Lane, London, NW3 5BA  
Tel: 020 89382038 Fax: 020 7435 8018  
Email: peru@tavi-port.nhs.uk



**Appendix F - TADS Honorary Contract**

The Tavistock and Portman **NHS**  
NHS Foundation Trust

**TAVISTOCK & PORTMAN NHS FOUNDATION TRUST****STATEMENT OF HONORARY ATTACHMENT**

Name: Aneliya Merolla  
Location: Tavistock Clinic  
120 Belsize Lane,  
London NW3 5BA  
Period of attachment: 10<sup>th</sup> March 2014 – 9<sup>th</sup> March 2015  
Details of attachment: Honorary Research Student  
Accountable to: Felicitas Rost

Conditions of AttachmentGeneral

1. This Statement of Honorary Attachment is to be read in conjunction with the Terms and Conditions as Applied to Honorary Contracts.
2. The attachment to the Trust does not constitute employment and there will not be an entitlement to any form of remuneration or other payment from the Trust either during the attachment or on its cessation.
3. For the duration of this honorary agreement you will be accountable to the above named for your activities within the Trust.
4. The Trust does not normally accept responsibility for articles lost or damaged on Trust property

Confidentiality

5. During the course of this attachment any matters of a confidential nature, including in particular but not exclusively information relating to the diagnosis and treatment of patients, individual staff records and details of any contracts entered into by the Trust, must under no circumstances be divulged to any unauthorised person or persons. Any breach of confidentiality will result in the termination of the attachment.

Health and Safety

6. The Trust has an obligation under the Health and Safety at Work Act 1974 to provide safe and healthy working conditions and methods. You are required to co-operate with the Trust to enable it to discharge its responsibilities under the Health and Safety at Work Act 1974 and to take reasonable care for the health and safety of yourself and others. Your attention is drawn to the provisions in the Terms and Conditions as Applied to Honorary Contracts which summarises your duties with regards to health and safety at work.

Conduct

7. You are required to conform for the duration of this attachment with any relevant provisions of the Rehabilitation of Offenders Act 1975 that may apply to you.
8. You are required to comply with such rules regarding conduct and administration as may be prescribed by the Trust from time to time.
9. You are required to comply with the Trust's policies which are available on request from the Human Resources Department.



**TAVISTOCK AND PORTMAN NHS FOUNDATION TRUST****TERMS AND CONDITIONS  
AS APPLIED TO HONORARY CONTRACTS****1. Health and Safety at Work**

The Tavistock and Portman NHS Foundation Trust ('the Trust') attaches the greatest importance to the safety of its honorary contract holders. It is necessary for management, honorary contract holders and staff to work together positively to achieve a situation, compatible with the provision of proper services to patients, where personal injuries and hazards to the health of staff and others can be reduced to a minimum.

It is accepted that it is a management function to do all that is possible in the field of construction, operation and maintenance of buildings, plant, equipment and facilities to achieve such a situation. Honorary contract holders are expected to work in such a way that accidents to themselves and others are avoided; to exercise responsibility; to report accidents or potential hazards promptly; and to recognise that they now have a legal obligation to co-operate with the Trust in all safety, health and welfare matters.

Honorary contract holders must, in their own interest, report any accident or injury, however trivial, arising out of the course of their employment, to the appropriate senior officer and furnish any statement required. Where appropriate, safety training will be provided by the Trust together with necessary safety devices and protective clothing.

**2. Personal Property**

It should be noted that the employing Trust cannot accept responsibility for the loss or damage to personal belongings and honorary contract holders are advised to make arrangements for insurance cover.

**3. Fire Precautions**

Honorary contract holders should familiarise themselves with the routine to be followed in the event of an outbreak of fire within the premises.

**4. Confidentiality and Data Protection**

All honorary contract holders working within this Trust whether on a permanent or temporary basis have a legal duty of confidence to patients. Inappropriate access and use of health records or abuse of computer systems or disclosure of any patient information that may, through the course of employment, be acquired may lead to disciplinary measures and possibly result in legal proceedings. Regardless of any action taken by the Trust, a breach of the Data Protection Act could result in criminal or civil action for damages.

**5. Research Governance**

The Trust manages all research in accordance with the requirements of the Research Governance Framework. As an honorary contract holder of the Trust you must comply with all reporting requirements, systems and duties of action put in place by the Trust to deliver research governance.

**6. 1959 and 1983 Mental Health Acts**

Honorary contract holders whose work brings them into contact with mentally ill or handicapped patients should be familiar with the requirements of the 1959 and 1983 Mental Health Acts, and in particular Sections 126 and 128 of the 1959 Act as amended by Section 1 of the Sexual Offences Act 1967. Further guidance will be provided where applicable.

**7. Services and Products Developed in Employment**

Honorary contract holders must not make Commercial Use of services or products developed wholly or partly whilst working in the Trust without the prior agreement of the Trust.

**8. Equal Opportunities/Statement of Intent**

The Trust is an equal opportunities employer. It is the policy of the Trust to ensure that no user of service, present or future employee, honorary contract holder or job applicant receives less favourable treatment on the grounds of their sex, sexual orientation, marital status, race, religion, age, creed, colour, ethnic origin or disability, nor is disadvantaged by any conditions or requirements which cannot be shown to be justified.

Revised September 2008

Termination

10. This attachment may be terminated on either party giving one week's written notice of termination.
11. The Trust reserves the right to terminate this attachment immediately in the event of your misconduct, poor performance or failure to comply with any of the terms of this statement. The Trust may refuse you access to any of its facilities and require you to leave the premises immediately pending its decision on whether to terminate your attachment.

Absence

12. Any absence which results in an inability to attend for the purpose of the attachment e.g. sickness, must be notified to your immediate superior as soon as possible.

Changes to Statement of Honorary Attachment


13. The Trust reserves the right to amend the terms of your conditions of attachment to the Trust at any time and any such changes will be notified to you in writing either by issue of a revised Statement of Honorary Attachment or a letter of amendment

Name: .....Charles Omoaka.....

Signature: ..........  
(Human Resources)

Date: .....17/3/14.....

I am prepared to accept the attachment on the above conditions.

Signature: ..........

Date: .....17.03.2014.....

**TAVISTOCK & PORTMAN NHS FOUNDATION TRUST****STATEMENT OF HONORARY ATTACHMENT**

Name: Aneliya Merolla

Location: Tavistock Clinic  
120 Belsize Lane,  
London NW3 5BA

Period of attachment: 10<sup>th</sup> March 2015 – 9<sup>th</sup> March 2016

Details of attachment: Honorary Research Student

Accountable to: Felicitas Rost

Conditions of AttachmentGeneral

1. This Statement of Honorary Attachment is to be read in conjunction with the Terms and Conditions as Applied to Honorary Contracts.
2. The attachment to the Trust does not constitute employment and there will not be an entitlement to any form of remuneration or other payment from the Trust either during the attachment or on its cessation.
3. For the duration of this honorary agreement you will be accountable to the above named for your activities within the Trust.
4. The Trust does not normally accept responsibility for articles lost or damaged on Trust property

Confidentiality

5. During the course of this attachment any matters of a confidential nature, including in particular but not exclusively information relating to the diagnosis and treatment of patients, individual staff records and details of any contracts entered into by the Trust, must under no circumstances be divulged to any unauthorised person or persons. Any breach of confidentiality will result in the termination of the attachment.

Health and Safety

6. The Trust has an obligation under the Health and Safety at Work Act 1974 to provide safe and healthy working conditions and methods. You are required to co-operate with the Trust to enable it to discharge its responsibilities under the Health and Safety at Work Act 1974 and to take reasonable care for the health and safety of yourself and others. Your attention is drawn to the provisions in the Terms and Conditions as Applied to Honorary Contracts which summarises your duties with regards to health and safety at work.

Conduct

7. You are required to conform for the duration of this attachment with any relevant provisions of the Rehabilitation of Offenders Act 1975 that may apply to you.
8. You are required to comply with such rules regarding conduct and administration as may be prescribed by the Trust from time to time.
9. You are required to comply with the Trust's policies which are available on request from the Human Resources Department.



Termination

10. This attachment may be terminated on either party giving one week's written notice of termination.
11. The Trust reserves the right to terminate this attachment immediately in the event of your misconduct, poor performance or failure to comply with any of the terms of this statement. The Trust may refuse you access to any of its facilities and require you to leave the premises immediately pending its decision on whether to terminate your attachment.


Absence

12. Any absence which results in an inability to attend for the purpose of the attachment e.g. sickness, must be notified to your immediate superior as soon as possible.

Changes to Statement of Honorary Attachment

13. The Trust reserves the right to amend the terms of your conditions of attachment to the Trust at any time and any such changes will be notified to you in writing either by issue of a revised Statement of Honorary Attachment or a letter of amendment

Name: Charles Omoaka

Signature:   
(Human Resources)

Date: 18.03.15

I am prepared to accept the attachment on the above conditions.

Signature:  .....

Date: 18.03.2015 .....

**Appendix G – The Shedler-Westen Personality Assessment Profile – II (SWAP-II)**

Score Desired	No w	SWAP-II Data Entry
7	8	<b>0</b>
6	10	<b>0</b>
5	12	<b>0</b>
4	14	<b>0</b>
3	16	<b>0</b>
2	18	<b>0</b>
1	22	<b>0</b>
0	100	<b>0</b>
		<b>Instructions</b> <ul style="list-style-type: none"> <li>• Select the <b>ID</b> cell and enter patient identifying information (e.g., patient ID number or initials).</li> <li>• Enter a score for each SWAP item (0=least descriptive, 7=most descriptive). Press the <b>Enter</b> key after each entry.</li> <li>• Click <b>Sort by score</b> to arrange the items in descending order by score (you will do this repeatedly as you work).</li> <li>• The blue table to the left shows the <b>Desired</b> score distribution and the score distribution <b>Now</b>.</li> <li>• Working from 7 to 0, adjust the scores until you have the correct distribution.</li> <li>• When the score distribution is correct, the numbers in the <b>Now</b> column turn green.</li> <li>• When finished, click <b>Save to database</b>.</li> </ul>
<b>ID ►</b>		<b>To obtain an authorized copy of this software or learn more about the SWAP family of assessment instruments, visit <a href="http://www.SWAPassessment.com">www.SWAPassessment.com</a>. Clinical interpretive reports are coming soon!</b>
	<b>Score</b>	
		1. Tends to feel guilty (e.g., may blame self or feel responsible for bad things that happen).
		2. Is able to use his/her talents, abilities, and energy effectively and productively.
		3. Takes advantage of others; has little investment in moral values (e.g., puts own needs first, uses or exploits people with little regard for their feelings or welfare, etc.).
		4. Has an exaggerated sense of self-importance (e.g., feels special, superior, grand, or envied).
		5. Tends to be emotionally intrusive (e.g., may not respect other people's needs for autonomy, privacy, etc.).
		6. Is troubled by recurrent obsessional thoughts that s/he experiences as intrusive.
		7. Appears conflicted about his/her racial or ethnic identity (e.g., undervalues and rejects, or overvalues and is preoccupied with, own cultural heritage).
		8. Tends to get into power struggles.
		9. When upset, has trouble perceiving both positive and negative qualities in the same person at the same time (e.g., may see others in black or white terms, shift suddenly from seeing someone as caring to seeing him/her as malevolent and intentionally hurtful, etc.).
		10. Believes that some important other has a special, seemingly magical ability to know his/her innermost thoughts or feelings (e.g., imagines rapport is so perfect that ordinary communication is superfluous).
		11. Tends to become attached quickly or intensely; develops feelings, expectations, etc. that are not warranted by the history or context of the

