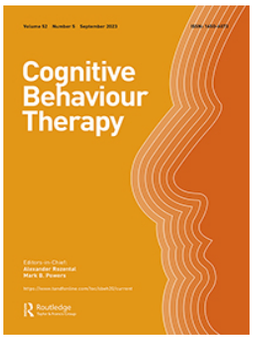

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Cognitive Behaviour Therapy (CBT) for Depersonalization Derealization Disorder (DDD): a self-controlled cross-over study of waiting list vs. active treatment

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ABSTRACT

Depersonalisation-Derealisation Disorder (DDD) has a prevalence of around 1% but is under-recognised and often does not respond to medical intervention. We report on a clinical audit of 36 participants with a diagnosis of chronic DDD who were sequentially recruited from a specialist DDD National Health Service clinic in London, United Kingdom, and who completed Cognitive Behavioural Therapy specifically adapted for DDD. The sample population had a mean age of 38.7 years (s.d. = 13.4), 61% were male and 69% were of White ethnicity. Three outcomes were assessed (Cambridge Depersonalisation Scale [CDS], Beck Depression Inventory [BDI], and the Beck Anxiety Inventory [BAI]) at three time points in a naturalistic, self-controlled, cross-over design. Hierarchical longitudinal analyses for outcome response clustered by patient were performed using scores from baseline, beginning, and end of therapy. All scores showed improvement during the treatment period, with medium effect sizes. CBT may be an effective treatment for DDD. However, treatment was not randomly assigned, and the sample was small. More research is needed, including the use of randomisation to assess the efficacy of CBT for DDD.

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Depersonalisation; derealisation; dissociation; CBT

Introduction

Depersonalisation-Derealisation Disorder (DDD) is classified as a dissociative disorder within diagnostic guidelines (DSM-5: American Psychiatric Association [APA], 2013). As the name implies, the disorder is comprised of two separate syndromes: *depersonalisation (DP)* and *derealisation (DR)*, although in most cases people experience both concurrently (Baker et al., 2003). With *depersonalisation* people report a subjective experience of feeling unreal, in a dream-like state, numbed and/or of being disconnected from their own internal processes, such as the felt sense of emotions, thoughts, memory, or body. *Derealisation* is where the sense of disconnection and unreality refers to the

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outside world in that a person's subjective perception of the environment feels unfamiliar and distorted (APA, 2013).

Transient, mild symptoms of both syndromes are common in the general population, triggered by factors such as fatigue, stress, and bereavement, with one survey finding 23% of those questioned reporting these experiences over the course of 1 year (Aderibigbe et al., 2001). However, for some people, the symptoms become chronic, cause functional impairment and distress, and meet criteria for a clinical diagnosis of DDD. Estimates of the prevalence of DDD in community samples across different cultural settings show a consistent rate of around 1% (Yang et al., 2022). Moreover, higher prevalence rates have been found in many patient populations (e.g. anxiety disorders and depression), where DDD symptoms may often be secondary to other conditions or exist as a co-morbid diagnosis (Hunter et al., 2004; Yang et al., 2022).

Despite depersonalisation symptoms first being described in 1898 when the term was coined (Sierra & Berrios, 1997), there has been a paucity of research. With no specific medication for the condition, a general lack of awareness of both the condition and treatment options, prognosis has been generally pessimistic. One of the key questions for researchers and clinicians is why are some experiences of depersonalisation (DP) or derealisation (DR) transient, whereas others become chronic? From a cognitive behavioural perspective, a potential answer is that a vicious cycle might be initiated which serves to maintain the symptoms. In a similar way to how the CBT model of panic describes how a single panic attack can develop into a panic disorder through unhelpful cognitions and behaviours, as well as physiological and emotional reactions (Clark, 1986), a comparable process may explain how transient depersonalisation and/or derealisation symptoms become chronic DDD. The cognitive behavioural model of DDD (Hunter et al., 2003) proposed that a vicious cycle of DDD may include catastrophic beliefs (such as "I'm losing touch with reality and going insane" and "I've damaged my brain with drugs"), thinking biases and processes (such as symptom focused attention and rumination), negative emotions (particularly anxiety), and behavioural changes (such as social avoidance and checking behaviours).

