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Psychological therapies for people with borderline personality disorder (Protocol)

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[Intervention Protocol]

Psychological therapies for people with borderline personality disorder

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ABSTRACT

This is a protocol for a Cochrane Review (Intervention). The objectives are as follows:

To assess the beneficial and harmful effects of psychological therapies for people with borderline personality disorder (BPD).

BACKGROUND

Description of the condition

Borderline personality disorder (BPD) is a condition first recognised in the 20th century (Gunderson 2009). Historically, the term BPD was coined by Adolph Stern to describe a condition in the 'borderland' between psychosis and neurosis (Stern 1938). Subsequent psychoanalytic contributions (especially that of Kernberg 1975) have reaffirmed this distinction, emphasising that the capacity to test reality remains grossly intact but is subject to subtle distortions, especially under stress.

According to current diagnostic criteria, BPD is characterised by a pervasive pattern of instability in affect regulation, impulse control, interpersonal relationships, and self-image (APA 2013; WHO 1993). Clinical hallmarks include emotional dysregulation, impulsive aggression, repeated self-injury, and chronic suicidal tendencies (Fonagy 2009; Lieb 2004). Whereas some authors have suggested that it is a variant of affective disorders (Akiskal 2004), others claim that it is only the causes of these diseases that partially overlap in BPD (Paris 2007). Despite the difficulties in defining and delimiting the condition, BPD is still being widely researched. Its importance stems from the considerable psychological suffering of the persons concerned (Stiglmayr 2005; Zanarini 1998), the debilitating functional impairments (Gunderson 2011a; Gunderson 2011b; Niesten 2016; Skodol 2002; Soeteman 2008b), and from the significant impact it has on mental health services (Cailhol 2015; Hörz 2010; Soeteman 2008a; Tyrer 2015; Zanarini 2004; Zanarini 2012).

The definition of BPD in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM), Fifth Edition (DSM-5; APA 2013), Fourth Edition Text Revision (DSM-IV-TR; APA 2000), and Fourth Edition (DMS-IV; APA 1994) comprises nine criteria that cover the features mentioned above. At least five criteria should be met for a definite categorical BPD diagnosis to be made, and four criteria for probable diagnosis (see Appendix 1). In the alternative diagnostic classification system of the World Health Organization (WHO), the *International Classification of Diseases*, which is currently in its tenth edition (ICD-10; WHO 1993), the relating condition is referred to as "Emotionally unstable personality disorder (F60.3)", of which there is an impulsive type (F60.30) and a borderline type (F60.31; see Appendix 2). The latter essentially overlaps with the DSM-IV definition. There are 10 possible criteria defined, which very closely reflect the DSM criteria, with the exception of one criterion not included in DSM ("4. Difficulty in maintaining any course of action that offers no immediate reward"; WHO 1993). Out of 10 possible criteria at least five must be met, one of which must be "a marked tendency to quarrelsome behaviour and to conflicts with others, especially when impulsive acts are thwarted or criticised".

The prevalence of BPD in the general population is about 1.5% (Torgersen 2012). In clinical populations, BDP occurs frequently (Munk-Jørgensen 2010), with studies reporting a prevalence ranging from 9.3% to 46.3% and a mean point prevalence across the studies of 28.5% (Torgersen 2012). Though BPD is predominantly diagnosed in women (75%; APA 2000; APA 2013), it is estimated to be equally frequent in men (Lenzenweger 2007; Ten Have 2016; Torgersen 2001; Torgersen 2012). BPD commonly co-occurs with mood disorders, substance use disorder, eating disorders, post-traumatic stress disorder (PTSD), attention-deficit hyperactivity disorder (ADHD), and is also associated with other personality disorders (Coid 2006; Lenzenweger 2007; Stepp 2012; Storebø 2014; Tomko 2014). Suicidal behaviour is reported to occur in up to 84% of people diagnosed with BPD (Goodman 2012; Soloff 2002), and comorbid mood disorders or substance use disorders are the most common risk factors associated with successful suicide attempts (Black 2004; Doyle 2016; Yen 2004). Although the short- to medium-term outcome of BPD is poor, there is some evidence that long-term follow-ups show a more favourable course, with remission rates of about 85% to 88% within 10 years (Gunderson 2011b; Zanarini 2007). Here, however, remission only means that diagnostic criteria are not fulfilled and does not indicate the absence of any symptoms. Indeed, whereas acute symptoms - such as self-mutilation, help-seeking suicide threats or attempts and impulsivity - in most cases decrease with time, affective symptoms reflecting areas of chronic dysphoria, such as chronic feelings of emptiness, intense anger or profound abandonment, largely remain (Zanarini 2007). Therefore the majority of people with BPD still have significant levels of symptoms and experience severe and persistent impairment in social functioning (Kongerslev 2015; Ng 2016). Risk factors for a poorer, long-term outcome are comorbid substance use disorders, PTSD, and anxiety cluster disorders (Zanarini 2005; Zanarini 2007), as well as a family history of psychiatric disorder (especially mood disorders and substance use disorders), demographic issues such as older age, longer treatment history, pathological childhood experiences, temperament issues and adult psychosocial functioning (Chanen 2012; De Fruyt 2014; Kongerslev 2015; Zanarini 2007). It is estimated that about 60% to 78% of people diagnosed with BPD attempt suicide (Links 2009), though the rate of completed suicides is far less. Zanarini and colleagues found suicide rates of 4.5% during a 16-year follow-up (Zanarini 2015b), whereas Stone 1993 reported a suicide rate of 8.5% after 16.5 years. Study estimates of the lifetime risk of suicide among people diagnosed with BPD range from 3% to 10% (Links 2009).

People with BPD have difficulties achieving and maintaining vocational and social functioning over time (Zanarini 2010). Furthermore, treatment-seeking people with personality disorders, such as BPD, pose a high economic burden on society (van Asselt 2007). Effective treatments could potentially decrease the high costs associated with the condition (Soeteman 2008a).The problem of deliberate self-harm is also a particular issue within this group (Ayodeji 2015; Kongerslev 2015; Linehan 1997; Rossouw 2012). In medical settings, people diagnosed with BPD often present after self-harming behaviour or in suicidal crisis and are treated in emergency settings, often involving repeated psychiatric hospitalisations (Cailhol 2015).