	relationship.
	12. Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.
	13. Tends to use his/her psychological or medical problems to avoid work or responsibility (whether consciously or unconsciously).
	14. Tends to blame own failures or shortcomings on other people or circumstances; attributes his/her difficulties to external factors rather than accepting responsibility for own conduct or choices.
	15. Lacks a stable sense of who s/he is (e.g., attitudes, values, goals, and feelings about self seem unstable or ever-changing).
	16. Tends to be angry or hostile (whether consciously or unconsciously).
	17. Tends to be ingratiating or submissive (e.g., consents to things s/he does not want to do, in the hope of getting support or approval).
	18. Tends to stir up conflict or animosity between other people (e.g., may portray a situation differently to different people, leading them to form contradictory views or work at cross purposes).
	19. Enjoys challenges; takes pleasure in accomplishing things.
	20. Tends to be deceitful; tends to lie or mislead.
	21. Tends to be hostile toward members of the opposite sex, whether consciously or unconsciously (e.g., may be disparaging or competitive).
	22. Tends to develop somatic symptoms in response to stress or conflict (e.g., headache, backache, abdominal pain, asthma, etc.).
	23. Tends to become involved in romantic or sexual “triangles” (e.g., is drawn to people who are already attached, sought by someone else, etc.).
	24. Tends to be unreliable and irresponsible (e.g., may fail to meet work obligations or honor financial commitments).
	25. Has difficulty acknowledging or expressing anger.
	26. Tends to get drawn into or remain in relationships in which s/he is emotionally or physically abused, or needlessly puts self in dangerous situations (e.g., walking alone or agreeing to meet strangers in unsafe places).
	27. Has panic attacks (i.e., episodes of acute anxiety accompanied by strong physiological responses).
	28. Tends to be preoccupied with concerns about dirt, cleanliness, contamination, etc. (e.g., drinking from another person’s glass, sitting on public toilet seats, etc.).
	29. Has difficulty making sense of other people’s behavior; tends to misunderstand, misinterpret, or be confused by others’ actions and reactions.
	30. Tends to feel listless, fatigued, or lacking in energy.
	31. Tends to show reckless disregard for the rights, property, or safety of others.
	32. Is capable of sustaining meaningful relationships characterized by genuine intimacy and caring.
	33. Is conflicted or inhibited about achievement or success (e.g., achievements may be below potential, may sabotage self just before attaining important goals, etc.).

	34. Tends to be sexually seductive or provocative (e.g., may be inappropriately flirtatious, preoccupied with sexual conquest, prone to “lead people on,” etc.).
	35. Tends to feel anxious.
	36. Tends to feel helpless, powerless, or at the mercy of forces outside his/her control.
	37. Finds meaning in belonging and contributing to a larger community (e.g., organization, neighborhood, church).
	38. Tends to feel s/he is not his/her true self with others; may feel false or fraudulent.
	39. Appears to gain pleasure or satisfaction by being sadistic or aggressive toward others (whether consciously or unconsciously).
	40. Tends to engage in unlawful or criminal behavior.
	41. Appears unable to describe important others in a way that conveys a sense of who they are as people; descriptions of others come across as two-dimensional and lacking in richness.
	42. Tends to feel envious.
	43. Tends to seek power or influence over others (whether in beneficial or destructive ways).
	44. When distressed, perception of reality can become grossly impaired (e.g., thinking may seem delusional).
	45. Is prone to idealizing people; may see admired others as perfect, larger than life, all wise, etc.
	46. Tends to be suggestible or easily influenced.
	47. Attempts to avoid or flee depressive feelings through excessive optimism, activity, energy, etc.
	48. Seeks to be the center of attention.
	49. Has fantasies of unlimited success, power, beauty, talent, brilliance, etc.
	50. Tends to feel life has no meaning.
	51. Tends to be liked by other people.
	52. Has little empathy; seems unable or unwilling to understand or respond to others’ needs or feelings.
	53. Seems to treat others primarily as an audience to witness own importance, brilliance, beauty, etc.
	54. Tends to feel s/he is inadequate, inferior, or a failure.
	55. Finds meaning and fulfillment in guiding, mentoring, or nurturing others.
	56. Appears to find little or no pleasure, satisfaction, or enjoyment in life’s activities.
	57. Religious or spiritual beliefs are central to his/her identity and experience.
	58. Has little or no interest in sex.
	59. Is empathic; is sensitive and responsive to other peoples’ needs and feelings.
	60. Tends to be shy or self-conscious in social situations.
	61. Tends to disparage qualities traditionally associated with own gender (e.g., a woman who disdains nurturance and overvalues power; a man who disdains power and overvalues emotional sensitivity).



	62. Tends to be preoccupied with food, diet, or eating.
	63. Is able to assert him/herself effectively and appropriately when necessary.
	64. Mood tends to cycle over intervals of weeks or months between excited and depressed states (high placement implies bipolar mood disorder).
	65. Attempts to dominate a significant other (e.g., spouse, lover, family member) through violence or intimidation.
	66. Is excessively devoted to work and productivity to the detriment of leisure and relationships.
	67. Tends to be stingy and withholding (e.g., of time, money, affection, ideas).
	68. Has a good sense of humor.
	69. Decisions and actions are unduly influenced by efforts to avoid perceived dangers; is more concerned with avoiding harm than pursuing desires.
	70. Has uncontrolled eating binges followed by “purges” (e.g., makes self vomit, abuses laxatives, fasts, etc.); has bulimic episodes.
	71. Tends to seek thrills, novelty, excitement, etc.; appears to require a high level of stimulation.
	72. Tends to perceive things in global and impressionistic ways (e.g., misses details, glosses over inconsistencies, mispronounces names).
	73. Tends to “catastrophize”; is prone to see problems as disastrous, unsolvable, etc.
	74. Expresses emotion in exaggerated and theatrical ways.
	75. Tends to think in concrete terms and interpret things in overly literal ways; has limited ability to appreciate metaphor, analogy, or nuance.
	76. Manages to elicit in others feelings similar to those s/he is experiencing (e.g., when angry, acts in such a way as to provoke anger in others; when anxious, acts in such a way as to induce anxiety in others).
	77. Tends to be needy or dependent.
	78. Tends to express anger in passive and indirect ways (e.g., may make mistakes, procrastinate, forget, become sulky, etc.).
	79. Attempts to deny or “override” fear or anxiety by rushing headlong into feared situations, taking unnecessary risks, etc.
	80. Tends to be sexually possessive or jealous; is preoccupied with concerns about real or imagined infidelity.
	81. Repeatedly re-experiences or re-lives a past traumatic event (e.g., has intrusive memories or recurring dreams of the event; is startled or terrified by present events that resemble or symbolize the past event).
	82. Is capable of hearing information that is emotionally threatening (i.e., that challenges cherished beliefs, perceptions, and self-perceptions) and can use and benefit from it.
	83. Beliefs and expectations seem cliché or stereotypical, as if taken from storybooks or movies.
	84. Tends to be competitive with others (whether consciously or unconsciously).
	85. Sexual orientation is central to his/her identity or sense of self.
	86. Tends to feel ashamed or embarrassed.

	87. Sense of identity revolves around a “cause,” movement, or label (e.g., adult child of alcoholic, adult survivor, environmentalist, born-again Christian, etc.); may be drawn to extreme or all-encompassing belief systems.
	88. Tends to be insufficiently concerned with meeting own needs; appears not to feel entitled to get or ask for things s/he deserves.
	89. Appears to have come to terms with painful experiences from the past; has found meaning in, and grown from such experiences.
	90. Is prone to painful feelings of emptiness (e.g., may feel lost, bereft, abjectly alone even in the presence of others, etc.).
	91. Is self-critical; sets unrealistically high standards for self and is intolerant of own human defects.
	92. Is articulate; can express self well in words.
	93. Seems naïve or innocent; appears to know less about the ways of the world than might be expected given his/her intelligence or background.
	94. Has an active and satisfying sex life.
	95. Appears comfortable and at ease in social situations.
	96. Tends to elicit dislike or animosity in others.
	97. Tends to use his/her physical attractiveness to an excessive degree to gain attention or notice.
	98. Tends to fear s/he will be rejected or abandoned.
	99. Appears to associate sex with danger (e.g., injury, punishment, contamination), whether consciously or unconsciously.
	100. Tends to think in abstract and intellectualized terms, even in matters of personal import.
	101. Generally finds contentment and happiness in life’s activities.
	102. Has a deep sense of inner badness; sees self as damaged, evil, or rotten to the core (whether consciously or unconsciously).
	103. Tends to have extreme reactions to perceived slights or criticism (e.g., may react with rage, humiliation, etc.).
	104. Appears to have little need for human company or contact; is emotionally detached or indifferent.
	105. Is suspicious; tends to assume others will harm, deceive, conspire against, or betray him/her.
	106. Tends to express emotion appropriate in quality and intensity to the situation at hand.
	107. Tends to express qualities or mannerisms traditionally associated with own gender to an exaggerated or stereotypical degree (i.e., a hyper-feminine woman; a hyper-masculine, “macho” man).
	108. Tends to restrict food intake to the point of being underweight and malnourished.
	109. Tends to engage in self-mutilating behavior (e.g., self-cutting, self-burning, etc.).
	110. Tends to become attached to, or romantically interested in, people who are emotionally unavailable.
	111. Has the capacity to recognize alternative viewpoints, even in matters that stir up strong feelings.

	112. Appears impervious to consequences; seems unable or unwilling to modify behavior in response to threats or negative consequences.
	113. Experiences little or no remorse for harm or injury caused to others.
	114. Tends to be critical of others.
	115. Is prone to violence (e.g., may break things or become physically assaultive).
	116. Tends to see own unacceptable feelings or impulses in other people instead of in him/herself.
	117. Is unable to soothe or comfort him/herself without the help of another person (i.e., has difficulty regulating own emotions).
	118. Has difficulty maintaining attention and focus on tasks; is easily distracted by sights, sounds, unrelated thoughts, or other competing stimuli.
	119. Tends to be inhibited or constricted; has difficulty allowing self to acknowledge or express wishes and impulses.
	120. Has moral and ethical standards and strives to live up to them.
	121. Is creative; is able to see things or approach problems in novel ways.
	122. Attempts to avoid feeling helpless or depressed by becoming angry instead.
	123. Tends to adhere rigidly to daily routines and become anxious or uncomfortable when they are altered.
	124. Tends to avoid social situations because of fear of embarrassment or humiliation.
	125. Appearance or manner seems odd or peculiar (e.g., grooming, hygiene, posture, eye contact, speech rhythms, etc. seem somehow strange or “off”).
	126. Appears to have a limited or constricted range of emotions.
	127. Tends to feel misunderstood, mistreated, or victimized.
	128. Fantasizes about ideal, perfect love.
	129. Tends to be conflicted about authority (e.g., may feel s/he must submit, rebel against, win over, defeat, etc.).
	130. Reasoning processes or perceptual experiences seem odd and idiosyncratic (e.g., may make seemingly arbitrary inferences; may see hidden messages or special meanings in ordinary events).
	131. Appears conflicted about experiencing pleasurable emotions; tends to inhibit excitement, joy, pride, etc.
	132. Tends to have numerous sexual involvements; is promiscuous.
	133. Tends to be dismissive, haughty, or arrogant.
	134. Tends to act impulsively (e.g., acts without forethought or concern for consequences).
	135. Is hypochondriacal; has exaggerated fears of contracting medical illness (e.g., worries excessively about normal aches and pains).
	136. Tends to believe in supernatural, paranormal, or superstitious phenomena or to be drawn to “alternative” belief systems (e.g., astrology, tarot, crystals, psychics, auras).
	137. Is confused, conflicted, or uncertain about his/her sexual orientation (e.g., may struggle to keep homosexual feelings out of awareness, have an exaggerated fear of homosexuality, etc.).
	138. Tends to enter altered, dissociated states when distressed (e.g., the self or world feels strange, unreal, or unfamiliar).