Empirical testing of the CBT model of DDD has validated these theoretical hypotheses (Hunter et al., 2014). When compared to a healthy control group, the DDD group were more likely to self-generate catastrophic, rather than normalising attributions for a range of common benign symptoms. Moreover, interrupting the process of symptom focused attention using stress-inducing experimental tasks with high cognitive demands resulted in a reduction of DDD symptoms, whereas healthy control and anxiety disorder groups reported an increase in depersonalisation/derealisation symptoms post-task.

The CBT model of DDD (Hunter et al., 2003) led to the first evaluation of the potential effectiveness of cognitive behavioural therapy for the condition (Hunter et al., 2005). In this study, 21 consecutive referrals of patients over a two-year period who were diagnosed with DDD by two psychiatrists using a semi-structured clinical interview (Present State Examination: Wing et al., 1974) were offered individual CBT for DDD, with a mean of 13 sessions. There were an additional four participants who met the exclusion criteria (n = 3) or dropped out of the study (n = 1). The results showed statistically significant improvements after intervention on two primary measures of, respectively, DDD (Cambridge Depersonalisation Scale: CDS, Sierra & Berrios, 2000); and general dissociation (Dissociative Experience Scale: DES, Bernstein & Putnam, 1986). The effect sizes

(estimated from the post-estimation eta-squared values) for these two outcomes were 0.37 (95% CI: 0.12 to 0.53) and 0.34 (95% CI: 0.10 to 0.51), respectively. In addition, there were statistically significant improvements on other clinical measures of depression (Beck Depression Inventory: BDI, Beck et al., 1961), anxiety (Beck Anxiety Inventory: BAI, Beck et al., 1988), and general functioning (Work and Social Adjustment Scale, Mundt et al., 2002). The effect sizes for these outcomes were 0.58 (95% CI: 0.35 to 0.70), 0.30 (95% CI: 0.07 to 0.47), and 0.49 (95% CI: 0.22 to 0.64), respectively. These results were encouraging given the duration of DDD in the sample (mean duration of 14 years) and over 60% of the sample also meeting criteria for moderate-severe depression or anxiety in addition to their DDD.

Since the 2005 paper, there have been three studies using adaptations of the Hunter et al. (2005) CBT for DDD protocol (Farrelly et al., under review; Flückiger et al., 2022) or a transdiagnostic CBT protocol (Mohajerin et al., 2020) which have shown promising results in reducing DDD/Dissociative Identity Disorder symptoms. Farrelly et al. (under review) examined the feasibility and acceptability of a brief, six-session therapy protocol adapted from the CBT for DDD model in individuals with both DDD and psychotic symptoms. This single-blind, randomised controlled trial of 21 participants found it both feasible and acceptable to participants, as well as showed promising results in that CDS mean total scores in the intervention group reduced at follow-up while scores in the control group increased. Flückiger et al. (2022) conducted a pilot study of an adaptation of the CBT for DDD model for use as an 8-session group therapy programme for adolescent and young adult outpatients. They found a significant reduction in CDS scores 6 months after the start of the programme, with the sessions on identifying and modifying dysfunctional assumptions about DDD symptoms rated the highest by the participants. However, the sample size was extremely small ($n = 8$), and there was no control group for comparison. Similarly, a case series of five patients with DID, where depersonalisation and derealisation symptoms were part of their presentations, showed significant improvements in dissociative symptoms (measured by the DES), anxiety, and depression, with none meeting criteria for DID at 6-month follow-up when treated with a transdiagnostic CBT protocol (Mohajerin et al., 2020).

However, all these studies have similar limitations of small sample size. Moreover, the data from the Hunter et al. (2005) study did not provide sufficient precision on which to base a sample size calculation for a subsequent adequately powered study. The uncertainty in the estimates of the treatment effects resulted in potential sample sizes that were unrealistic. We therefore conducted a subsequent audit (presented here) using a self-controlled cross-over design, which increases the power of the statistical analyses as the intraparticipant variability is reduced. Our hypothesis was that CBT is clinically effective for the treatment of DDD. We have used the results of this interim study to gain funding for a feasibility RCT which we will report in a subsequent communication.