In summary, BPD is a condition that has been extensively studied. It has a major impact on health facilities as those affected often present in crisis. Recovery from symptoms or functional impairment (or both) was previously considered likely for only a low percentage of people diagnosed with BPD. However, the long-term course, in terms of symptomatic recovery, is favourable (Zanarini 2012). Nonetheless, people diagnosed with BPD continue to have considerable interpersonal and functional problems, and sustainable recovery appears difficult to attain (Biskin 2015; Kongerslev 2015; Rossouw 2012).

Description of the intervention

About three quarters of people with BPD present to mental health care professionals (Tomko 2014), and they are even more likely to do so than people with mood, anxiety, or other personality disorders (Ansell 2007). Most will receive psychological interventions (Goodman 2010; Tomko 2014), and often for relatively long periods of time (e.g. for a period of one year or longer) (Ansell 2007; Zanarini 2015a).

A broad range of therapies exist for BPD. The therapy can be either individual or in a group, or a combination of these two treatment modalities. As for most other mental disorders, the psychological interventions can be based on the traditional, major psychotherapeutic schools, such as psychodynamic psychotherapy, cognitive behaviour therapy (CBT) or client-centered/humanistic therapy.

In addition, several specific treatment approaches have been developed within recent decades to meet the challenges of BPD treatment (Bateman 2015). These disorder-specific approaches are usually precisely structured and manualised (Bateman 2015). Strategies are provided for addressing interpersonal challenges, such as emotional dysregulation and impulsivity, which are core problems for people diagnosed with BPD and could lead to difficulties in forming a therapeutic alliance. Most BPD-specific psychological interventions involve multimodal therapy, treatment contracts, actively taking measures to minimise premature non-completion of treatment, providing a crisis intervention protocol and encouraging the patients' sense of agency (Bateman 2015; Clarkin 2012; De Groot 2008; Kongerslev 2015; Livesley 2012; Weinberg 2011). They are typically highly focused on affect and the therapeutic relationship, with a relatively active therapist implementing interventions within a supportive and validating atmosphere (Bateman 2015; Clarkin 2012; De Groot 2008; Kongerslev 2015; Livesley 2012; Weinberg 2011).

Among the specific psychological interventions for people diagnosed with BPD, the most commonly used are: transference-focused therapy (Clarkin 1999; Yeomans 2015), mentalisation-based treatment (Bateman 2004; Bateman 2006; Bateman 2016), dialectical behaviour therapy (Linehan 1993; Linehan 2015), cognitive analytic therapy (Chanen 2014; Ryle 1997), schema-focused therapy (Arntz 2009; Young 2003), and the systems training for emotional predictability and problem solving (Black 2009). Most of these treatments are designed as outpatient treatments of 6 to 24 months duration with 1- or 2-weekly individual sessions. Some also include additional group therapy sessions, inpatient or day-hospital therapeutic community treatment and psychoeducation. Broadly speaking, psychodynamic therapies aim to help their patients understand and reflect on their inner mental processes and make links between their past and current difficulties. Treatments based on CBT place emphasis on self-directed learning processes: patients are encouraged to identify their core beliefs, evaluate and modify their behaviour accordingly, and gain new experiences.

Dialectical behavioural therapy (DBT; Linehan 1993) is a complex psychological therapy that was developed using some of the principles of CBT in combination with mindfulness-based and Zen-Buddhistic and dialectical thinking strategies. It aims to change behaviour and enhance the ability to tolerate difficult or painful feelings by focusing on improving skills in stress tolerance, emotion regulation, interpersonal behaviour, and mindfulness.

Mentalisation-based therapy (Bateman 2004; Bateman 2016) is a complex psychodynamic and attachment-based psychological therapy programme that aims to increase the reflective functioning or mentalising capacity of the individual, helping the person to understand and recognise the feelings they evoke in others and the feelings they experience themselves, as well as improving the capacity for emotion regulation in interpersonal relations.

Schema-focused therapy (SFT; Young 2003) draws from both cognitive-behavioural and psychoanalytic theories and helps people with BPD to identify their self-defeating core themes arising from unmet emotional needs in childhood and presenting as maladaptive coping styles in adulthood. The goal of SFT is to aid patients in getting their needs met in adaptive ways.

Transference-focused psychotherapy (TFP; Clarkin 1999) strives to achieve integrated representations of self and others, modification of primitive defence operations, and resolution of identity diffusion by analysis of the transference within the therapeutic relationship. Primitive object relations, which can be polarised and split, may be transformed to advanced or mature object relations characterised by more integrated object relations. TFP relies on techniques of clarification, confrontation and transference interpretation within the relationship between patient and therapist.

Cognitive analytic therapy (Ryle 1997) assumes that people with BPD typically experience rapid switching from one self-state to another in a dissociate manner. The aim is to work with the patient cognitively to identify procedural sequences, chains of events, emotions, thoughts and motivations to understand how a target problem (like self-harm) is established and maintained, and to identify reciprocal roles (i.e. how early experiences are replayed later in life).

The systems training for emotional predictability and problem solving (Black 2009) combines group-based psychoeducation with skills training, and targets biased social cognition driven by cognitive filters or schemas.

Relaxation techniques and patient education programmes will be considered their own intervention class (i.e. not CBT or psychoanalytically based), as long as they are not explicitly grounded in or taken from a specific treatment approach (such as psychoeducation according to the DBT approach, CBT, or the SFT approach, etc.).

How the intervention might work

Evidence-based psychological therapies are based on assumptions about causality, core symptoms, and maintenance of the disorder (Kazdin 2004; Livesley 2003; Livesley 2004). The various psychotherapeutic approaches to BPD claim different mechanisms of action according to their respective models of disease causation (Huprich 2015; Livesley 2004; Livesley 2016). However, they also contain a number of common elements that can account for why a number of seemingly different approaches appear to be effective in ameliorating BPD symptoms (Bateman 2015; Fonagy 2014; Kongerslev 2015; Weinberg 2011), including: a clear and highly structured treatment framework; an explicit model of BPD symptomatology; a consistent focus on the therapeutic relationship, affect regulation, tolerance of emotional states, and biases in social cognition; a high priority given to self-harm and suicidal behavior; active therapists who deliver both support and validation as well as explorative and change-oriented interventions; mix of treatment formats (e.g. includes both individual and group therapy); and

therapist support in the form of supervision and regular meetings. Following Weinberg 2011:

1. 'emotional dysregulation' (e.g. intense anger and affective instability) is addressed through attention to affect, including raising awareness of emotional states, their triggers, and enhancing tolerance and regulative strategies;
2. 'behavioral dysregulation' (e.g. impulsivity, self-harm and suicidal behaviours) is addressed through change-oriented interventions including, for example, challenging negative thoughts, skills training, behavioral experiments, praise, and limit setting; and
3. 'interpersonal dysfunction' (e.g. unstable relationships and stress-related paranoid ideation) is treated using interventions that enhance the social-cognitive (or mentalising) capacities of the BPD patient, through making basic and often negatively biased automatic assumptions explicit and more realistic or adaptive, and through paying attention to the establishment and maintenance of a safe and sound working alliance within the therapy sessions.