	139. Tends to hold grudges; may dwell on insults or slights for long periods.
	140. Sexual fantasies or activities are unusual, idiosyncratic, or rigidly scripted (e.g., dominance, submission, voyeurism, fetishes, etc.).
	141. Is invested in seeing and portraying self as emotionally strong, untroubled, and emotionally in control, despite clear evidence of underlying insecurity, anxiety, or distress.
	142. Tends to make repeated suicidal threats or gestures, either as a “cry for help” or as an effort to manipulate others.
	143. Tends to believe s/he can only be appreciated by, or should only associate with, people who are high-status, superior, or otherwise “special.”
	144. Tends to see self as logical and rational, uninfluenced by emotion; prefers to operate as if emotions were irrelevant or inconsequential.
	145. Thought processes or speech tend to be circumstantial, vague, rambling, digressive, etc. (e.g., may be unclear whether s/he is being metaphorical or whether thinking is confused or peculiar).
	146. Tends to elicit boredom in others (e.g., may talk incessantly, without feeling, or about inconsequential matters).
	147. Tends to abuse drugs or alcohol.
	148. Has little psychological insight into own motives, behavior, etc.
	149. Tends to feel like an outcast or outsider.
	150. Tends to identify with admired others to an exaggerated degree, taking on their attitudes, mannerisms, etc. (e.g., may be drawn into the “orbit” of a strong or charismatic personality).
	151. Appears to experience the past as a series of disjointed or disconnected events; has difficulty giving a coherent account of his/her life story.
	152. Tends to repress or “forget” distressing events, or distort memories of distressing events beyond recognition.
	153. Relationships tend to be unstable, chaotic, and rapidly changing.
	154. Tends to draw others into scenarios, or “pull” them into roles, that feel alien or unfamiliar (e.g., being uncharacteristically insensitive or cruel, feeling like the only person in the world who can help, etc.).
	155. Tends to describe experiences in generalities; is reluctant to provide details, examples, or supporting narrative.
	156. Has a disturbed or distorted body-image (e.g., may see self as unattractive, grotesque, disgusting, etc.).
	157. Tends to become irrational when strong emotions are stirred up; may show a significant decline from customary level of functioning.
	158. Appears afraid of commitment to a long-term love relationship.
	159. Tends to deny or disavow own need for nurturance, caring, comfort, etc. (e.g., may regard such needs as weakness, avoid depending on others or asking for help, etc.)
	160. Lacks close friendships and relationships.
	161. Tends to deny, disavow, or squelch his/her own realistic hopes, dreams, or desires to protect against anticipated disappointment (whether consciously or unconsciously).
	162. Expresses contradictory feelings or beliefs without being disturbed by the inconsistency; has little need to reconcile or resolve contradictory ideas.

	163. Appears to want to “punish” self; creates situations that lead to unhappiness, or actively avoids opportunities for pleasure and gratification.
	164. Tends to be self-righteous or moralistic.
	165. Tends to distort unacceptable wishes or feelings by transforming them into their opposite (e.g., may express excessive concern while showing signs of unacknowledged hostility, disgust about sexual matters while showing signs of unacknowledged excitement, etc.).
	166. Tends to alternate between undercontrol and overcontrol of needs and impulses (e.g., sometimes acts on desires impulsively while at other times denying them entirely).
	167. Is simultaneously needy of, and rejecting toward, others (e.g., craves intimacy and caring, but tends to reject it when offered).
	168. Struggles with genuine wishes to kill him/herself.
	169. Is afraid or conflicted about becoming like a parent (or parent figure) about whom s/he has strong negative feelings (e.g., may go to lengths to avoid or reject attitudes or behaviors associated with that person).
	170. Tends to be oppositional, contrary, or quick to disagree.
	171. Appears to fear being alone; may go to great lengths to avoid being alone.
	172. Seems unable to settle into, or sustain commitment to, identity-defining life roles (e.g., career, occupation, lifestyle, etc.).
	173. Tends to become absorbed in details, often to the point that s/he misses what is significant.
	174. Expects self to be “perfect” (e.g., in appearance, achievements, performance, etc.).
	175. Tends to be conscientious and responsible.
	176. Tends to confuse own thoughts, feelings, or personality traits with those of others (e.g., may use the same words to describe him/herself and another person, believe the two share identical thoughts and feelings, etc.).
	177. Repeatedly convinces others of his/her commitment to change but then reverts to previous maladaptive behavior; tends to convince others that “this time is really different.”
	178. Has a pervasive sense that someone or something necessary for happiness has been lost forever, whether consciously or unconsciously (e.g., a relationship, youth, beauty, success).
	179. Tends to be energetic and outgoing.
	180. Has trouble making decisions; tends to be indecisive or to vacillate when faced with choices.
	181. Tends to choose sexual or romantic partners who seem inappropriate in terms of age, status (e.g., social, economic, intellectual), etc.
	182. Tends to be controlling.
	183. Is psychologically insightful; is able to understand self and others in subtle and sophisticated ways.
	184. Verbal statements seem incongruous with accompanying affect, or incongruous with accompanying non-verbal messages.
	185. Is prone to intense anger, out of proportion to the situation at hand (e.g., has rage episodes).

	186. Has difficulty directing both tender feelings and sexual feelings toward the same person (e.g., sees others as nurturing and virtuous or sexy and exciting, but not both).
	187. Tends to feel guilty or ashamed about his/her sexual interests or activities (whether consciously or unconsciously).
	188. Work-life and/or living arrangements tend to be chaotic or unstable (e.g., job or housing situation seems always temporary, transitional, or ill-defined).
	189. Tends to feel unhappy, depressed, or despondent.
	190. Appears to feel privileged and entitled; expects preferential treatment.
	191. Emotions tend to change rapidly and unpredictably.
	192. Tends to be overly concerned with rules, procedures, order, organization, schedules, etc.
	193. Lacks social skills; tends to be socially awkward or inappropriate.
	194. Tends to be manipulative.
	195. Tends to be preoccupied with death and dying.
	196. Finds meaning and satisfaction in the pursuit of long-term goals and ambitions.
	197. Tends to seek out or create interpersonal relationships in which s/he is in the role of caring for, rescuing, or protecting the other.
	198. Has trouble acknowledging or expressing anger toward others, and instead becomes depressed, self-critical, self-punitive, etc. (i.e., turns anger against self).
	199. Tends to be passive and unassertive.
	200. Tends to ruminate; may dwell on problems, replay conversations in his/her mind, become preoccupied with thoughts about what could have been, etc.

**Appendix H – The SWAP –II Personality Health Syndrome (adapted from Westen et al., 2012)**

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**Personality Health**

*This prototype represents optimal personality health. Degree of match with this prototype provides a measure of adaptive psychological strengths. The more individuals match this prototype, the more they are able to engage in meaningful and mature relationships, find meaning and satisfaction in life's pursuits, and make effective use of their talents and abilities.*

Individuals who match this prototype are capable of sustaining meaningful relationships characterized by genuine intimacy and caring. They are empathic and responsive to others' needs and feelings and have the capacity to recognize alternative viewpoints, even when emotions are strong. They have moral and ethical standards, strive to live up to them, and tend to be conscientious and responsible. They appear comfortable in social situations, are able to assert themselves effectively and appropriately when necessary, tend to be energetic and outgoing, and tend to be liked by others. They tend to have satisfying sex lives. They are psychologically insightful and able to understand themselves and others in nuanced ways. They are capable of hearing and making effective use of information that is emotionally threatening, and have generally come to terms with painful experiences from the past, finding meaning in the experiences and growing from them. Individuals who match this prototype tend to express emotion appropriate in quality and intensity to the situation at hand. They generally find contentment and happiness in life's activities. They find meaning and fulfillment in guiding or nurturing others, in belonging and contributing to a larger community, and in the pursuit of long-term goals and ambitions. Individuals who match this prototype are able to use their talents, abilities, and energy effectively and productively. They enjoy challenges and take pleasure in accomplishing things. They are able to express themselves verbally, have a sense of humor, and tend to see things and approach problems in creative ways.

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Appendix I - Hamilton Depression Rating Scale (HDRS)

ASSESSMENT OF DEPRESSION																				
Item No.	Score Range	Symptom	Score																	
1	0-4	Depressed mood	<table><tr><th colspan="2">Grading</th></tr><tr><td>0</td><td>Absent</td></tr><tr><td>1</td><td>Mild or trivial</td></tr><tr><td>2</td><td rowspan="2">Moderate</td></tr><tr><td>3</td></tr><tr><td>4</td><td>Severe</td></tr><tr><td>0</td><td>Absent</td></tr><tr><td>1</td><td>Slight or doubtful</td></tr><tr><td>2</td><td>Clearly present</td></tr></table>	Grading		0	Absent	1	Mild or trivial	2	Moderate	3	4	Severe	0	Absent	1	Slight or doubtful	2	Clearly present
Grading																				
0	Absent																			
1	Mild or trivial																			
2	Moderate																			
3																				
4	Severe																			
0	Absent																			
1	Slight or doubtful																			
2	Clearly present																			
2	0-4	Guilt																		
3	0-4	Suicide																		
4	0-2	Insomnia, initial																		
5	0-2	„ middle																		
6	0-2	„ delayed																		
7	0-4	Work and interests																		
8	0-4	Retardation																		
9	0-2	Agitation																		
10	0-4	Anxiety, psychic																		
11	0-4	„ somatic																		
12	0-2	Somatic symptoms, gastrointestinal																		
13	0-2	„ „ general																		
14	0-2	Genital symptoms																		
15	0-4	Hypochondriasis																		
16	0-2	Loss of insight																		
17	0-2	„ „ weight																		
18	0-2	Diurnal variation { M E																		
19	0-4	Depersonalization, etc.																		
20	0-4	Paranoid symptoms																		
21	0-2	Obsessional symptoms																		



**Appendix J - Hamilton Depression Rating Scale (HDRS) – TADS Scoring Sheet****Hamilton Rating Scale for Depression****Scoring Sheet****Patient ID:****Date:****Rater:**