Methods

Participants and sampling

Participants were consecutive referrals to a public health system (UK National Health Service) specialist Depersonalisation-Derealisation Disorder clinic over

a period of 4 years and 7 months. To obtain funding for a referral to this tertiary clinic, participants had already been given a primary diagnosis of DDD by their local referrer, but in addition all participants were assessed by the first author who is a consultant clinical psychologist (ECMH) experienced in DDD to ensure they all met diagnostic criteria for DDD according to DSM-5. All participants gave informed consent to be included in the research.

In total there were 50 participants who were eligible for treatment and added to the waiting list. However, 14 of these were not included in the final analysis. The reasons for their exclusion were as follows: only seen for assessment and not funded for therapy ($n = 1$); dropped out before intervention sessions were conducted, i.e. before session 4 ($n = 4$); referred to another more relevant service to complete therapy (e.g. trauma/alcohol services) ($n = 3$); only funded for initial six sessions ($n = 4$); and dropped out of therapy after some intervention but missing end of therapy data ($n = 2$). There were no significant differences between those who completed therapy and those who were included in the final analysis ($n = 36$) and those who did not complete therapy and were excluded ($n = 14$) in terms of baseline demographic or baseline characteristics.

Participants who had completed eight or more therapy sessions were included in this study as this was deemed a sufficient "dose" to have potentially had an effect. This was based on several factors. Although UK guidelines for CBT for mild-moderate conditions indicate that the typical number of therapy sessions might be between 5 and 8, the recommended number of CBT sessions for conditions of similar severity to the DDD sample here, such as moderate to severe depression is 16 (National Institute for Health and Care Excellence [NICE], 2022). The mean number of sessions in the 2005 study which showed potential effectiveness of CBT for DDD was 13. Moreover, as the initial phase of three to four sessions of individualised, formulation-driven CBT (such as CBT for DDD) was not focused on intervention but comprised of extended assessment, psychoeducation, and shared formulation, it was unlikely that those attending fewer than eight sessions would have had sufficient time on CBT interventions to be able to attribute any change to the intervention. Most participants were funded for 16 sessions of treatment. However, some completed within a smaller number of sessions, and some had additional sessions requested, and subsequently, funded. At the time of analysis, 31 clients (86%) had completed their course of therapy. An additional five clients (14%) were included who had partially completed their course of therapy as we had requested further sessions, but these had not been delivered at the time of analysis. The mean number of CBT sessions was 18.1 (range 8–40).

In addition to DDD, 28 clients (78%) had at least one co-morbid disorder, 10 (28%) had major depressive disorder, 4 (11%) had one or more anxiety disorders, 1 (3%) had post-traumatic stress disorder, 1 (3%) had obsessive-compulsive disorder and agoraphobia, 10 (28%) had major depressive disorder and an anxiety disorder, 3 (8%) had major depressive disorder, GAD, and a psychosis spectrum disorder, and 1 (3%) had major depressive disorder, GAD, and alcohol misuse. Descriptive statistics of demographic and clinical characteristics at baseline in those who were excluded from the final analysis ($n = 14$), those who completed therapy ($n = 36$), and the total sample ($n = 50$) are shown in Table 1.

Table 1. Table of baseline demographic and clinical characteristics.

	Included in Analysis					
	No *N* = 14 (28.0%)		Yes *N* = 36 (72.0%)		Total *N* = 50 (100%)	
Demographic characteristics	n	(%)	n	(%)	n	(%)
Age/Years						
[20–28)	3	(21.4%)	7	(19.4%)	10	(20.0%)
[28–33)	3	(21.4%)	7	(19.4%)	10	(20.0%)
[33–40)	2	(14.3%)	8	(22.2%)	10	(20.0%)
[40–50)	5	(35.7%)	5	(13.9%)	10	(20.0%)
[50–76]	1	(7.1%)	9	(25.0%)	10	(20.0%)
Sex						
Male	12	(85.7%)	22	(61.1%)	34	(68.0%)
Female	2	(14.3%)	14	(38.9%)	16	(32.0%)
Ethnicity						
White	11	(78.6%)	25	(69.4%)	36	(72.0%)
Other	3	(21.4%)	11	(30.6%)	14	(28.0%)
Employment Status						
Professional	0	(0.0%)	5	(13.9%)	5	(10.0%)
Skilled	5	(35.7%)	4	(11.1%)	9	(18.0%)
Semi-skilled	1	(7.1%)	5	(13.9%)	6	(12.0%)
Unskilled	0	(0.0%)	1	(2.8%)	1	(2.0%)
Student	3	(21.4%)	6	(16.7%)	9	(18.0%)
Retired	0	(0.0%)	3	(8.3%)	3	(6.0%)
Unemployed	5	(35.7%)	12	(33.3%)	17	(34.0%)
DDD characteristics	Mean	(sd)	Mean	(sd)	Mean	(sd)
Clinical characteristics						
Duration of DDD	17.14	(12.15)	14.92	(15.18)	15.54	(14.31)
Age of onset of DDD	18.21	(6.77)	23.75	(10.01)	22.20	(9.49)
Comorbidities*						
No comorbid disorders	1	(7.1%)	8	(22.2%)	9	(18.0%)
Major depressive disorder	2	(14.3%)	10	(27.8%)	12	(24.0%)
One or more anxiety disorders	0	(0.0%)	4	(11.1%)	4	(8.0%)
MDD & Anxiety	8	(57.1%)	10	(27.8%)	18	(36.0%)
MDD, GAD & Psychosis Spectrum	1	(7.1%)	3	(8.3%)	4	(8.0%)
MDD, GAD & Alcohol	1	(7.1%)	1	(2.8%)	2	(4.0%)
MDD, GAD & PD	1	(7.1%)	0	(0.0%)	1	(2.0%)