There is a risk that psychological therapies might not be helpful for all patients, either due to the interventions delivered or through factors in the relationship between the patient and therapist (Kongerslev 2015; Lilienfeld 2007; Parry 2016), and very little research has been done on this in patients with BPD. The effectiveness of the therapy depends on the skills of the therapist to create the possibility for change with each patient. There is, therefore, the added complexity that the relationship or working alliance between the therapist and the patient is itself an 'active ingredient' of the therapy and that the quality of this relationship is an important predictor of outcome (Horvath 2011; Norcross 2011). There is no guarantee that the therapy will deliver what was specified in the manual or what was investigated in a randomised clinical trial (Parry 2016).

Finally, it should be noted that the highly structured treatment organisation, including regular meetings between staff to discuss patients as well as supervision and managerial support to staff, is presumably important not only for the provision of effective psychological therapy for persons diagnosed with BPD, but also in order to prevent iatrogenic harm (Hutsebaut 2012).

Why it is important to do this review

People with BPD and their family and friends experience high levels of psychological suffering. Moreover, BPD is associated with considerable social costs in terms of service use (e.g. presentation to emergency clinics due to self-harm or suicidal crises and repeated hospitalisations) and poor psychosocial functioning (e.g. inability to complete education or get/maintain a job). Against this background, identification of effective psychological therapies for BPD is important.

Our review aims to provide a systematic summary of the evidence from randomised controlled trials (RCTs) in order to help people with BPD, their family and friends, mental health care workers, and policy and decision managers in general, to make informed decisions about evidence-based treatment for BPD.

This is a protocol for an update of the two previous Cochrane Reviews on psychological therapies for BPD (Binks 2006; Stoffers-Winterling 2012). In addition to updating the former Cochrane Reviews, our study also seeks to address some of methodological limitations of both past and current reviews (Bateman 2015; Cristea 2017; Kliem 2010), by publishing a protocol for our review and using a broad and updated search strategy.

OBJECTIVES

To assess the beneficial and harmful effects of psychological therapies for people with borderline personality disorder (BPD).

METHODS

Criteria for considering studies for this review

Types of studies

Randomised controlled trials (RCTs).

Types of participants

Patients of all ages, in any setting, with a formal diagnosis of BPD according to the*Diagnostic and Statistical Manual of Mental Disorders* (DSM) Third Edition (DSM-III; APA 1980), Third Edition Revised (DSM-III-R; APA 1987), Fourth Edition (DSM-IV; APA 1994), Fourth Edition Text Revision (DSM-IV-TR; APA 2000), and Fifth Edition (DSM-5; APA 2013), with or without comorbid conditions.

To meet our inclusion criterion, at least 70% of study participants will have to have a formal diagnosis of BPD. We will also include studies involving subsamples of BPD patients provided that data on these patients are available separately (we will ask for separate data from trials including less than 70% BPD participants). We will not include studies that focus on people with mental impairment, organic brain disorder, dementia or other severe neurologic/neurodevelopmental diseases, should there be any.

Types of interventions

Any defined psychological intervention, regardless of theoretical orientation (e.g. psychodynamic therapy, CBT, systemic therapy or eclectic therapies designed for BPD treatment), in any kind of

treatment setting (e.g. inpatient, outpatient or day clinic), compared to:

1. unspecific control interventions/optional use such as standard care, treatment as usual (TAU) or waiting list (the first two can be combined in a meta-analysis as different forms of TAU, with a subgroup analysis investigating the statistical heterogeneity; see Subgroup analysis and investigation of heterogeneity);
2. specific psychotherapeutic interventions (well-defined and theory-driven); and
3. unspecific control interventions/obligatory use (e.g. community treatments, clinical management, general management, general psychiatric management; Links 2015).

Unspecific controls include conditions where a patient is free to use any treatment (except the comparison treatment), or to use no treatment at all. Unspecific controls at point 3 (above) include treatments that patients need to utilise, be it a very closely defined single intervention or participation in a broad clinical management programme. We will allow concomitant treatments providing they are applied to both treatment conditions.

We will also accept trials on relaxation techniques, such as autogenic training or meditation regimens; and patient education programmes, such as self-management and community-based education programs.

Types of outcome measures

Outcomes can either be self-rated by patients or observer-rated by clinicians. We will include only adequately validated measures (plus spontaneous reporting of adverse events).

We will analyse all outcomes at post-treatment and at six months' follow-up or longer.

Primary outcomes

1. BPD severity, as assessed by, for example, the Zanarini Rating Scale for Borderline Personality Disorder (Zan-BPD; Zanarini 2003); the Borderline Personality Disorder Severity Index, Fourth version (BPDSI-IV; Arntz 2003) or the Clinical Global Impression Scale for Borderline Personality Disorder Patients (CGI-BPD; Perez 2007).
2. Self-harm, in terms of proportion of participants with self-harming behaviour, or as assessed by, for example, the Deliberate Self-harm Inventory (DSHI; Gratz 2001) or the Self-harm Behavior Questionnaire (SHBQ; Guttierez 2001).
3. Suicide-related outcomes, as assessed by, for example, the Suicidal Behaviours Questionnaire (SBQ; Osman 2001) or the Beck Scale for Suicidal Ideation (BSSI; Beck 1979), or in terms of the proportion of patients with suicidal acts.
4. Functioning, as assessed by, for example, the Global Assessment Scale (GAS; Endicott 1976), the Global Assessment of Functioning Scale (GAF; APA 1987) or the Social Functioning Questionnaire (SFQ; Tyrer 2005).