Please cross (X) appropriate box

<b><u>SYMPTOM</u></b>		<b><u>SCORE</u></b>				
		<b><u>0</u></b>	<b><u>1</u></b>	<b><u>2</u></b>	<b><u>3</u></b>	<b><u>4</u></b>
1. Depressed Mood	R					
2. Guilt	C					
3. Suicide	C					
4. Insomnia, initial	S					
5. Insomnia, middle	S					
6. Insomnia, delayed	S					
7. Work & Interests	R					
8. Retardation	R					
9. Agitation	C					
10. Anxiety, psychic	A					
11. Anxiety, somatic	A					
12. Somatic Symptoms, gastrointest.	A					
13. Somatic Symptoms, general	A					
14. Genital Symptoms	R					
15. Hypochondriasis	A					
16. Loss of Weight	L					
17. Insight	A					
18. Diurnal Variation	D					
19. Depersonalization / Derealization	C					
20. Paranoid Symptoms	C					
21. Obsessional Symptoms	C					

Total	
Anx	
Loss weight	
Diurnal	
Cognitive	
Retardation	
Sleep	

**Depression Free Days:**



Over the last week	Not at all	Only occasionally	Sometimes	Often	Most or all the time	OFFICE USE ONLY
15 I have felt panic or terror	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> P
16 I made plans to end my life	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> R
17 I have felt overwhelmed by my problems	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> W
18 I have had difficulty getting to sleep or staying asleep	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> P
19 I have felt warmth or affection for someone	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/>
20 My problems have been impossible to put to one side	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> P
21 I have been able to do most things I needed to	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> F
22 I have threatened or intimidated another person	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> R
23 I have felt despairing or hopeless	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> P
24 I have thought it would be better if I were dead	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> R
25 I have felt criticised by other people	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> F
26 I have thought I have no friends	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> F
27 I have felt unhappy	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> P
28 Unwanted images or memories have been distressing me	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> P
29 I have been irritable when with other people	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> F
30 I have thought I am to blame for my problems and difficulties	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> P
31 I have felt optimistic about my future	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> W
32 I have achieved the things I wanted to	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> F
33 I have felt humiliated or shamed by other people	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> F
34 I have hurt myself physically or taken dangerous risks with my health	<input type="checkbox"/> 0	<input type="checkbox"/> 1	<input type="checkbox"/> 2	<input type="checkbox"/> 3	<input type="checkbox"/> 4	<input type="checkbox"/> R

THANK YOU FOR YOUR TIME IN COMPLETING THIS QUESTIONNAIRE

Total Scores	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>	→	<input type="text"/>	→	<input type="text"/>
Mean Scores	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>		<input type="text"/>		<input type="text"/>
(Total score for each dimension divided by number of items completed in that dimension)	(W)	(P)	(F)	(R)		All items		All minus R

**Appendix L – Global Assessment of Functioning (GAF)****Global Assessment of Functioning (GAF) Scale**

(From DSM-IV-TR, p. 34.)

Consider psychological, social, and occupational functioning on a hypothetical continuum of mental health-illness. Do not include impairment in functioning due to physical (or environmental) limitations.

Code	(Note: Use intermediate codes when appropriate, e.g., 45, 68, 72.)
100   91	Superior functioning in a wide range of activities, life's problems never seem to get out of hand, is sought out by others because of his or her many positive qualities. No symptoms.
90   81	Absent or minimal symptoms (e.g., mild anxiety before an exam), good functioning in all areas, interested and involved in a wide range of activities, socially effective, generally satisfied with life, no more than everyday problems or concerns (e.g. an occasional argument with family members).
80   71	If symptoms are present, they are transient and expectable reactions to psychosocial stressors (e.g., difficulty concentrating after family argument); no more than slight impairment in social, occupational or school functioning (e.g., temporarily failing behind in schoolwork).
70   61	Some mild symptoms (e.g. depressed mood and mild insomnia) OR some difficulty in social, occupational, or school functioning (e.g., occasional truancy, or theft within the household), but generally functioning pretty well, has some meaningful interpersonal relationships.
60   51	Moderate symptoms (e.g., flat affect and circumstantial speech, occasional panic attacks) OR moderate difficulty in social, occupational, or school functioning (e.g., few friends, conflicts with peers or co-workers).
50   41	Serious symptoms (e.g., suicidal ideation, severe obsessional rituals, frequent shoplifting) OR any serious impairment in social, occupational, or school functioning (e.g., no friends, unable to keep a job).
40   31	Some impairment in reality testing or communication (e.g., speech is at times illogical, obscure, or irrelevant) OR major impairment in several areas, such as work or school, family relations, judgment, thinking, or mood (e.g., depressed man avoids friends, neglects family, and is unable to work; child frequently beats up younger children, is defiant at home, and is failing at school).
30   21	Behavior is considerably influenced by delusions or hallucinations OR serious impairment in communication or judgment (e.g., sometimes incoherent, acts grossly inappropriately, suicidal preoccupation) OR inability to function in almost all areas (e.g., stays in bed all day; no job, home, or friends).
20   11	Some danger of hurting self or others (e.g., suicide attempts without clear expectation of death; frequently violent; manic excitement) OR occasionally fails to maintain minimal personal hygiene (e.g., smears feces) OR gross impairment in communication (e.g., largely incoherent or mute).
10   1	Persistent danger of severely hurting self or others (e.g., recurrent violence) OR persistent inability to maintain minimal personal hygiene OR serious suicidal act with clear expectation of death.
0	Inadequate information.

**Appendix Ma. SWAP-II Correlations at baseline**

<b>Patient ID</b>	<b>ICC</b>	<b>ICC Correction</b>	<b>Correlation</b>	<b>Correlation correction</b>	<b>Rates used</b>
102	0.83	0.91	0.71	0.83	
106	0.83	0.91	0.71	0.83	
108	0.69	0.82	0.53	0.69	
109	0.97	0.98	0.94	0.97	
117	0.76	0.87	0.62	0.76	
119	0.76	0.86	0.61	0.76	
126	0.61	0.76	0.44	0.61	
128	0.73	0.84	0.58	0.73	
130	0.64	0.78	0.47	0.64	
133	0.93	0.96	0.87	0.93	
134	0.79	0.88	0.65	0.79	
140	0.59	0.74	0.42	0.59	
154	0.79	0.88	0.65	0.79	
155	0.85	0.92	0.74	0.85	
157	0.74	0.85	0.59	0.74	
159	0.80	0.89	0.67	0.80	
161	0.56	0.72	0.39	0.56	
164	0.74	0.85	0.58	0.73	
169	0.73	0.84	0.58	0.73	
171	0.74	0.85	0.59	0.74	
172	0.50	0.67	0.34	0.50	R3 & R1
176	0.76	0.86	0.62	0.76	
180	0.75	0.86	0.60	0.75	
183	0.59	0.74	0.42	0.59	
184	0.59	0.74	0.42	0.59	
188	0.63	0.78	0.46	0.63	
194	0.64	0.78	0.47	0.64	
199	0.66	0.79	0.49	0.66	
201	0.61	0.76	0.44	0.61	
208	0.58	0.73	0.41	0.58	R3 & R1
209	0.70	0.82	0.53	0.69	
217	0.61	0.76	0.44	0.61	R3 & R1
223	0.54	0.70	0.37	0.54	
237	0.78	0.88	0.65	0.78	
251	0.72	0.84	0.57	0.72	
255	0.84	0.92	0.73	0.84	
261	0.56	0.71	0.39	0.56	
265	0.96	0.98	0.93	0.96	
266	0.68	0.81	0.51	0.68	
272	0.73	0.84	0.57	0.73	
276	0.83	0.91	0.71	0.83	
289	0.72	0.84	0.56	0.72	R3 & R2

291	0.76	0.86	0.61	0.76	
296	0.60	0.75	0.43	0.60	
299	0.73	0.84	0.57	0.73	
301	0.78	0.88	0.64	0.78	
302	0.71	0.83	0.55	0.71	
308	0.77	0.87	0.63	0.77	
315	0.70	0.82	0.54	0.70	
321	0.62	0.76	0.45	0.62	
323	0.79	0.88	0.65	0.79	
324	0.75	0.86	0.60	0.75	
326	0.77	0.87	0.63	0.77	R3 & R2
329	0.71	0.83	0.55	0.71	R3 & R1
334	0.61	0.76	0.44	0.61	
345	0.70	0.82	0.53	0.69	
351	0.66	0.79	0.49	0.66	
354	0.74	0.85	0.59	0.74	
367	0.73	0.85	0.58	0.73	
374	0.56	0.72	0.39	0.56	
380	0.68	0.81	0.51	0.68	
402	0.70	0.82	0.53	0.70	
407	0.80	0.89	0.67	0.80	
<b>Average</b>	<b>0.80</b>	<b>0.89</b>	<b>0.67</b>	<b>0.80</b>	

**Appendix Mb. SWAP-II Correlations at the end of treatment**

<b>Patient ID</b>	<b>ICC</b>	<b>ICC Correction</b>	<b>Correlation</b>	<b>Correlation correction</b>	<b>Rates used</b>
119	0.884	0.792	0.938	0.884	
180	0.806	0.674	0.893	0.805	
343	0.803	0.671	0.891	0.803	
147	0.802	0.699	0.890	0.823	
218	0.792	0.655	0.884	0.792	
301	0.786	0.648	0.880	0.786	
308	0.786	0.648	0.880	0.786	
325	0.786	0.648	0.880	0.786	
350	0.784	0.645	0.879	0.784	R2 & R3
159	0.778	0.636	0.875	0.778	
181	0.767	0.622	0.868	0.767	
265	0.766	0.621	0.867	0.766	
131	0.765	0.619	0.867	0.765	
161	0.763	0.687	0.866	0.814	
188	0.755	0.606	0.860	0.755	
209	0.755	0.606	0.860	0.755	
262	0.754	0.605	0.860	0.754	
402	0.754	0.605	0.860	0.754	
109	0.753	0.604	0.859	0.753	
143	0.753	0.603	0.859	0.752	
348	0.75	0.6	0.857	0.750	
389	0.749	0.599	0.856	0.749	
242	0.746	0.595	0.855	0.746	
282	0.742	0.59	0.852	0.742	
317	0.738	0.584	0.849	0.737	
133	0.737	0.583	0.849	0.737	
126	0.736	0.582	0.848	0.736	
314	0.735	0.581	0.847	0.735	
155	0.726	0.57	0.841	0.726	
128	0.725	0.568	0.841	0.724	
127	0.724	0.567	0.840	0.724	
199	0.724	0.567	0.840	0.724	R2 & R3
208	0.713	0.553	0.832	0.712	
169	0.709	0.549	0.830	0.709	
326	0.701	0.54	0.824	0.701	R2 & R3
102	0.698	0.536	0.822	0.698	
104	0.697	0.535	0.821	0.697	
223	0.697	0.534	0.821	0.696	
274	0.697	0.535	0.821	0.697	
340	0.682	0.517	0.811	0.682	
352	0.679	0.514	0.809	0.679	
175	0.669	0.503	0.802	0.669	
217	0.669	0.503	0.802	0.669	

154	0.666	0.559	0.800	0.717	
195	0.664	0.497	0.798	0.664	R2 & R3
331	0.662	0.495	0.797	0.662	
172	0.655	0.487	0.792	0.655	
407	0.653	0.485	0.790	0.653	
353	0.638	0.469	0.779	0.639	
114	0.632	0.462	0.775	0.632	R1 & R3
184	0.628	0.458	0.771	0.628	
259	0.626	0.456	0.770	0.626	
140	0.624	0.454	0.768	0.624	R1 & R3
289	0.618	0.447	0.764	0.618	
164	0.612	0.441	0.759	0.612	R2 & R3
241	0.611	0.44	0.759	0.611	
384	0.61	0.439	0.758	0.610	R1 & R3
108	0.609	0.438	0.757	0.609	
203	0.602	0.43	0.752	0.601	
600	0.59	0.42	0.742	0.592	
380	0.574	0.403	0.729	0.574	R1 & R2
227	0.573	0.402	0.729	0.573	R2 & R3
251	0.571	0.4	0.727	0.571	R1 & R2
<b>Average</b>	<b>0.706</b>	<b>0.552</b>	<b>0.825</b>	<b>0.708</b>	