*MDD = Major Depressive Disorder, GAD = Generalised Anxiety Disorder, PD = Personality Disorder.

Therapy process

Following assessment, participants were placed on a waiting list for CBT for DDD, whilst continuing to receive treatment as usual from their local services. When therapy started, participants were offered weekly or fortnightly (depending on how often they could attend) individual CBT for DDD therapy sessions. All therapies were delivered by the first author (ECMH) as she was the only clinical psychologist employed by the service during that time. She was the clinical lead of the service and had undertaken post-graduate CBT training and developed the CBT for DDD treatment protocol used in the 2005 study. To evaluate therapist adherence to the CBT for DDD protocol, clinical summaries of the content of each session were coded and rated by the first and second authors (ECMH and CLMW). Inter-rater reliability was high, with a correlation coefficient of 0.96 and Cronbach's alpha of 0.98.

CBT for DDD intervention

In CBT, each disorder has its own specific model that identifies the content and processes that need to be addressed in the treatment of that condition. This specificity increases the efficacy of the intervention. The CBT model of DDD and earlier intervention study (Hunter, 2013; Hunter et al., 2003, 2005) includes an initial phase of psychoeducation about DDD, extended assessment via diary keeping, and the development of a shared, individualised understanding with the client of the predisposing and precipitating factors that led to the onset of their DDD, as well as the factors that are currently perpetuating these symptoms. Through this process, the person with DDD can understand how their depersonalisation-derealisation response is a psychological mechanism to help them from feeling overwhelmed, by creating a sense of detachment from reality, as well as numbing their cognitive, emotional, and physiological response. This initial phase of psychoeducation and shared formulation may comprise the first 3–4 sessions.

The second phase of intervention includes working with cognitive content, cognitive processes, behavioural change, and emotion regulation. During these sessions, the therapist helps the client to identify and restructure catastrophic thought content regarding the meaning and consequences of DDD such as: "I am losing touch with reality and going mad; I have damaged my brain through taking drugs; there is no treatment for this condition so I will never get better". The therapist helps the client to develop more factual and helpful alternative beliefs through reviewing the evidence for and against their catastrophic thoughts. This will lessen their fear of DDD symptoms and help to break their maintenance cycle. Moreover, CBT for DDD addresses typical cognitive processes seen in DDD, such as excessive researching about DDD, symptom monitoring, and self-focused, attention, as well as worry and rumination about the symptoms. It also identifies common patterns of behaviour which serve to perpetuate symptoms such as avoidance. Other CBT for DDD interventions includes training the client in the use of "grounding strategies", to increase a sense of connection and present moment awareness, as well as learning emotional regulation skills. CBT interventions for any co-morbid disorders that are formulated to be maintaining the DDD are also included in the treatment package. This second phase of intervention will comprise most of the therapy sessions. With individualised, formulation-driven CBT, the therapist is given flexibility within the protocol to determine optimal targets and the order for these interventions according to the needs of the specific client. The third and final phases of therapy includes a review and summary of what has been learnt to create a staying well plan for the client. This final phase typically takes 1–2 sessions. For the core and optional interventions included in CBT for DDD see Figure 1.