Secondary outcomes

1. Anger, as assessed by, for example, the "Hostility" subscale of the Symptom Checklist-90-Revised (SCL-90-R; Derogatis 1994) or the State-Trait Anger Expression Inventory (STAXI; Spielberger 1988).
2. Affective instability, as assessed by, for example, the relevant item or subscale on the Zan-BPD (Zanarini 2003), CGI-BPD (Perez 2007) or BPDSI-IV (Arntz 2003).
3. Chronic feelings of emptiness, assessed by, for example, the relevant item or subscale on the Zan-BPD (Zanarini 2003), CGI-BPD (Perez 2007) or BPDSI-IV (Arntz 2003).
4. Impulsivity, as assessed by, for example, the Barrett Impulsiveness Scale (BIS; Barrett 1995), or the Anger, Irritability and Assault Questionnaire (AIAQ; Coccaro 1991).
5. Interpersonal problems, as assessed by, for example, the Inventory of Interpersonal Problems (IIP; Horowitz 1988), or the relevant item or subscale on the Zan-BPD (Zanarini 2003), CGI-BPD (Perez 2007), BPDSI-IV (Arntz 2003), or SCL-90-R (Derogatis 1994).
6. Abandonment, as assessed by, for example, the relevant item or subscale on the Zan-BPD (Zanarini 2003), CGI-BPD (Perez 2007) or BPDSI-IV (Arntz 2003).
7. Identity disturbance, assessed by, for example, the relevant item or subscale on the Zan-BPD (Zanarini 2003), CGI-BPD (Perez 2007) or BPDSI-IV (Arntz 2003).
8. Dissociation and psychotic-like symptoms, as assessed by, for example, the Dissociative Experience Scale (DES; Bernstein 1986), or the Brief Psychiatric Rating Scale (BPRS; Overall 1962).
9. Depression, as assessed by, for example, the Beck Depression Inventory (BDI; Beck 1961) or the Montgomery Åsberg Depression Rating Scale (MADRS; Montgomery 1979).
10. Attrition, in terms of patients lost after randomisation in each group.
11. Adverse effects, as measured by use of standardised psychometric rating scales such as the Systematic Assessment for Treatment Emergent Events (SAFTEE; Levine 1986), laboratory values or spontaneous reporting. We define adverse events as unfavourable outcomes that occur during or after psychotherapy but are not necessarily caused by it (see Chapter 14 in the *Cochrane Handbook for Systematic Reviews of Interventions*; Loke 2011). We will divide any reported adverse events into severe and non-severe, according to the International Committee of Harmonization guidelines (ICH 1996). Serious adverse events are defined as any event that leads to death, is life-threatening, requires inpatient hospitalisation or prolongation of existing hospitalisation, results in persistent or significant disability, and any important medical event that may have jeopardised the participant's health or requires intervention to prevent it. All other adverse events will be considered non-serious.

Search methods for identification of studies

Electronic searches

We will search the electronic databases and trials registers listed below.

1. Cochrane Central Register of Controlled Trials (CENTRAL; current issue), in the Cochrane Library, which includes the Cochrane Developmental, Psychosocial and Learning Problems Specialised Register
2. MEDLINE Ovid (1948 onwards)
3. Embase Ovid (1980 onwards)
4. CINAHL EBSCOhost (Cumulative Index to Nursing and Allied Health Literature; 1980 onwards)
5. PsycINFO Ovid (1806 onwards)
6. ERIC EBSCOhost (Education Resources Information Center; 1966 onwards)
7. BIOSIS Previews Web of Science Clarivate Analytics (1969 onwards)
8. Web of Science Core Collection Clarivate Analytics (1900 onwards)
9. Sociological Abstracts ProQuest (1952 onwards)
10. LILACS (Latin American and Caribbean Health Science Information database; lilacs.bvsalud.org/en)
11. OpenGrey (www.opengrey.eu)
12. Copac National, Academic and Specialist Library Catalogue (COPAC; copac.jisc.ac.uk)
13. ProQuest Dissertations and Theses A&I (1743 onwards)
14. DART Europe E-Theses Portal (www.dart-europe.eu/basic-search.php)
15. Networked Digital Library of Theses and Dissertations (NDLTD; www.ndltd.org)
16. Australian New Zealand Clinical Trials Registry (ANZCTR; www.anzctr.org.au/BasicSearch.aspx)
17. ClinicalTrials.gov (clinicaltrials.gov)
18. EU Clinical Trials Register (www.clinicaltrialsregister.eu/ctr-search/search)
19. ISRCTN Registry (www.isrctn.com)
20. UK Clinical Trials Gateway (www.ukctg.nihr.ac.uk/#popoverSearchDivId)
21. WHO International Clinical Trials Registry Platform (ICTRP; who.int/ictrp/en)

The search strategy for MEDLINE is in Appendix 1 and we will modify it for other databases using the appropriate syntax and controlled terms. We will not limit our searches by language, year of publication, or type of publication. We will seek translation of the relevant sections of non-English language articles.

Searching other resources

We will handsearch relevant journals, including: the Journal of Personality Disorders; the American Journal of Psychiatry; JAMA Psychiatry; British Journal of Psychiatry; ACTA Psychiatrica Scandinavica; Journal of the American Academy of Child and Adolescent Psychiatry; Personality Disorders: Theory, Research and Treatment; and the Journal of Clinical Psychiatry. Additionally, we will contact researchers working in the field by email to ask for unpublished data. We will also check abstracts of key conferences for BPD (congresses of the European and the International Society for the Study of Personality Disorders; ESSPD and ISSPD respectively) and will ask for any relevant unpublished data. We will trace cross-references from relevant literature.

Data collection and analysis

We will conduct this review according to the guidelines set out in the *Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2011a), and perform analyses using the latest version of Review Manager 5 (RevMan 5), Cochrane's statistical software (Review Manager 2014).

Selection of studies

Six review authors (OJS, JMSW, BAV, JTM, MLK, SSN) will work in three pairs and independently screen titles and abstracts of all records retrieved by the searches; we will resolve uncertainty or disagreement by consensus. For records that could be eligible RCTs, we will obtain the full-text report and assess it for eligibility based on the inclusion criteria (see Criteria for considering studies for this review). The review authors will discuss disagreements, and if they cannot reach an agreement, they will consult a third review author (KL or ES). We will list apparently relevant RCTs that do not fulfil the inclusion criteria with reasons for exclusion in the 'Characteristics of excluded studies' tables. We will use Covidence software to keep track of appraised trials and decisions. To ensure transparency of study selection, we will provide flow charts according to the QUOROM statement, showing how many records have been excluded for a certain reason (Moher 1999).

Data extraction and management

All review authors will extract data. The review authors will work in pairs and will complete the data collection form independently to ensure accuracy. We will resolve disagreements by discussion or by using an arbiter (ES) if required. OJS and JMSW will enter data into RevMan 5 (Review Manager 2014). In those cases where there are not enough data or data are unclear in the published trial reports, we will contact the study authors, requesting them to supply the missing information. We will develop data extraction forms to facilitate standardisation of data extraction.