**Appendix N – ICC and inter-rater correlations for the borderline-dysregulated items**

SWAP-II item number	Correlation coefficient	Correlation correction	ICC	ICC correction
9	0.132	0.23	0.227	0.37
11	0.518	0.68	0.588*	0.74
12	0.537	0.70	0.698*	0.82
15	-0.027	-0.06	-0.054	-0.11
18	0.615	0.76	0.752**	0.86
73	-0.197	-0.49	-0.445	-1.60
76	-0.255	-0.68	-0.486	-1.89
77	0.376	0.55	0.543*	0.70
90	0.208	0.34	0.344	0.51
98	0.312	0.48	0.468*	0.64
109	1	1.00	0	0.00
117	-0.002	0.00	-0.003	-0.01
127	0.432	0.60	0.601*	0.75
134	0.02	0.04	0.038	0.07
142	CANNOT BE COMPUTED		0 variance	0 variance
153	0.325	0.49	0.487*	0.66
154	-0.149	-0.35	-0.291	-0.82
157	0.282	0.44	0.427*	0.60
168	0.38	0.55	0.548*	0.71
171	0.008	0.02	0.016	0.03
185	0.296	0.46	0.438*	0.61
188	0.836	0.91	0.91**	0.95
191	0.49	0.66	0.63	0.77

\* Coefficient indicating “fair” inter-rater agreement; \*\* Coefficient indicating “excellent” inter-rater agreement

**Appendix P. SWAP-II baseline independent T-test results**

Group Statistics					
	Allocation group	N	Mean	Std. Deviation	Std. Error Mean
SWAP-II Depressive Qfactor prototype BL	TAU	62	61.87	6.615	.840
	Treatment	66	62.47	6.090	.750
SWAP-II Anxious-Avoidant Qfactor prototype BL	TAU	62	57.53	6.498	.825
	Treatment	66	54.98	6.865	.845
SWAP-II Dependent-Victimized Qfactor prototype BL	TAU	62	51.13	6.669	.847
	Treatment	66	53.71	6.516	.802
SWAP-II Schizoid-Schizotypal Qfactor prototype BL	TAU	62	58.26	6.324	.803
	Treatment	66	57.76	6.448	.794
SWAP-II Antisocial-Psychopathic Qfactor prototype BL	TAU	62	47.27	5.701	.724
	Treatment	66	47.67	5.330	.656
SWAP-II Paranoid Qfactor prototype BL	TAU	62	51.32	6.233	.792
	Treatment	66	50.83	6.025	.742
SWAP-II Narcissistic Qfactor prototype BL	TAU	62	46.58	7.482	.950
	Treatment	66	49.71	6.809	.838
SWAP-II Borderline-Dysregulated Qfactor prototype BL	TAU	62	48.24	4.430	.563
	Treatment	66	48.56	5.959	.734
SWAP-II Obsessional Qfactor prototype BL	TAU	62	50.63	5.806	.737
	Treatment	66	51.20	7.019	.864
SWAP-II Hysteric-Histrionic Qfactor prototype BL	TAU	62	39.61	6.492	.824
	Treatment	66	41.97	7.160	.881
SWAP-II Personality Health Qfactor prototypeBL	TAU	62	46.52	7.511	.954
	Treatment	66	46.30	5.781	.712

Independent Samples Test										
		Levene's Test for Equality of Variances				t-test for Equality of Means				
		F	Sig.	t	df	Sig. (2- tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
SWAP-II Depressive Qfactor prototype BL	Equal variances assumed	1.661	.200	-.533	126	.595	-.599	1.123	-2.821	1.624
	Equal variances not assumed			-.532	123.395	.596	-.599	1.126	-2.827	1.630
	Equal variances assumed	.385	.536	2.153	126	.033	2.547	1.183	.206	4.889
	Equal variances not assumed			2.157	125.992	.033	2.547	1.181	.210	4.885
SWAP-II Anxious- Avoidant Qfactor prototype BL	Equal variances assumed	.344	.558	-	126	.028	-2.583	1.166	-4.890	-.276
	Equal variances not assumed			2.216						
	Equal variances assumed			-	125.070	.029	-2.583	1.166	-4.892	-.275
	Equal variances not assumed			2.215						
SWAP-II Dependent- Victimized Qfactor prototype BL	Equal variances assumed	.004	.947	.443	126	.659	.500	1.130	-1.735	2.736
	Equal variances not assumed			.443	125.759	.658	.500	1.129	-1.734	2.735
	Equal variances assumed	1.284	.259	-.403	126	.688	-.392	.975	-2.322	1.537
	Equal variances not assumed			-.402	123.907	.689	-.392	.977	-2.326	1.541

	Equal variances assumed	.394	.531	.452	126	.652	.489	1.083	-1.655	2.633
SWAP-II Paranoid Qfactor prototype BL	Equal variances not assumed			.451	124.828	.653	.489	1.085	-1.657	2.636
	Equal variances assumed	.099	.754	-	126	.015	-3.131	1.263	-5.632	-.631
SWAP-II Narcissistic Qfactor prototype BL	Equal variances not assumed			2.479						
	Equal variances not assumed			-	122.979	.015	-3.131	1.267	-5.640	-.623
				2.471						
SWAP-II Borderline- Dysregulated Qfactor prototype BL	Equal variances not assumed	3.604	.060	-.342	126	.733	-.319	.933	-2.165	1.528
	Equal variances not assumed			-.345	119.800	.731	-.319	.924	-2.149	1.512
	Equal variances not assumed	2.327	.130	-.497	126	.620	-.568	1.143	-2.829	1.693
SWAP-II Obsessional Qfactor prototype BL	Equal variances not assumed			-.500	124.048	.618	-.568	1.136	-2.816	1.680
	Equal variances not assumed	.001	.973	-	126	.054	-2.357	1.211	-4.752	.039
SWAP-II Hysteric- Histrionic Qfactor prototype BL	Equal variances not assumed			1.947						
	Equal variances not assumed			-	125.847	.053	-2.357	1.207	-4.745	.031
				1.953						
SWAP-II Personality Health Qfactor prototypeBL	Equal variances not assumed	4.564	.035	.181	126	.857	.213	1.180	-2.123	2.549
	Equal variances not assumed			.179	114.498	.858	.213	1.190	-2.144	2.570

**Appendix Q. SWAP-II end of treatment independent T-test results**

<b>Group Statistics</b>					
	Allocation group	N	Mean	Std. Deviation	Std. Error Mean
Normalized T-score - SWAP-II Qfactor prototype scale - Depressive End	TAU	55	63.73	7.194	.970
	Treatment	55	61.47	8.397	1.132
Normalized T-score - SWAP-II Qfactor prototype scale - Anxious-Avoidant End	TAU	55	57.60	6.364	.858
	Treatment	55	56.80	7.090	.956
Normalized T-score - SWAP-II Qfactor prototype scale - Dependent-Victimized End	TAU	55	52.00	7.850	1.059
	Treatment	55	51.29	6.391	.862
Normalized T-score - SWAP-II Qfactor prototype scale - Schizoid-Schizotypal End	TAU	55	58.65	6.818	.919
	Treatment	55	56.87	6.950	.937
Normalized T-score - SWAP-II Qfactor prototype scale - Antisocial-Psychopathic End	TAU	55	47.71	5.520	.744
	Treatment	55	47.44	6.253	.843
Normalized T-score - SWAP-II Qfactor prototype scale - Paranoid End	TAU	55	53.38	6.026	.813
	Treatment	55	51.29	6.425	.866
Normalized T-score - SWAP-II Qfactor prototype scale - Narcissistic End	TAU	55	49.53	6.170	.832
	Treatment	55	49.98	8.449	1.139
Normalized T-score - SWAP-II Qfactor prototype scale - Borderline-Dysregulated End	TAU	55	49.67	4.647	.627
	Treatment	55	47.05	7.230	.975
Normalized T-score - SWAP-II Qfactor prototype scale - Obsessional End	TAU	55	53.27	6.892	.929
	Treatment	55	55.40	6.525	.880
Normalized T-score - SWAP-II Qfactor prototype scale - Hysteric-Histrionic End	TAU	55	37.13	6.110	.824
	Treatment	55	39.22	6.288	.848
Normalized T-score - SWAP-II Qfactor prototype scale - Personality Health End	TAU	55	43.00	6.681	.901
	Treatment	55	46.00	7.510	1.013

Independent Samples Test										
		Levene's Test for Equality of Variances				t-test for Equality of Means				
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Normalized T-score - SWAP-II Qfactor prototype scale - Depressive End	Equal variances assumed	1.693	.196	1.512	108	.133	2.255	1.491	-.701	5.210
	Equal variances not assumed			1.512	105.517	.133	2.255	1.491	-.702	5.211
Normalized T-score - SWAP-II Qfactor prototype scale - Anxious-Avoidant End	Equal variances assumed	1.235	.269	.623	108	.535	.800	1.285	-1.747	3.347
	Equal variances not assumed			.623	106.763	.535	.800	1.285	-1.747	3.347
Normalized T-score - SWAP-II Qfactor prototype scale - Dependent- Victimized End	Equal variances assumed	1.449	.231	.520	108	.604	.709	1.365	-1.996	3.415
	Equal variances not assumed			.520	103.730	.605	.709	1.365	-1.998	3.416
Normalized T-score - SWAP-II Qfactor prototype scale - Schizoid- Schizotypal End	Equal variances assumed	.006	.938	1.357	108	.178	1.782	1.313	-.820	4.384
	Equal variances not assumed			1.357	107.961	.178	1.782	1.313	-.820	4.384
Normalized T-score - SWAP-II Qfactor prototype scale - Antisocial- Psychopathic End	Equal variances assumed	.527	.469	.242	108	.809	.273	1.125	-1.957	2.502
	Equal variances not assumed			.242	106.362	.809	.273	1.125	-1.957	2.502
Normalized T-score - SWAP-II Qfactor prototype scale -	Equal variances assumed	.005	.946	1.760	108	.081	2.091	1.188	-.264	4.445