Clinical measures

Clients were asked to complete three standardised self-report clinical questionnaires at three time points: assessment, start of therapy, and end of therapy. The latter was determined according to when treatment was deemed to be completed, rather than a set number of sessions or a fixed time point.

Cambridge Depersonalisation Scale: *Trait (CDS-Trait)* (Sierra & Berrios, 2000) is the primary outcome measure and the only validated measure specifically for DDD. The CDS is a 29-item questionnaire that measures the frequency on a scale of (0–4) and

***Phase 1:* Psychoeducation / shared formulation**

- Psychoeducation about depersonalisation/derealisation and the function of these symptoms
- Keeping a DDD severity diary over 1-2 weeks to analyse factors contributing to fluctuations in severity of symptoms.
- Building individualised CBT shared formulations for their current DDD symptoms, including predisposing and precipitating factors.

Phase 2: CBT Interventions

Cognitive

- Identifying unhelpful, catastrophic, thoughts about depersonalisation/derealisation
- Cognitive restructuring by reviewing the evidence for and against unhelpful DDD related thoughts

Thinking processing

- Psychoeducation about common thinking biases
- Understanding how self-focussed attention can maintain DDD and alternative strategies
- Reducing hyper-vigilance / symptom monitoring / checking behaviours

Behavioural

- Psychoeducation about safety seeking behaviours
- Behavioural experiments to test out beliefs
- Graded exposure to avoided situations

Emotion regulation

- Examining the role of emotions associated with DDD
- Identifying anxiety/ distress management strategies
- Psychoeducation about grounding strategies and practice of these

Phase 3: *Review and relapse prevention*

- Summary of what has been learnt from the sessions
- Depersonalisation/derealisation staying well plan

Optional Interventions

- CBT for co-morbid disorders maintaining DDD such as anxiety disorders, depression, low self-esteem, perfectionism
- Trauma focused CBT
- Chair work to understand the function of their DDD
- Third wave CBT interventions

NB: number and type of components delivered dependent upon individual formulation

Figure 1. Core and optional interventions in CBT for DDD.

duration on a scale of (0–6) of a range of DDD symptoms. Item scores are summed to a score of 10 for each item, with a total score of 290. This scale has high internal consistency (Cronbach's alpha = 0.89) and good reliability (Split half = 0.92), has been shown to be able to differentiate patients with DDD from other clinical groups and has a high correlation ($r = 0.80$) with the depersonalisation subscale of the Dissociative Experiences Scale (Bernstein & Putnam, 1986) which is the most widely used measure of dissociation. The standard questionnaire asks the respondent to consider the previous 6-month period for review. However, to assess change and ensure there was no overlap when repeating measures, this period was changed to the previous 1-month for the pre-therapy and post-therapy timepoints.

Beck Anxiety Inventory (BAI) (Beck et al., 1988) and *Beck Depression Inventory (BDI)* (Beck et al., 1961) were used to measure the most common secondary diagnoses of those with DDD. These measures are two of the most widely used validated measures for anxiety and depression. The BAI has excellent overall internal consistency and a high test–retest correlation ($r = 0.67$) (Fydrich et al., 1992). Results have consistently shown good internal consistency and test–retest reliability of the BDI (Subica et al., 2014). Both are self-administered questionnaires that measure the severity of a range of, respectively, anxiety and depression symptoms. There are 21 items on each measure, scored from 0 to 3 for each item, with a total score of 63. The Beck Anxiety Inventory has a reported Cronbach's alpha of 0.92 (Beck et al., 1988) and the Beck Depression Inventory has a reported Cronbach's alpha of 0.85 (Ambrosini et al., 1991).