Assessment of risk of bias in included studies

All review authors will assess the risk of bias in each included study using Cochrane's tool for assessing risk of bias (Higgins 2017). Data extractors will independently assign each trial to one of three categories (low risk of bias, unclear (uncertain) risk of bias or high risk of bias), according to guidelines provided in the *Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2017). We will consider trials with one or more unclear or high risk of bias domains as trials at high risk of bias overall and will define trials that have a low risk of bias in all domains to be at low risk of bias overall. Given the risk of overestimating beneficial intervention effects and underestimating harmful intervention effects in RCTs with unclear or inadequate methodological quality (Kjaergard 2001; Lundh 2012; Moher 1998; Savović 2012a; Savović 2012b; Schulz 1995; Wood 2008), we will also assess the influence of risk of bias on our results (see Sensitivity analysis). We will call upon a third review author (ES) to resolve any ongoing disagreements, if necessary.

Considering bias due to lack of blinding is undoubtedly of importance, but it remains unclear how to best deal with this issue in research practice (Boutron 2008). We have decided not to judge the likelihood of detection bias due to inadequate blinding of patients and personnel, because it is almost impossible to blind therapists and patients in psychological therapy outcome research. We will, however, assess the likelihood of detection bias due to inadequate blinding of outcome assessors.

In accordance with Cochrane's guidelines (Higgins 2017), we have included other potential sources of bias as a final bias component. Here, we will include the likelihood of performance bias due to inadequate treatment adherence, the likelihood of bias due to different amounts of attention given to the treatment groups (attention bias), and other potential sources of bias.

The risk of bias components and the criteria for assigning judgements of low, unclear or high risk of bias are shown in Appendix 4.

Measures of treatment effect

Continuous data

For continuous data, we will compare the mean score between the two groups to give a mean difference (MD) and present this with 95% confidence intervals (CIs). We will use the overall MD, where possible, to compare the outcome measures from trials. We will estimate the standardised MD (SMD) where different outcome measures are used to measure the same construct in the trials. We will calculate SMDs on the basis of post-treatment results and, in separate analyses, follow-up data. We will bundle follow-up data in six-month steps. Where the direction of a scale is opposite to most of the other scales, we will multiply the corresponding mean values by −1 to ensure adjusted values. If the trials do not report means and standard deviations but report other values like t-tests and P values, we will try to transform these into standard deviations.

Dichotomous data

We will summarise dichotomous data as risk ratios (RR) with 95% CIs. We will calculate the risk difference (RD) and the number needed to treat for an additional beneficial outcome (NNTB) or for an additional harmful outcome (NNTH) if there is a significant effect of the intervention.

Unit of analysis issues

Repeated observations

We will calculate study estimates on the basis of post-treatment group results. We will conduct separate analyses for data from different points of measurement (i.e. post-treatment, follow-up data of six-months' intervals, where we will use the last measurement within these intervals). We will not use interim observations.

Cross-over trials

We plan to include data from randomised cross-over studies up to the point of first cross-over (first period only) (Curtin 2002). We will not consider outcomes from subsequent periods due to the likelihood of carry-over effects from the preceding treatment(s). We will not combine repeated participant observations in one meta-analysis.

Cluster-randomised trials

Where trials have used cluster randomisation, we anticipate that investigators will have presented their results after appropriately controlling for clustering effects (robust standard errors or hierarchical linear models). If it is unclear whether a cluster-randomised trial has used appropriate controls for clustering, we will contact the investigators for further information. Where appropriate controls have not been used, we will request and reanalyse individual participant data using multilevel models that control for clustering. Following this, we will analyse effect sizes and standard errors in RevMan 5 (Review Manager 2014), using the generic inverse method (Higgins 2011b). If there is insufficient information to control for clustering, we will enter outcome data into RevMan 5 using individuals as the units of analysis (Review Manager 2014), and then conduct a sensitivity analysis to assess the potential biasing effects of inadequately controlled cluster-randomised trials (Donner 2002); see Sensitivity analysis. If individual participant data are not available, we will look for information on intraclass correlation coefficients to adjust for the potential clustering effects.

Adjustment for multiplicity

We will adjust the P values and CIs for multiplicity due to the many secondary outcome comparisons following the method described by Jakobsen 2014.

Dealing with missing data

We will try to obtain any missing data, including incomplete outcome data, by contacting trial authors. We will report this information in the 'Risk of bias' tables.

We will evaluate the methods used to handle the missing data in the publications and to what extent it was likely that the missing data influenced the results of outcomes of interest. We will calculate effect sizes on the basis of ITT data, it that is possible. If only available case analysis data are reported, we will calculate effect sizes on this basis. Where dichotomous data are not presented on the basis of ITT data, we will add the number of participants lost in each group to the participants with unfavourable results, acting on the assumption that most patients with BPD do not get lost at random.

For continuous outcomes, we will discuss each trial's methodology for dealing with missing continuous data (e.g. last observation carried forward or modified intention-to-treat approach). We will use per protocol analysis, as available from the trial reports (that is, results are based on the number of patients at follow-up).

If data are not reported in an immediately usable way and require processing before being analysed, we will consult a statistician.

We will assess results derived from statistically processed data in sensitivity analyses. See Sensitivity analysis.

Assessment of heterogeneity

We will assess studies for clinical homogeneity with respect to type of therapy, therapy setting and control group. We will evaluate the methodological heterogeneity by comparing the design of trials. For any studies judged as homogeneous and adequate for pooling, we will investigate statistical heterogeneity by both visual inspection of the graphs and the I^2 statistic (Higgins 2003). We will judge I^2 values between 0% and 40% to indicate little heterogeneity, between 30% and 60% to indicate moderate heterogeneity, between 50% and 90% to indicate substantial heterogeneity, and between 75% and 100% to indicate considerable heterogeneity (Deeks 2017). We will also assess statistical heterogeneity using the Chi^2 test ($P < 0.10$) and report tau^2 – an estimate of between-study variance.

We intend to carry out meta-analyses even if there is substantial concern about heterogeneity, but we will interpret the results with caution, and discuss possible reasons and investigate them by conducting subgroup analyses. See Subgroup analysis and investigation of heterogeneity.