Paranoid End	Equal			1.760	107.559	.081	2.091	1.188	-.264	4.445
	variances not assumed									
	Equal	3.931	.050	-.322	108	.748	-.455	1.411	-3.251	2.342
Normalized T-score	variances									
- SWAP-II Qfactor	assumed									
prototype scale -	Equal			-.322	98.841	.748	-.455	1.411	-3.254	2.345
Narcissistic End	variances not assumed									
	Equal	9.472	.003	2.259	108	.026	2.618	1.159	.321	4.915
Normalized T-score	variances									
- SWAP-II Qfactor	assumed									
prototype scale -	Equal			2.259	92.111	.026	2.618	1.159	.317	4.920
Borderline-	variances not assumed									
Dysregulated End	Equal	.143	.706	-	108	.099	-2.127	1.280	-4.664	.409
	variances			1.662						
Normalized T-score	assumed									
- SWAP-II Qfactor	Equal			-	107.679	.099	-2.127	1.280	-4.664	.409
prototype scale -	variances not assumed			1.662						
Obsessional End	Equal	.000	.984	-	108	.080	-2.091	1.182	-4.434	.253
	variances			1.769						
Normalized T-score	assumed									
- SWAP-II Qfactor	Equal			-	107.911	.080	-2.091	1.182	-4.434	.253
prototype scale -	variances not assumed			1.769						
Hysteric-Histrionic	Equal									
End	variances									
	assumed									
Normalized T-score	Equal	.720	.398	-	108	.029	-3.000	1.355	-5.687	-.313
- SWAP-II Qfactor	variances			2.213						
prototype scale -	assumed									
Personality Health	Equal			-	106.552	.029	-3.000	1.355	-5.687	-.313
End	variances not assumed			2.213						

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**Appendix Qa. Patient borderline-dysregulated scores at baseline and end-of-treatment – TAU group**

	TAU											
Patient ID	101	102	184	195	208	227	259	261	291	297	323	397
Borderline-dysregulated baseline score	53	55*	49	59*	46	51	50	43	55*	59*	49	55*
Borderline-dysregulated end-of-treatment score	56*†	48†	56*†	57*	56*†	57*†	56*†	57*†	45†	60***†	55*†	53†
Borderline-dysregulated Change score	-3	7	-7	2	-10	-6	-6	-14	10	-1	-6	3
HDRS baseline score	28	17	21	26	23	15	18	12	23	22	18	23
HDRS end-of-treatment score	30	13	20	n/a	n/a	16	17	21	16	n/a	25	24
HDSR 18m change score	-2	4	1	n/a	n/a	-1	1	-9	7	n/a	-7	-1
HDRS end-of-follow-up score	26	20	24	30	30	24	18	20	18	n/a	n/a	20
HDSR 42m change score	2	-3	-3	-4	-7	-9	0	-8	5	n/a	n/a	3

\*Score indicates “features”

\*\* Score indicates “disorder”

† Categorical change, compared to the baseline profile

**Appendix Qb. Patient borderline-dysregulated scores at baseline and end-of-treatment – LTPP group**

	LTPP															
Patient ID	114	131	154	171	178	183	198	199	255	301	305	314	319	324	351	370
Borderline-dysregulated baseline score	64**	40	52	47	51	55*	55*	56*	64**	55*	57*	54	40	58*	55*	56*
Borderline-dysregulated end-of-treatment score	58*†	57*†	57*†	56*†	56*†	n/a	46†	63**†	60**	49†	55*	58*†	57*†	n/a	52†	44†
Borderline-dysregulated end-of-treatment change score	12	-17	-5	-9	-5	n/a	9	-7	4	6	2	-4	-14	n/a	3	12
HDRS baseline score	29	19	24	21	21	17	16	17	21	18	12	17	22	21	24	14
HDRS end-of-treatment score	20	18	22	26	20	n/a	12	21	12	16	n/a	21	14	n/a	29	12
HDSR 18m change score	9	1	2	-5	1	n/a	4	-4	9	2	n/a	-4	7	n/a	-5	2
HDRS end-of-follow-up score	25	16	30	28	27	n/a	4	23	14	n/a	n/a	12	17	n/a	22	16
HDSR 42m change score	4	3	-6	-7	-6	n/a	12	-6	7	n/a	n/a	5	5	n/a	2	-2

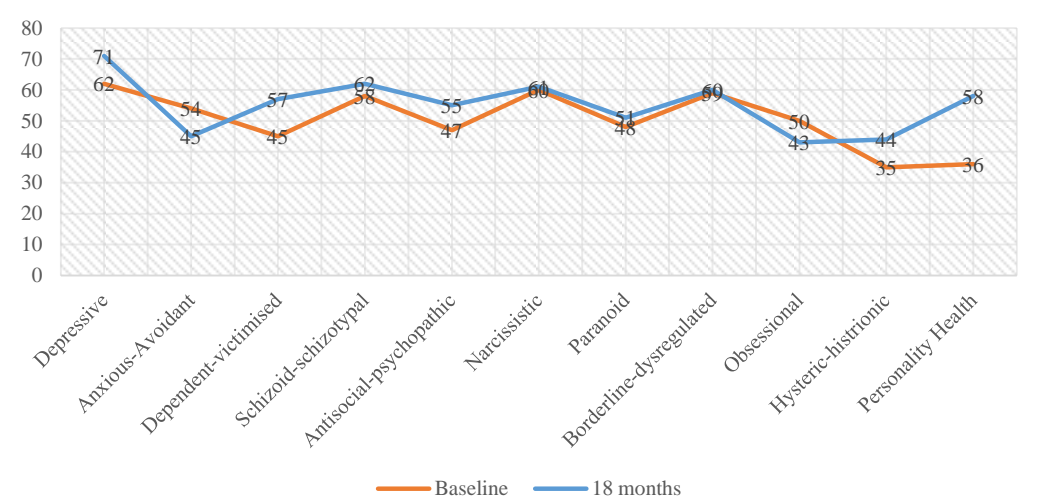
\*Score indicates “features”

\*\* Score indicates “disorder”

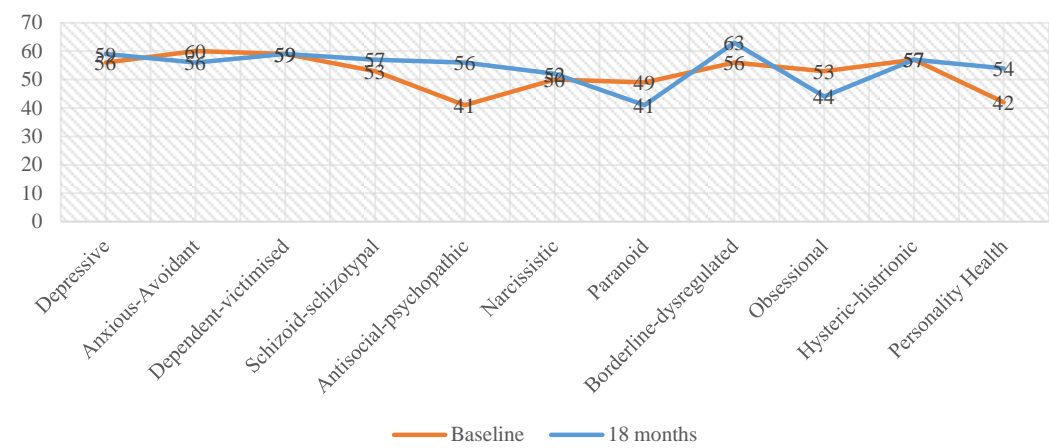
† Categorical change, compared to the baseline profile

**Appendix R. Borderline-dysregulated personality disorder patients’ full SWAP-II personality profiles**

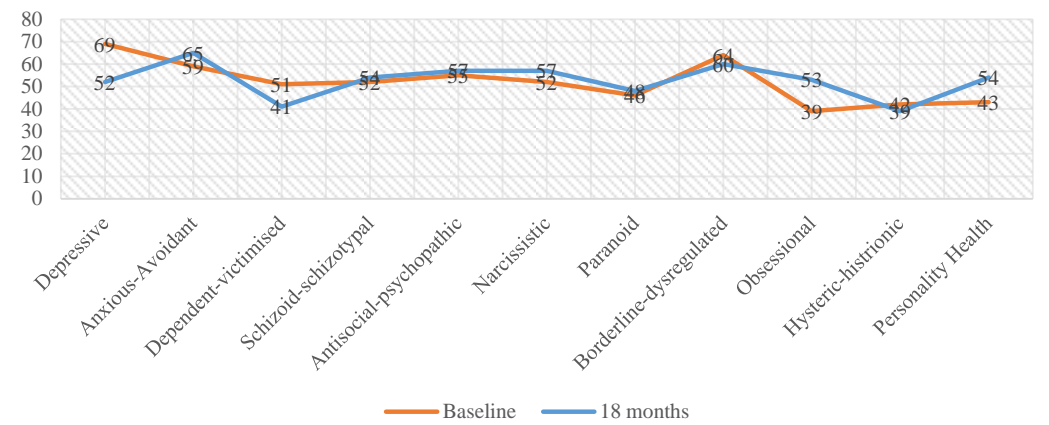
**Patient 297 (TAU)**



**Patient 199 (LTPP)**



**Patient 255 (LTPP)**



### Appendix S – Correlation coefficients between the SWAP-II Q-factor personality change scores and the HDRS-17, GAF and CORE-OM change scores

Allocation group	Borderline Dysregulated Change Score	Personality Health Change Score 18m	Emotional Dysregulation Trait Change Score 18m	Depressive Q-factor change score	Anxious-Avoidant Q-factor change score	Dependent-Victimised Q-factor change score	Schizoid-Schizotypal Q-factor change score	Antisocial Q-factor change score	Paranoid Q-factor change score	Narcissistic Q-factor change score	Obsessional Q-factor change score	Hysterical-Histrionic Q-factor change score	Hamilton Change Score at 18 months	Hamilton Change Score at 42 months	GAF Change Score at 18 months	GAF Change Score at 42 months	CORE Change Score at 18 months	CORE Change Score at 42 months
TAU	Borderline Dysregulated Change Score	1	.500**	.726**														
	N	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
	Personality Health Change Score 18m	.500**	1	.300*														
	N	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
	Emotional Dysregulation Trait Change Score 18m	.726**	.300*	1														
	N	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
	Depressive Q-factor change score	0.162	0.185	0.004	1													
TAU	N	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
	Anxious-Avoidant Q-factor change score	0.26	.412**	0.202	.457**	1												
	N	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
	Dependent-Victimised Q-factor change score	-0.218	-0.055	-0.256	.400**	0.159	1											
	N	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
	Schizoid-Schizotypal Q-factor change score	-0.163	.275*	-.296*	0.05	0.027	-0.146	1										
	N	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35

Allocation group	Borderline Dysregulated Change Score	Personality Health Change Score 18m	Emotional Dysregulation Trait Change Score 18m	Depressive Q-factor change score	Anxious-Avoidant Q-factor change score	Dependent-Victimised Q-factor change score	Schizoid-Schizotypal Q-factor change score	Antisocial Q-factor change score	Paranoid Q-factor change score	Narcissistic Q-factor change score	Obsessional Q-factor change score	Hysterio-Histrionic Q-factor change score	Hamilton Change Score at 18 months	Hamilton Change Score at 42 months	GAF Change Score at 18 months	GAF Change Score at 42 months	CORE Change Score at 18 months	CORE Change Score at 42 months
Antisocial Q-factor change score	0.071	0.111	0.021	-.339*	-.557**	-0.265	.301*		1 .309*	0.256	-0.181	-0.01	0.039	-0.007	0.048	0.014	0.013	-0.247
N	55	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
Paranoid Q-factor change score	.455**	.349**	.382**	-.393**	-0.12	-.606**		0.028	.309*	1 .525**	-0.038	-0.219	0.142	0.141	0.083	0.083	0.214	0.112
N	55	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
Narcissistic Q-factor change score	0.066	0.228	0.103	-0.26	-.288*	-.268*		-0.181	0.256	.525**	1	0.232	-0.167	-0.117	0.002	-0.055	-0.076	-0.078
N	55	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
Obsessional Q-factor change score	-.378**	-0.077	-0.245	-0.238	-0.147	0.012	-0.258	-0.181	-0.038	0.232	1	-0.128	-0.24	-0.258	-0.19	-.369*	-0.078	0.128
N	55	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
Hysterio-Histrionic Q-factor change score	-0.056	-0.087	-0.174	-0.124	-0.057	.503**		0.014	-0.01	-0.219	-0.167	-0.128	1	-0.123	-0.19	-0.15	-0.291	-0.093
N	55	55	55	55	55	55	55	55	55	55	55	55	46	45	44	44	34	35
Borderline Dysregulated Change Score	1	.454**	.596**	0.058	0.041	.303*		-0.154	0.233	.439**	0.156	-0.219	0.247	0.224	.319*	0.131	0.239	0.306
N	54	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41
Personality Health Change Score 18m	.454**	1	0.182	0.245	.471**	0.141	0.249	0.257	.458**	0.22	0.133	-0.02	0.2	.398**	.319*	.415**	0.306	.461**
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Emotional Dysregulation Trait Change Score 18m	.596**	0.182	1	-0.022	-0.098	0.045	-0.211	0.137	.291*	0.08	-0.212	0.089	0.245	0.166	0.169	0.022	0.046	0.021
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42