Statistical analysis

Data were visualised by scatterplots and bar charts to identify anomalous entries. Normality was assessed by quantile–quantile plots and histograms. Scatterplots were used to explore the assumptions for regression. Because the data comprised repeated measures at three time-points (baseline, start of CBT and end of CBT), longitudinal linear hierarchical analyses were carried out using time between appointments as the time variable. This model allows for intrapersonal correlation due to repeated visits, maximum use of data where missingness is present and the potential to model random effects. Analyses were controlled for the epidemiological variables of gender, age, and ethnicity. We tested the null hypothesis that there was no difference in the change in outcome scores between entering the service and starting active psychological therapy vs. the change in scores during the period for which psychological therapies were offered. We were not able to control for additional variables due to the limited sample size. Effect sizes are reported and are calculated by dividing the difference between the two group means by the pooled standard deviation for continuous outcomes error. We performed *post-hoc* sample size calculations (with a power of 90% and an alpha of 5%) to estimate the sample sizes required to find a difference between the two groups in the treatment phase of all three outcomes. For CDS, BDI, and BAI, the sample sizes required were 15, 30, and 40, respectively. The passage of time was accounted for by entering the visit period into the model. The regression models were built by forward selection using Akaike and Bayesian Information Criteria as

indicators of relative model fit. Post modelling verification of the models was carried out by plotting scatter graphs of residuals vs. fitted values and by examining caterpillar plots for abnormal outliers and influencers.

Results

Changes in outcome scores by treatment period for the three outcome measures were calculated, see Table 2.

Patients spent a mean of 10.6 months (s.d. = 5.9, range 1–26) on the waiting list and 17.3 months (s.d. 13.4, range 5–56 months) in therapy. There was no statistical evidence to support a statistically significant effect size in CDS or BAI between baseline and start of treatment, however there were statistically significant effect sizes during the treatment period. Participants showed a statistically significant decrease in both BDI effect sizes during the waiting period and during the treatment period. There were no differences in outcome scores between sex and age categories for any of the outcomes. There were no differences in clients' baseline scores (on all three measures) for those who improved vs. those who did not improve. Figure 2 shows the changes in CDS, BAI, and BDI scores at assessment, pre-therapy, and post therapy. This shows that there were clinically significant changes in all three measures for the treatment period, with medium effect sizes, compared to changes to the measures over the waiting period.

Discussion

The study results showed that clients experienced a reduction in symptom severity for DDD following CBT for DDD, as well as those with anxiety and depression. Although there was no randomisation of the sequence of interventions, the substantial period that participants were on a waiting list before receiving their CBT meant that they were able to act as their own controls, with symptom change over waiting list time compared to change over intervention time. For DDD and anxiety symptoms, there was no evidence for a change in symptom severity during the waiting period, but only after CBT for DDD.

Table 2. Outcome scores, sample distribution, regression coefficients and effect sizes for completers ($n = 36$) and total sample ($n = 50$).

Outcome	n	Phase	Mean (SD)	Coefficient [95% CI]*	Effect Size (d) [95% CI] **
CDS	36	Waiting Period	154.20 (63.15)	−4.25 [−16.59 to 8.09]	−0.07 [−0.26 to 0.13]
	36	Treatment Period	135.06 (69.01)	−35.99 [−48.45 to −23.52]	−0.52 [−0.70 to −0.34]
	50	Waiting Period	151.51 (62.55)	−6.60 [−4.42 to −17.60]	−0.11 [−0.28 to 0.07]
	50	Treatment Period	132.27 (68.76)	−33.67 [−45.14 to −22.20]	−0.49 [−0.66 to −0.32]
BDI	36	Waiting Period	24.97 (11.25)	−4.00 [−7.74 to −0.26]	−0.35 [−0.69 to −0.02]
	36	Treatment Period	19.10 (10.93)	−7.81[−11.58 to −4.04]	−0.71 [−1.06 to −0.37]
	49	Waiting Period	25.64 (10.89)	−3.89 [0.49 to 7.29]	−0.36 [−0.67 to −0.05]
	49	Treatment Period	19.80 (10.91)	−7.63 [−11.18 to −4.08]	−0.70 [−1.03 to −0.37]
BAI	36	Waiting Period	20.48 (11.33)	−0.03 [−3.26 to 3.19]	0 [−0.28 to 0.29]
	36	Treatment Period	17.28 (11.58)	−5.90 [−9.13 to −2.66]	−0.51[−0.79 to −0.23]
	49	Waiting Period	21.17 (11.63)	−0.52 [−2.40 to 3.44]	−0.05 [−0.30 to 0.21]
	49	Treatment Period	17.60 (11.84)	−5.57 [−8.59 to −2.56]	−0.47 [−0.73 to −0.22]

*Adjusted for gender, age, and ethnicity.
**Calculated by dividing the coefficient by the standard pooled standard deviation.