Assessment of reporting biases

We will provide funnel plots for comparisons with sufficient primary studies and we will perform Egger's statistical test for small-study effects (Egger 1997). We will only use funnel plots if there are 10 or more studies in the meta-analysis, as recommended in the *Cochrane Handbook for Systematic Reviews of Interventions* (Sterne 2017).

Data synthesis

We will perform statistical analysis according to recommendations in the latest version of the *Cochrane Handbook for Systematic Reviews of Interventions* (Deeks 2017). In carrying out the meta-analysis, we will use the inverse-variance method, to give more weight to more precise estimates from studies with less variance (mostly larger studies). This minimises the imprecision of the pooled effect estimate, and it is a common and simple approach to conducting a meta-analysis (Deeks 2017). We will divide the doses and the controls into different comparisons, ensuring that the treatment comparisons will be comparable and homogeneous. We will use the random-effects model for meta-analysis, since we expect some degree of clinical heterogeneity to be present in most cases, though not too substantial to prevent pooling in principle. For trials with a high level of statistical heterogeneity, and where the amount of clinical heterogeneity makes it inappropriate to use these trials in meta-analyses, we will provide a narrative description of the trial results. If data pooling seems feasible, we will pool the primary studies' effects and calculate their 95% CIs. If a trial provides more than one measure for the same outcome construct (e.g. several questionnaires for the assessment of depression), we will select the one used most often in the whole pool of included studies for effect size calculation, in order to minimise heterogeneity of outcomes in form and content. If a study reports data of two assessment instruments that are equally frequently used, two review authors will discuss the issue and choose the one which is, in its content, most appropriate for assessing BPD patients. We would prefer to use observer-rated measures as the primary analysis.

Considering the various types of interventions identified by the previous version of this review (Stoffers-Winterling 2012), we will arrange the results according to classes of interventions, defined as follows.

1. Individual psychotherapy
2. Group psychotherapy
3. Family therapy
4. Any combination of individual, family or group psychotherapy

Psychotherapy will be defined as "treatment of mental illness or emotional disturbances primarily by verbal or nonverbal communication" (NLM 2009).

Subgroup analysis and investigation of heterogeneity

We will conduct a subgroup analysis to make hypotheses about the subgroups mentioned below.

1. Therapeutic schools (e.g. CBT, psychodynamic, psychoeducation)
2. Age (15 to under 18 years of age, 18 to 50 years of age, above 50 years of age)

3. Sex (male versus female)
4. Comorbidity (patients with comorbidity versus patients without comorbidity)
5. Treatment intensity (once a week compared to more than once a week)
6. Duration (less than 6 months, 6 to 12 months and over 12 months)
7. Mode of therapy (individual compared to group therapy; mixed therapy (i.e. a combination of individual and group therapy, or a therapy not akin to individual or group therapy, e.g. family therapy) compared to individual therapy; and mixed therapy compared to group therapy
8. Setting (outpatient compared to inpatient)
9. Concurrent-drug interventions (trials with concurrent-drug interventions compared to those without)

Heterogeneity-adjusted required information size and Trial Sequential Analysis

Trial Sequential Analysis (TSA) is a methodology that combines a required information size (RIS) calculation for a meta-analysis with the threshold for statistical significance (Brok 2008; Brok 2009; Thorlund 2009; Wetterslev 2008). TSA is a tool for quantifying the statistical reliability of the data in a cumulative meta-analysis, adjusting P values for sparse data and for repetitive testing on accumulating data (Brok 2008; Brok 2009; Thorlund 2009; Wetterslev 2008).

Comparable to the a priori sample size estimation in a single randomised trial, a meta-analysis should include an RIS calculation at least as large as the sample size of an adequately powered single trial to reduce the risk of random error. TSA calculates the RIS in a meta-analysis and provides an alpha-spending boundary to adjust the significance level for sparse data and repetitive testing on accumulating data (CTU 2011; Wetterslev 2008); hence the risk of random error can be assessed. Multiple analysis of accumulating data when new trials emerge leads to repeated significant testing and hence introduces multiplicity. Thus use of a conventional P value is prone to exacerbate the risk of random error (Berkey 1996; Lau 1995). Meta-analyses not reaching the RIS are analysed with trial sequential alpha-spending monitoring boundaries analogous to interim monitoring boundaries in a single trial (Wetterslev 2008). This approach will be crucial in coming updates of the review. We will calculate an RIS on all outcomes in the review. If a TSA does not reveal significant findings (no crossing of the alpha-spending boundary and no crossing of the conventional boundary of P = 0.05) before the RIS has been reached, then the conclusion should either be that more trials are needed to reject or accept an intervention effect that was used for calculation of the required sample size or - in case the cumulated Z-curve enters the futility area - the anticipated effect can be rejected. We will calculate the a priori diversity-adjusted required information size (that is, the number of patients required to detect or reject a specific intervention effect in the meta-analysis), and perform a TSA for the primary outcomes based on the following a priori assumptions.

1. The standard deviation of the primary outcome is 1.0
2. An anticipated intervention effect equal to Hedge's g of 0.5
3. A maximum type I error of 5% (alpha)
4. A maximum type II error of 20% (beta; equal to a minimum 80% power)
5. A priori anticipated 50% diversity (Brok 2008; Brok 2009; Thorlund 2009; Wetterslev 2008; Wetterslev 2009).

We will also calculate a post hoc, low bias, risk diversity-adjusted required information size (that is, the number of patients required to detect or reject a specific intervention effect in the meta-analysis), and perform a TSA for the primary outcomes based on the following estimated assumptions.

1. The standard deviation of the primary outcome in patients in the control group of trials with low risk of bias
2. The estimated intervention effect in trials with low risk of bias
3. A maximum type I error of 5% (alpha)
4. A maximum type II error of 20% (beta; equal to a minimum 80% power)
5. The estimated diversity in the trials included in the meta-analysis (Brok 2008; Brok 2009; Thorlund 2009; Wetterslev 2008)

'Summary of findings' tables

We will use the GRADE approach to construct a 'Summary of findings' table in which to document all review outcomes. The GRADE approach appraises the quality of a body of evidence based on the extent to which one can be confident that an estimate of effect or association reflects the item being assessed. Considerations are due to: within-trial risk of bias; directness of the evidence; heterogeneity of the data; precision of effect estimates; and risk of publication bias (Andrews 2013a; Andrews 2013b; Balshem 2011; Brunetti 2013; Guyatt 2011a; Guyatt 2011b; Guyatt 2011c; Guyatt 2011d; Guyatt 2011e; Guyatt 2011f; Guyatt 2011g; Guyatt 2011h; Guyatt 2013a; Guyatt 2013b; Guyatt 2013c; Mustafa 2013). When possible, we will use the MD or the RR, and we will use Trial Sequential Analysis (TSA) to rate the imprecision (Jakobsen 2014). We will report the four primary outcomes (BPD severity, self-harm, suicide-related outcomes and functioning) and the three secondary outcomes (interpersonal problems, attrition, and adverse events) in a 'Summary of findings' table for the main comparison (Atkins 2004).