Allocation group	Borderline Dysregulated Change Score	Personality Health Change Score 18m	Emotional Dysregulation Trait Change Score 18m	Depressive Q-factor change score	Anxious-Avoidant Q-factor change score	Dependent-Victimised Q-factor change score	Schizoid-Schizotypal Q-factor change score	Antisocial Q-factor change score	Paranoid Q-factor change score	Narcissistic Q-factor change score	Obsessional Q-factor change score	Hysterical-Histrionic Q-factor change score	Hamilton Change Score at 18 months	Hamilton Change Score at 42 months	GAF Change Score at 18 months	GAF Change Score at 42 months	CORE Change Score at 18 months	CORE Change Score at 42 months
Depressive Q-factor change score	0.058	0.245	-0.022	1	.353**	0.185	0.151	-.327*	-0.138	-0.148	-.304*	-0.262	0.119	.357*	0.164	.499**	.311*	0.227
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Anxious-Avoidant Q-factor change score	0.041	.471**	-0.098	.353**	1	0.032	0.114	-.311*	-0.042	-0.018	0.027	-.321*	0.228	.436**	.404**	.568**	0.261	.351*
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Dependent-Victimised Q-factor change score	.303*	0.141	0.045	0.185	0.032	1	0.004	-0.062	-0.109	-.312*	-0.261	.570**	0.07	0.255	-0.229	0.063	0.101	0.039
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Schizoid-Schizotypal Q-factor change score	-0.154	0.249	-0.211	0.151	0.114	0.004	1	0.121	-0.051	-.346*	0.093	-0.223	-0.157	0.062	-0.032	0.15	-0.134	0.017
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Antisocial Q-factor change score	0.233	0.257	0.137	-.327*	-.311*	-0.062	0.121	1	.286*	0.143	0.052	0.143	-0.018	-0.023	-0.12	-0.038	0.002	0.007
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Paranoid Q-factor change score	.439**	.458**	.291*	-0.138	-0.042	-0.109	-0.051	.286*	1	.578**	0.186	-0.041	0.23	0.115	0.115	-0.019	0.21	0.198
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Narcissistic Q-factor change score	0.156	0.22	0.08	-0.148	-0.018	-.312*	-.346*	0.143	.578**	1	.444**	-0.142	0.133	-0.015	0.203	-0.105	0.148	0.068
N	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42

Obsessional Q-factor change score	-0.219	0.133	-0.212	-.304*		0.027	-0.261	0.093	0.052	0.186	.444**	1	-0.238	-0.016	-0.036	0.039	-0.164	-0.192	-0.104
N	54	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42
Hysterio-Histrionic Q-factor change score	0.247	-0.02	0.089	-0.262	-.321*	.570**		-0.223	0.143	-0.041	-0.142	-0.238	1	-0.013	-0.099	-0.275	-.344*	0.02	-0.085
N	54	54	54	54	54	54	54	54	54	54	54	54	54	50	46	50	46	41	42

\*\* Correlation is significant at the 0.01 level (2-tailed); \* Correlation is significant at the 0.05 level (2-tailed);

**Appendix Ta. Borderline-dysregulated baseline item correlation with the HDRS-17, CORE-OM and GAF change scores at 18- and 42-months**

Allocation group			HDRS-17 Change Score at 18 months	HDRS-17 Change Score at 42 months	GAF Change Score at 18 months	GAF Change Score at 42 months	CORE Change Score at 18 months	CORE Change Score at 42 months
TAU	Tends to become attached quickly or intensely	Pearson Correlation	-0.013	-0.047	0.065	0.005	0.157	0.087
		N	46	45	44	44	35	36
	Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.	Pearson Correlation	0.161	0.192	0.196	0.087	0.067	-0.082
		N	46	45	44	44	35	36
	Tends to stir up conflict or animosity between other people	Pearson Correlation	0.177	0.175	0.048	0.055	0.099	0.003
		N	46	45	44	44	35	36
	Tends to be needy or dependent	Pearson Correlation	0.008	-0.029	-0.014	0.099	0.088	-0.101
		N	46	45	44	44	35	36
	Tends to fear she/he will be rejected or abandoned	Pearson Correlation	0.219	0.005	0.098	-0.09	.385*	.382*
		N	46	45	44	44	35	36
	Tends to feel misunderstood, mistreated or victimized	Pearson Correlation	0.025	0.071	-0.092	-0.007	0.045	0.062
		N	46	45	44	44	35	36
	Relationships tend to be unstable, chaotic, and rapidly changing	Pearson Correlation	-.313*	-0.202	-.344*	-0.02	0.068	0.065
		N	46	45	44	44	35	36
	Tend to become irrational when strong emotions are stirred up	Pearson Correlation	0.153	0.003	0.171	-0.157	0.05	-0.171
		N	46	45	44	44	35	36
	Struggles with genuine wishes to kill him/herself	Pearson Correlation	-0.075	-0.099	-0.158	-0.091	-0.219	-.345*
		N	46	45	44	44	35	36
	Is prone to intense anger, out of proportion with the situation at hand	Pearson Correlation	0.025	-0.045	0.138	0.108	0.103	-0.049
		N	46	45	44	44	35	36



LTPP	Work life and/or living arrangements tend to be chaotic or unstable	Pearson Correlation	0.12	-0.044	0	-0.065	0.136	-0.234
		N	46	45	44	44	35	36
	Tends to feel unhappy, depressed or despondent	Pearson Correlation	0.027	0.151	-0.114	0.124	0.1	0.006
		N	46	45	44	44	35	36
	Emotions tend to change rapidly and unpredictably	Pearson Correlation	0.146	0.164	0.129	0.235	0.169	-0.035
		N	46	45	44	44	35	36
	Tends to become attached quickly or intensely	Pearson Correlation	-0.045	-0.058	0.24	-0.116	-0.077	-0.304
		N	51	46	51	46	42	42
	Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.	Pearson Correlation	-0.172	0.018	0.056	0.202	-0.12	-0.112
		N	51	46	51	46	42	42
	Tends to stir up conflict or animosity between other people	Pearson Correlation	0.153	0.222	0.042	0.214	.363*	.418**
		N	51	46	51	46	42	42
	Tends to be needy or dependent	Pearson Correlation	0.046	0.134	-0.113	0.122	-0.17	-0.211
		N	51	46	51	46	42	42
	Tends to fear she/he will be rejected or abandoned	Pearson Correlation	0.089	0.122	0.037	0.156	0.069	0.075
		N	51	46	51	46	42	42
	Tends to feel misunderstood, mistreated or victimized	Pearson Correlation	0.078	0.003	0.2	-0.003	0.087	0.104
		N	51	46	51	46	42	42
	Relationships tend to be unstable, chaotic, and rapidly changing	Pearson Correlation	0.117	0.016	-0.025	-0.053	-0.202	-0.173
		N	51	46	51	46	42	42
	Tend to become irrational when strong emotions are stirred up	Pearson Correlation	-0.14	0.181	-0.029	0.29	-0.089	0.138
		N	51	46	51	46	42	42
	Struggles with genuine wishes to kill him/herself	Pearson Correlation	0.06	0.205	0.014	0.026	-0.104	0.108
		N	51	46	51	46	42	42
	Is prone to intense anger, out of proportion with the	Pearson Correlation	-0.033	-0.133	-0.019	-0.088	-0.176	-0.254

situation at hand

	N	51	46	51	46	42	42
Work life and/or living arrangements tend to be chaotic or unstable	Pearson Correlation	0.112	-0.007	0.186	-0.067	0.068	-0.076
	N	51	46	51	46	42	42
Tends to feel unhappy, depressed or despondent	Pearson Correlation	-0.097	-0.235	0.035	-0.023	-0.034	-0.157
	N	51	46	51	46	42	42
Emotions tend to change rapidly and unpredictably	Pearson Correlation	-0.063	-0.017	0.132	0.036	-0.109	-0.185
	N	51	46	51	46	42	42

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\*\* Correlation is significant at the 0.01 level (2-tailed); \* Correlation is significant at the 0.05 level (2-tailed).

**Appendix Tb Borderline-dysregulated items change score correlation with the HDRS-17, CORE-OM and GAF change scores at 18- and 42-months**

Allocation group			HDRS Change Score 18m	HDRS Change Score 42m	GAF Change Score 18m	GAF Change Score 42m	CORE Change Score 18m	CORE Change Score 42m
TAU	Tends to become attached quickly or intensely	Pearson Correlation N	-0.024 39	-0.141 39	-0.034 38	-0.192 39	0.065 31	-0.009 35
	Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.	Pearson Correlation N	0.091 39	.386* 39	0.186 38	0.302 39	0.182 31	0.157 35
	Tends to stir up conflict or animosity between other people	Pearson Correlation N	-0.012 39	0.055 39	0.091 38	0.162 39	0.06 31	0.312 35
	Tends to be needy or dependent	Pearson Correlation N	-0.14 39	-0.121 39	0.022 38	0.155 39	-0.085 31	-0.111 35
	Tends to fear she/he will be rejected or abandoned	Pearson Correlation N	0.246 39	0.034 39	0.013 38	-0.029 39	0.239 31	0.197 35
	Tends to feel misunderstood, mistreated or victimized	Pearson Correlation N	0.253 39	.385* 39	0.142 38	0.26 39	0.208 31	0.202 35
	Relationships tend to be unstable, chaotic, and rapidly changing	Pearson Correlation N	-0.307 39	- .480** 39	-0.176 38	-0.232 39	-0.091 31	-0.19 35
	Tend to become irrational when strong emotions are stirred up	Pearson Correlation N	-0.022 39	0.069 39	-0.056 38	-0.001 39	0.338 31	0.161 35
	Struggles with genuine wishes to kill him/herself	Pearson Correlation N	-0.08 39	-0.051 39	-0.016 38	0.133 39	-0.078 31	-0.196 35
	Is prone to intense anger, out of proportion with the situation at hand	Pearson Correlation N	-0.02 39	0.147 39	0.093 38	.317* 39	0.063 31	0.181 35
	Work life and/or living arrangements tend to be chaotic or unstable	Pearson Correlation N	0.177 39	-0.169 39	0.129 38	-0.237 39	0.139 31	-0.301 35
	Tends to feel unhappy, depressed or despondent	Pearson Correlation N	0.262 39	0.093 39	0.21 38	0.115 39	0.04 31	-0.228 35
	Emotions tend to change rapidly and unpredictably	Pearson Correlation	-0.088	0.137	0.102	.445**	0.142	0.059