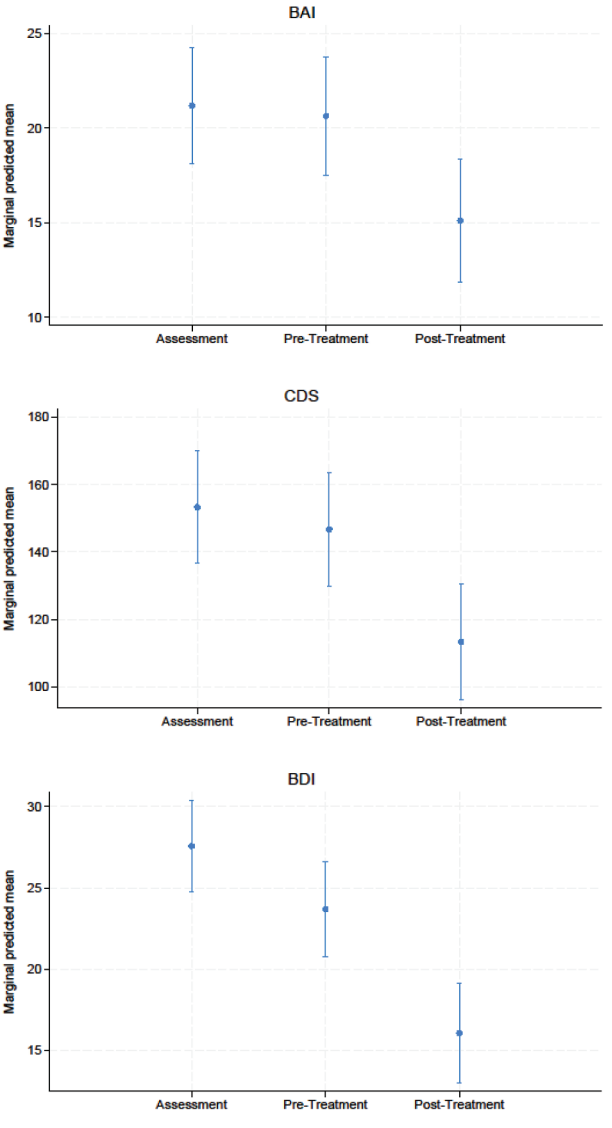

Figure 2. Changes in outcome measures at assessment, pre-treatment and post-treatment for completers ($n = 36$) sample.

Depression symptoms improved during the waiting period, and more so after the intervention had been delivered.

Given the chronicity of DDD in this group, with a mean of nearly 15 years of symptoms, obtaining a significant reduction in symptom severity with an average of 18 sessions is very promising. Moreover, as the intervention was delivered in a tertiary service this meant that all participants had already received treatment in either primary and/or secondary care services, but still met diagnostic criteria at the start of the study. Furthermore, the majority (78%) had at least one co-morbid diagnosis, making their

presentation more complex, and a third (33.3%) were unemployed due to the severity of their symptoms.

These outcomes replicate those from the previous evaluation of CBT for DDD (Hunter et al., 2005). The participants in both the 2005 and current studies were strikingly similar in terms of demographics and severity of clinical presentation, although the current sample was more ethnically diverse (which suggests improved access to specialist services). In both studies, statistically significant improvements were also found post-therapy in DDD, anxiety and depression scores. From this audit, the interpretation of the effect sizes for the Cohen's d statistic was around the medium range of magnitude (where 0.5 is a medium effect size; 0.8 a large effect size), with an effect size of −0.49 for the CDS and −0.47 for the BAI during the treatment period. For the BDI the effect size during the treatment period was larger at −0.70. In the previous audit, the interpretation of the effect sizes for the eta squared statistic (where >0.14 is a large effect size) showed that all effect sizes were in the large range of magnitude with effect sizes of 0.37 for the CDS, 0.30 for the BAI and 0.58 for the BDI.