Sensitivity analysis

We will assess the impact of heterogeneity on the overall pooled effect estimate by removing studies ('outliers') that are contributing to the heterogeneity. We will remove outliers one by one and assess the impact on the overall outcome.

We will conduct sensitivity analyses to determine whether findings are sensitive to the following.

1. Decisions made during the review process (our assessment of the level of clinical heterogeneity)
2. Impact of bias (studies with low and high risk of bias)
3. Type of model used for analysis (repeating the analysis using the fixed-effect model to test the robustness of the results)
4. Type of data collection (for example, different ways to measure depression)
5. Imputed data (comparing analyses with available outcome data with those using an intention-to-treat (ITT) approach)

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* *Indicates the major publication for the study*

APPENDICES

Appendix 1. DSM diagnostic criteria for BPD (301.83)

DSM Third Edition (DSM-III; APA 1980) **301.83 BPD**	DSM Fourth Edition Text Revision (DSM-IV-TR; APA 2000) **301.83 BPD**	DSM Fifth Edition (DSM-5; APA 2013) **301.83 BPD**
Diagnostic criterion A 5 of the following are required 1. Impulsivity or unpredictability in at least	**Diagnostic criterion A** A pervasive pattern of instability of interpersonal relationships, self-image, and af-	**Diagnostic criterion A** A pervasive pattern of instability of interpersonal relationships, self-image, and af-

(*Continued*)

2 areas that are potentially self-damaging (e.g. spending, sex, substance use, shoplifting, overeating, physically self-damaging acts) 2. A pattern of unstable and intense interpersonal relationships (e.g. marked shifts of attitude, idealization, devaluation, manipulation (consistently using others for one's own ends)) 3. Inappropriate, intense anger or lack of control of anger (e.g. frequent displays of temper, constant anger) 4. Identity disturbance manifested by uncertainty about several issues relating to identity, such as self-image, gender identity, long-term goals or career choice, friendship patterns, values, and loyalties (e.g. 'Who am I', 'I feel like I am my sister when I am good') 5. Affective instability, marked shifts from normal mood to depression, irritability or anxiety, usually lasting a few hours and only rarely more than a few days, with a return to normal mood 6. Intolerance of being alone (e.g. frantic efforts to avoid being alone, depressed when alone) 7. Physically self-damaging acts (e.g. suicidal gestures, self-mutilation, recurrent accidents or physical fights) 8. Chronic feelings of emptiness or boredom	fects, and marked impulsivity beginning by early adulthood and present in a variety of contexts, as indicated by 5 (or more) of the following 1. Frantic efforts to avoid real or imagined abandonment (note: do not include suicidal or self-mutilating behavior, which is covered in criterion 5) 2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation 3. Identity disturbance: markedly and persistently unstable self-image or sense of self 4. Impulsivity in at least 2 areas that are potentially self-damaging (e.g. spending, sex, substance abuse, reckless driving, binge eating) (note: do not include suicidal or self-mutilating behavior, which is covered in criterion 5) 5. Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior 6. Affective instability due to a marked reactivity of mood (e.g. intense episodic dysphoria, instability, or anxiety usually lasting a few hours and only rarely more than a few days) 7. Chronic feelings of emptiness 8. Inappropriate, intense anger or difficulty controlling anger (e.g. frequent displays of temper, constant anger, recurrent physical fights) 9. Transient, stress-related paranoid ideation or severe dissociate symptoms	fects and marked impulsivity, beginning by early adulthood and present in a variety of contexts, as indicated by 5 (or more) of the following 1. Frantic efforts to avoid real or imagined abandonment (note: do not include suicidal or self-mutilating behavior, which is covered in criterion 5) 2. A pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation 3. Identity disturbance: markedly and persistently unstable self-image or sense of self 4 Impulsivity in at least 2 areas that are potentially self-damaging (e.g. spending, sex, substance abuse, reckless driving, binge eating) (note: do not include suicidal or self-mutilating behavior, which is covered in criterion 5) 5. Recurrent suicidal behavior, gestures or threats, or self-mutilating behavior 6. Affective instability due to a marked reactivity of mood (e.g. intense episodic dysphoria, irritability or anxiety of mood) usually lasting a few hours and only rarely more than a few days 7. Chronic feelings of emptiness 8. Inappropriate, intense anger or difficulty controlling anger (e.g. frequent displays of temper, constant anger, recurrent physical fights) 9. Transient, stress-related paranoid ideation or severe dissociative symptoms
Diagnostic criterion B If under 18, does not meet the criteria for Identity Disorder		

BPD: Borderline personality disorder; DSM: Diagnostic and Statistical Manual of Mental Disorders

Appendix 2. ICD-10 research criteria for emotionally unstable personality disorder (F60.3)

F60.30: ICD-10 Emotionally unstable personality disorder, impulsive type	F60.31: Emotionally unstable personality disorder, borderline type
Diagnostic criterion A The general criteria of personality disorder (F60) must be met	**Diagnostic criterion A** The general criteria of personality disorder (F60) must be met
Diagnostic criterion B At least 3 of the following must be present, 1 of which is 2 1. Marked tendency to act unexpectedly and without consideration of the consequences 2. Marked tendency to quarrelsome behaviour and to conflicts with others, especially when impulsive acts are thwarted or criticized 3. Liability of outbursts of anger or violence, with inability to control the resulting behavioural explosions 4. Difficulty in maintaining any course of action that offers no immediate reward 5. Unstable and capricious mood	**Diagnostic criterion B** At least 3 of the symptoms mentioned above in criterion B (F60.30) must be present, and, in addition, at least 2 of the following 6. Disturbances in, and uncertainty about, self-image, aims and internal preferences (including sexual) 7. Liability to become involved in intense and unstable relationships, often leading to emotional crises 8. Excessive efforts to avoid abandonment 9. Recurrent threats or acts of self-harm 10. Chronic feelings of emptiness
ICD-10: International Classification of Diseases, Tenth Edition	