LTPP		N	36	36	35	36	29	32
	Tends to become attached quickly or intensely	Pearson Correlation	-0.051	-0.039	0.191	-0.077	-0.122	-0.261
		N	44	41	44	41	37	38
	Emotions tend to spiral out of control, leading to extremes of anxiety, sadness, rage, etc.	Pearson Correlation	-0.087	0.183	-0.06	0.203	-0.104	-0.016
		N	44	41	44	41	37	38
	Tends to stir up conflict or animosity between other people	Pearson Correlation	0.096	0.276	0.018	0.158	0.163	.350*
		N	44	41	44	41	37	38
	Tends to be needy or dependent	Pearson Correlation	-0.083	0.009	-0.085	0.148	-0.213	-0.062
		N	44	41	44	41	37	38
	Tends to fear she/he will be rejected or abandoned	Pearson Correlation	0.033	-0.027	0.133	0.016	0.05	0.03
		N	44	41	44	41	37	38
	Tends to feel misunderstood, mistreated or victimized	Pearson Correlation	0.241	0.077	.358*	0.068	0.114	0.163
		N	44	41	44	41	37	38
	Relationships tend to be unstable, chaotic, and rapidly changing	Pearson Correlation	0.114	0.142	-0.04	0.076	-0.019	0.049
		N	44	41	44	41	37	38
	Tend to become irrational when strong emotions are stirred up	Pearson Correlation	0.11	.327*	-0.013	0.253	-0.028	0.144
		N	44	41	44	41	37	38
	Struggles with genuine wishes to kill him/herself	Pearson Correlation	0.094	0.213	-0.034	0.168	-0.031	0.095
		N	44	41	44	41	37	38
	Is prone to intense anger, out of proportion with the situation at hand	Pearson Correlation	0.103	-0.049	-0.121	-0.141	-0.05	-0.22
		N	44	41	44	41	37	38
	Work life and/or living arrangements tend to be chaotic or unstable	Pearson Correlation	-0.048	-0.065	0.02	-0.046	0.05	-0.091
		N	44	41	44	41	37	38
	Tends to feel unhappy, depressed or despondent	Pearson Correlation	0.052	0.058	0.252	.318*	0.006	-0.025
		N	44	41	44	41	37	38
	Emotions tend to change rapidly and unpredictably	Pearson Correlation	0.09	0.216	-0.06	0.043	-0.14	-0.047
		N	44	41	44	41	37	38

\*\* Correlation is significant at the 0.01 level (2-tailed); \* Correlation is significant at the 0.05 level (2-tailed).

**Appendix U. Regression Analysis****Regression statistics on the SWAP-II borderline-dysregulated score at baseline as a predictor of the HDRS-17 change scores at 18- and 42 months**

<b>Model Summary</b>					
Allocation group	Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
TAU	1	.324 <sup>a</sup>	.105	.084	5.666
LTPP	1	.224 <sup>a</sup>	.050	.030	7.028

a. Predictors: (Constant), Hamilton Change Score at 18 months

<b>ANOVA<sup>a</sup></b>						
Allocation group	Model		Sum of Squares	df	Mean Square	F
TAU	1	Regression	.297	1	.297	.006
		Residual	2013.442	44	45.760	
		Total	2013.739	45		
LTPP	1	Regression	4.387	1	4.387	.157
		Residual	1372.437	49	28.009	
		Total	1376.824	50		

a. Dependent Variable: Hamilton Change Score at 18 months

b. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

<b>Coefficients<sup>a</sup></b>						
Allocation group	Model		Unstandardized Coefficients		Standardized Coefficients	t
			B	Std. Error	Beta	
TAU	1	(Constant)	3.736	12.963		.288
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	-.021	.266	-.012	-.081
LTPP	1	(Constant)	6.560	6.658		.985
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	-.053	.135	-.056	-.396

a. Dependent Variable: Hamilton Change Score at 18 months

**Model Summary**

Allocation group	Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
TAU	1	.007 <sup>a</sup>	.000	-.023	5.79943
Treatment	1	.054 <sup>a</sup>	.003	-.020	6.71059

a. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

**ANOVA<sup>a</sup>**

Allocation group	Model	Sum of Squares	df	Mean Square	F	Sig.
TAU	1	Regression	.075	1	.075	.002
		Residual	1446.236	43	33.633	.962 <sup>b</sup>
		Total	1446.311	44		
LTPP	1	Regression	5.807	1	5.807	.129
		Residual	1981.410	44	45.032	.721 <sup>b</sup>
		Total	1987.217	45		

a. Dependent Variable: Hamilton Change Score at 42 months

b. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

**Coefficients<sup>a</sup>**

Allocation group	Model	Unstandardized Coefficients B	Std. Error	Standardized Coefficients Beta	t	Sig.
TAU	1	(Constant)	.164	10.196	.016	.987
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	.010	.211	.007	.962
		(Constant)	.897	8.338	.108	.915
Treatment	1	SWAP-II Borderline-Dysregulated Q-factor prototype BL	.061	.171	.054	.721
		(Constant)	.897	8.338	.108	.915

a. Dependent Variable: Hamilton Change Score at 42 months

**Regression statistics on the SWAP-II borderline-dysregulated score at baseline as a predictor of the GAF change scores at 18- and 42 months**

Model Summary					
Allocation group	Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
TAU	1	.189 <sup>a</sup>	.036	.013	8.58311
LTPP	1	.079 <sup>a</sup>	.006	-.014	8.06620

a. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

ANOVA <sup>a</sup>						
Allocation group	Model	Sum of Squares	df	Mean Square	F	Sig.
TAU	1	Regression	114.119	1	114.119	1.549
		Residual	3094.131	42	73.670	.220 <sup>b</sup>
		Total	3208.250	43		
LTPP	1	Regression	19.920	1	19.920	.306
		Residual	3188.119	49	65.064	.583 <sup>b</sup>
		Total	3208.039	50		

a. Dependent Variable: GAF Change Score at 18 months

b. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

Coefficients <sup>a</sup>						
Allocation group	Model	Unstandardized Coefficients	Standardized Coefficients	t	Sig.	
		B	Std. Error	Beta		
TAU	1	(Constant)	-16.480	16.706		-.986
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	.425	.341	.189	1.245
		(Constant)	13.717	10.147		1.352
LTPP	1	SWAP-II Borderline-Dysregulated Q-factor prototype BL	-.114	.206	-.079	-.553
		(Constant)				.183

a. Dependent Variable: GAF Change Score at 18 months

Model Summary					
Allocation group	Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
TAU	1	.210 <sup>a</sup>	.044	.021	8.28995
LTPP	1	.135 <sup>a</sup>	.018	-.004	10.96153

a. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

ANOVA <sup>a</sup>							
Allocation group	Model		Sum of Squares	df	Mean Square	F	Sig.
TAU	1	Regression	133.508	1	133.508	1.943	.171 <sup>b</sup>
		Residual	2886.379	42	68.723		
		Total	3019.886	43			
LTPP	1	Regression	97.780	1	97.780	.814	.372 <sup>b</sup>
		Residual	5286.829	44	120.155		
		Total	5384.609	45			

a. Dependent Variable: GAF Change Score at 42 months

b. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

Coefficients <sup>a</sup>							
Allocation group	Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
			B	Std. Error	Beta		
TAU	1	(Constant)	-16.614	14.599		-1.138	.262
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	.420	.301	.210	1.394	.171
LTPP	1	(Constant)	-1.005	13.597		-.074	.941
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	.252	.279	.135	.902	.372

a. Dependent Variable: GAF Change Score at 42 months



**Regression statistics on the SWAP-II *borderline-dysregulated* score at baseline as a predictor of the CORE-OM change scores at 18- and 42 months**

Model Summary					
Allocation group	Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
TAU	1	.005 <sup>a</sup>	.000	-.030	.70649
LTPP	1	.086 <sup>a</sup>	.007	-.017	.72929

a. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

ANOVA <sup>a</sup>						
Allocation group	Model	Sum of Squares	df	Mean Square	F	Sig.
TAU	1	Regression	.000	1	.000	.001
		Residual	16.471	33	.499	.976 <sup>b</sup>
		Total	16.472	34		
LTPP	1	Regression	.158	1	.158	.297
		Residual	21.275	40	.532	.589 <sup>b</sup>
		Total	21.433	41		

a. Dependent Variable: CORE Change Score at 18 months

b. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

Coefficients <sup>a</sup>						
Allocation group	Model	Unstandardized Coefficients	Standardized Coefficients	t	Sig.	
		B	Std. Error	Beta		
TAU	1	(Constant)	-.006	1.385	-.004	.996
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	.001	.029	.005	.976
		(Constant)	-.161	1.024	-.157	.876
LTPP	1	SWAP-II Borderline-Dysregulated Q-factor prototype BL	.011	.021	.086	.589
		(Constant)				

a. Dependent Variable: CORE Change Score at 18 months

Model Summary					
Allocation group	Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
TAU	1	.048 <sup>a</sup>	.002	-.027	.85757
LTPP	1	.084 <sup>a</sup>	.007	-.018	.76618

a. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

ANOVA <sup>a</sup>							
Allocation group	Model		Sum of Squares	df	Mean Square	F	Sig.
TAU	1	Regression	.059	1	.059	.080	.779 <sup>b</sup>
		Residual	25.005	34	.735		
		Total	25.064	35			
LTPP	1	Regression	.168	1	.168	.287	.595 <sup>b</sup>
		Residual	23.481	40	.587		
		Total	23.650	41			

a. Dependent Variable: CORE Change Score at 42 months

b. Predictors: (Constant), SWAP-II Borderline-Dysregulated Q-factor prototype BL

Coefficients <sup>a</sup>							
Allocation group	Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.	
		B	Std. Error	Beta			
TAU	1	(Constant)	.435	1.720		.253	.802
		SWAP-II Borderline-Dysregulated Q-factor prototype BL	-.010	.036	-.048	-.283	.779
		(Constant)	.044	.965		.046	.964
Treatment	1	SWAP-II Borderline-Dysregulated Q-factor prototype BL	.011	.020	.084	.536	.595

a. Dependent Variable: CORE Change Score at 42 months

**Regression statistics for the HDRS-17, GAF and CORE-OM change scores regressed on the borderline-dysregulated baseline score**

Allocation Group	Measure	F	Sig.
<b>TAU</b>	HDRS-17 Change Score 18m	.006	.936
	HDRS-17 Change Score 42m	.002	.962
	GAF Change Score 18m	1.549	.220
	GAF Change Score 42m	1.943	.171
	CORE-OM Change Score 18m	.001	.976
	CORE-OM Change Score 42m	.080	.779
<b>LTPP</b>	HDRS-17 Change Score 18m	.157	.694
	HDRS-17 Change Score 42m	.129	.721
	GAF Change Score 18m	.306	.583
	GAF Change Score 42m	.814	.372
	CORE-OM Change Score 18m	.297	.589
	CORE-OM Change Score 42m	.287	.595