It is likely that the difference in the magnitude of the observed effect sizes between the current and previous studies is attributable to the results in the current study being adjusted vs. non-adjusted in the earlier study. In both the current and 2005 studies the greatest improvement occurred in depressive symptoms during the treatment phase. This is an interesting finding, and there are a few possible interpretations of this result. Often if there is co-morbid depression to another primary condition, CBT therapists will treat the depression symptoms first to prevent depression symptoms such as hopelessness adversely affecting progress in treating other conditions. It may be that CBT is particularly effective in treating depression and/or that depressive symptoms are more responsive in the short term than DDD symptoms. There is some suggestion for the latter in the 2005 study, as DDD scores continued to improve after therapy with statistically significant change between end of therapy and 6-month follow-up, whereas depression scores plateaued in the same time period. A larger scale RCT would enable the impact of improvements to depression to be tested further with a mediation analysis via structural equation modelling.

When results from the current audit and the earlier 2005 study are added to other studies where CBT has been used to treat DDD, whether in adolescents (Flückiger et al., 2022), in those with DDD and psychosis (Farrelly et al., under review) or those with DDD in addition to DID (Mohajerin et al., 2020), there is increasing evidence of the effectiveness of the CBT approach, even in samples with considerable clinical complexity. Moreover, the Farrelly et al. and Flückiger et al. studies demonstrate that this effectiveness can be delivered even with a very small number of sessions (six and eight session interventions, respectively) and in a group format (Flückiger et al., 2022).

Although follow-up data was not collected in the current study nor the Farrelly et al. study, in the other studies (Flückiger et al., 2022; Hunter et al., 2005; Mohajerin et al., 2020), where follow-up data has been collected DDD scores improved further between end of therapy and 6-month follow up or were maintained at 6-month follow-up, indicating that a CBT intervention for DDD symptoms may have longer term benefits that extend beyond the duration of therapy itself.

In terms of future research, it would be useful to replicate these initial positive findings for CBT for DDD in both larger samples of those with primary DDD and samples of those with other primary diagnoses who have comorbid DDD. Longer follow-up periods would also help to calculate the duration of any benefits from the CBT for DDD. Further research would be helpful to determine the specific key components of CBT for DDD that are required to facilitate clinical change, such as identifying and modifying dysfunctional assumptions about DDD, as was reported most helpful in the Flückiger et al. (2022) sample. This could be conducted using both quantitative and qualitative data methodologies. Such research would help clinicians see if similarly effective outcomes as reported here can be obtained with fewer sessions, given the demands on healthcare providers.

Limitations

There were several limitations in the methodology of this study. This cohort was not randomised, and therefore the ability to draw stronger inferential conclusions is limited. The sample size was relatively small, and more work must be carried out on a larger sample with more varied composition in terms of age, occupation, and ethnicity before these results could be generalised to a larger population with confidence. A structured diagnostic interview was not used to assign diagnosis and diagnostic change was not measured as an outcome. We did not correct the results for multiple comparisons although we only undertook and reported three statistical analyses. Clinical staff assessing outcomes were not blinded so bias could account for some of the results. The CBT for DDD was carried out in a specialist clinic and by one therapist with many years of experience working with DDD, and a future study could assess inter-rater reliability between treatment outcomes from different therapists. The time period for DDD symptoms was changed on the Cambridge Depersonalisation Scale from the standard 6-month period to a shorter 1-month period at pre and post-therapy time periods to avoid overlap with data collected at earlier time points, but this could have affected the validity of the scale. In terms of therapy provision, there were differences in the frequency that CBT was delivered (i.e. weekly vs. fortnightly) due to the distance that some clients had to travel for sessions; furthermore, although the CBT for DDD delivered was the same as in the 2005 study, the therapy was not manualised. Both these factors could contribute to inconsistencies in delivery, potentially affecting the results and making replication more difficult. There was no follow-up data after the end of therapy, and therefore the duration of this improvement could not be measured.

Conclusions

CBT specifically adapted for DDD reduced symptoms of DDD, as well as anxiety and depression, at the end of an average of 18 therapy sessions in a sample of patients with chronic symptoms. These findings replicate an earlier study in 2005 but with a larger and more ethnically diverse sample. Considering the findings of the current study, the 2005 study and the other papers that examined the treatment effects of CBT for dissociation

generally, and DDD more specifically (Farrelly et al., under review; Flückiger et al., 2022; Mohajerin et al., 2020), the CBT approach shows considerable promise when working with dissociative disorders. However, a randomised controlled trial of CBT for DDD in routine services with non-specialist clinicians that includes follow-up data would be the next step in evaluating the effectiveness of this intervention.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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