Appendix 3. Medline search strategy

1 Borderline Personality Disorder/
2 ((borderline or border-line) adj3 (state* or personalit*)).kf,tw.
3 ("Axis II" or "Cluster B" or flamboyant or "F60.3" or "F60.30" or "F60.31").kf,tw.
4 (idealization adj5 devaluation).kf,tw.
5 ((vulnerable or hyperbolic) adj3 temperament).kf,tw.
6 (((unstab* or instab* or poor or disturb* or fail* or weak or dysregulat*) adj3 (self* or impuls* or interperson* or identit* or relationship* or emotion* or affect*)) and (personality or character or PD)).kf,tw.
7 (impulsiv* adj5 (behavio?r or character or personalit*)).kf,tw.
8 (self adj3 (injur* or damag* or destruct* or harm* or hurt* or mutilat*)).kf,tw.
9 (suicidal adj3 behavio?r).kf,tw.
10 (feel* adj3 (empt* or bored*)).kf,tw.
11 (anger adj5 control*).kf,tw.
12 (risk-taking adj3 behavio?r).kf,tw.
13 or/1-12
14 randomized controlled trial.pt.
15 controlled clinical trial.pt.
16 randomi#ed.ab.
17 placebo.ab.
18 randomly.ab.
19 trial.ab.
20 groups.ab.
21 drug therapy.fs.
22 or/14-21
23 exp Animals/ not Humans/
24 22 not 23

25 13 and 24

Appendix 4. 'Risk of bias' components and criteria for assigning judgements

Selection bias

Random sequence generation

1. **Low risk of bias**. The method used was adequate (e.g. computer-generated random numbers, table of random numbers) or was unlikely to introduce selection bias.
2. **Unclear risk of bias**. Information was insufficient for assessment of whether the method used could introduce selection bias.
3. **High risk of bias**. The method used was likely to introduce bias.

Allocation concealment

1. **Low risk of bias**. The method used (e.g. central allocation) was unlikely to bias allocation to groups.
2. **Unclear risk of bias**. Information was insufficient for assessment of whether the method used could bias allocation to groups.
3. **High risk of bias**. The method used (e.g. open random allocation schedule) could bias allocation to groups.

Detection bias: Blinding of outcome assessment

1. **Low risk of bias**. The method of blinding was described and blinding was conducted in a satisfactory way.
2. **Unclear risk of bias**. Information was insufficient for assessment of whether the type of blinding used was likely to bias the estimate of effect.
3. **High risk of bias**. No blinding or incomplete blinding.

Aattrition bias: Incomplete outcome data

1. **Low risk of bias**. Underlying reasons for missing data probably would not affect outcome measurement, as all missing data can be considered as missing at random or all data were reported.
2. **Unclear risk of bias**. Information was insufficient for assessment of whether missing data or the method used to handle missing data was likely to bias the estimate of effect.
3. **High risk of bias**. The crude estimate of effects could be biased given the reasons for the missing data, or the methods used to handle missing data are unsatisfactory.

Reporting bias: Selective outcome reporting

1. **Low risk of bias**. The trial protocol was available and all pre-specified outcomes of interest were reported.
2. **Unclear risk of bias**. Information was insufficient for assessment of whether selective outcome reporting could have occurred.
3. **High risk of bias**. Not all of the primary outcomes specified beforehand were reported or participants were excluded after randomisation.

Other potential sources of bias

Treatment adherence bias

1. **Low risk of bias**. Means were undertaken to assure adequate treatment adherence; for example, by regular supervision or use of adherence ratings of videotaped or audio-taped therapy sessions.
2. **Unclear risk of bias**. Insufficient information to assess the extent of adequate treatment adherence.
3. **High risk of bias**. Inadequate treatment adherence. No means were undertaken to assure adequate treatment adherence.

Attention bias

1. **Low risk of bias**. The treatment conditions were sufficiently similar in duration and intensity.
2. **Unclear risk of bias**. Insufficient information in regards to treatment duration and intensity.
3. **High risk of bias**. One treatment condition was markedly more intense or was of longer duration than other condition(s).

Affiliation bias

1. **Low risk of bias**. Principal investigator is not the developer of the treatment under investigation (if compared to a control condition), or both treatment developers are involved if two treatments are directly compared.
2. **Unclear risk of bias**. Insufficient information to assess affiliation bias.
3. **High risk of bias**. Principal investigator is developer of the treatment under investigation (if compared to a control condition), or only one of the treatment developers is involved if two treatments are directly compared.

Other sources of bias

1. **Low risk of bias**. The trial appeared to be free of other sources of bias.
2. **Unclear risk of bias**. Information was inadequate for assessment of other possible sources of bias.
3. **High risk of bias**. Other sources of bias were identified.

CONTRIBUTIONS OF AUTHORS

All authors contributed to writing this protocol. Ole Jakob Storebø is the guarantor for the review.

DECLARATIONS OF INTEREST

Jutta M Stoffers-Winterling is a board-certified psychologist (CBT), who has worked on a Dialectical Behaviour Therapy (DBT) ward, and attended courses on DBT and Schema-focused therapy (SFT).

Ole Jakob Storebø is an Editor with CDPLP. He is involved in a trial investigating group mentalization-based treatment (MBT) for adolescents with BPD.

Jessica T Mattivi's institution received a grant from the Federal Ministry for Education and Research (BMBF) for a systematic review on psychosocial interventions for self-harm in adolescents.

Birgit A Völlm - none known.

Mickey Kongerslev is a certified specialist in psychotherapy from the Danish Psychological Association. He has received training in group analysis, CBT, and MBT.

Mie Poulsgaard Jørgensen - none known.

Signe Nielsen - none known.

Maja Lærke Kielsholm - none known.

Erlend G Faltinsen - none known.

Klaus Lieb (KL) is an Editor with CDPLP. He is a board-certified cognitive behaviour therapist with a special interest in schema therapy. KL has been involved in trials investigating inpatient DBT (Bohus 2004); and inpatient schema-focused therapy (Reiss 2014). KL's institution received a grant from the German Federal Ministry of Education and Research for this review.

Erik Simonsen is a board-certified therapist in group analysis.

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External sources

- None, Other.

NOTES

This is a new protocol for a new review, which will replace the current published review: Stoffers JM, Völlm BA, Rücker G, Timmer A, Huband N, Lieb K. Psychological therapies for people with borderline personality disorder. Cochrane Database of Systematic Reviews 2012, Issue 8. Art. No.: CD005652. DOI: 10.1002/14651858.CD005652.pub2.